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Review Article

Epidemiology of Blackleg Disease in Ethiopia

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Abstract

Blackleg is an infectious bacterial disease of cattle and rarely of other ruminants. This bacteria is caused by *Clostridium chauvoei* which is an anaerobic, gram positive, motile, rod-shaped bacillus bacterium and persists in the soil as resistant spores. Blackleg is an acute or subacute but chronic disease may occur. It occurs most frequently in animals 6-24 months of age and the disease mainly affects none vaccinated as well as animals in good nutritional condition. It produces persistent spores when conditions are not ideal and spores are highly resistant to environmental factors and disinfectants. Infected ruminants do not directly transmit the disease to other animals. The bacteria enter the body through the alimentary mucosa after ingestion of contaminated feed. Secretion of cytolytic toxins that cause necrosis of vascular endothelia .The toxins are absorbed into the animal's bloodstream which makes the animal acutely sick and causes rapid death. Economic importance due to blackleg is loss of animals, milk production and draft oxen, and cost for treatment and vaccination. Fatality rate of blackleg in fully susceptible populations approaches 100%. Clinical Signs include lethargy anorexia, reluctance to move lameness and recumbence. When superficial muscles are affected, swelling and crepitus are evident. Cattle found dead of blackleg are lying on the side with the affected hind limb stands out stiffly, bloating and putrefaction occur quickly and blood-stained from exudates, nostrils and anus. The disease can be diagnosed using laboratory diagnosis, Immune Fluorescent, Cell Culture and PCR. Control and prevention relies mainly on vaccination.

Keywords: Hematogenous; Fluorescent; Cytolytic toxins; *Clostridium chauvoei*

Introduction

Ethiopia is one of the countries with the largest number of livestock in Africa and livestock production plays a major role in the development of Ethiopia's agriculture. The cattle population in Ethiopian is estimated to be 59 million and 49 thousand (SCA, 2017). In Ethiopia, cattle play an important role in the livestock industry through provision of meat, milk, manure, and row materials for the processing industries. However, diseases are an important bottle-neck for livestock productivity in our country. Among many livestock diseases, blackleg is one of particular interest disease in the Ethiopia. Blackleg is an infectious disease of cattle and rarely other ruminants, caused by *Clostridium chauvoei*, and characterized mainly by necrohemorrhagic myositis. *Clostridium chauvoei* is an anaerobic, gram-positive bacillus that persists in the soil as resistant spores [1].

Blackleg occurs most frequently in animal's 6-24 mo of age, and cases in animals outside that age range are very rare [1,2]. Most losses due to blackleg occur when cattle are between the ages of 6 months and 2 years (Sarah 2013). In addition, Blackleg seldom affects cattle older than 2 years of age, most likely due to immunity induced by vaccines or natural exposure. However, sporadic cases do occur in cattle older than 2 years and are often associated with the reuse of needles for multiple injections. It is also indicated blackleg to be a problem in cattle less than 4 months old that do not receive adequate passive immunity through colostrums [3].

Control relies mainly on vaccination carried out at the onset of an di outbreak using the whole broth culture suspension of *Clostridium chauvoei* vaccine (local isolate) produced by the National Veterinary Institute (NVI, Ethiopia).

The objectives of this manuscript is thus

- To review on Blackleg Disease in Ethiopia

Literature Review

Etiology

The causative agent, *Clostridium chauvoei*, is Gram-positive, motile, rod-shaped anaerobic bacterium that can produce environmentally persistent spores when conditions are not ideal for growth (Sarah 2013). Clostridium was discovered in 1887 and was later named after a French veterinarian, JBA Chauveau [4]. It is a gram-positive, anaerobic, and spore-forming rod found in the soil, feces, and the digestive tract of many animals [1] mainly in the form of spores that are highly resistant to environmental factors Zand many disinfectants. This microorganism sporulates readily, producing ovoid, and usually sub-terminal, spores. Central and occasionally terminal spores also may occur. Iodine, chlorine, and other oxidizing disinfectants readily destroy vegetative and sporulated forms of *C. chauvoei*. Spores, however, tolerate the action of quaternary ammonium and phenolic disinfectants, and boiling [5]. On blood agar, most colonies of *C. chauvoei* are circular and surrounded by a thin ring of hemolysis. This characteristic somewhat depends upon the source of the red blood cells in the media and the strain of the isolate [5].

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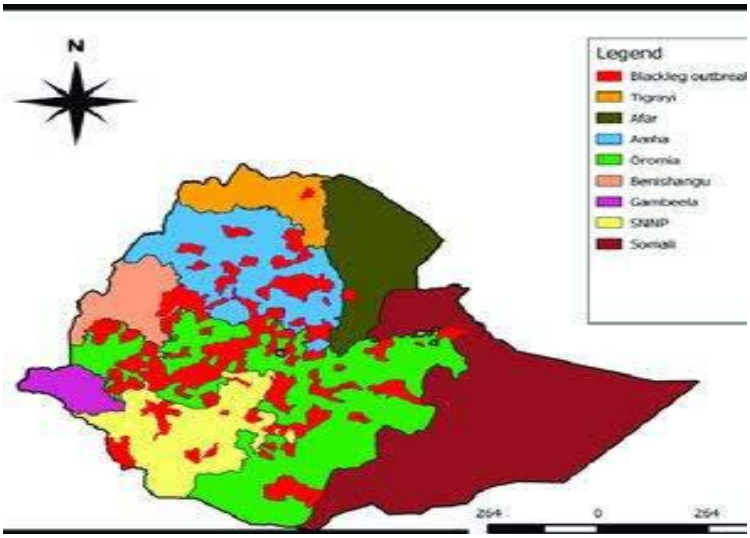

Figure 1:

Epidemiology

Blackleg affects mainly non vaccinated cattle between 6 mo and 2 y of age, with occasional cases occurring in animals outside this age range. The disease affects mostly animals in good nutritional condition, often on pasture [6].

Despite the frequently repeated claim that both spores and vegetative forms of *C. chauvoei* are found in feces of healthy and sick animals, little information is available in this regard in the scientific literature. The current, but unproven, dogma is that regardless of the origin, the spores of *C. chauvoei* can contaminate the soil, survive in the environment for decades, and can infect animals grazing on contaminated pastures Falquet et al., [7]. Infections by *C. chauvoei* of mostly unknown pathogenesis have been reported in goats, deer, oryx, elephants, horses, pigs, mink, fresh-water fish, whales, frogs, and hens. Gas gangrene and enterocolitis [8] associated with *C. chauvoei* have been reported, very rarely in humans (Figure 1).

Transmission: Mode of transmission: In sheep the disease is almost always wound infection. Infection of such wounds at hearing and docking and of the novel of birth may cause the development of local lesson. Infections of the vulvas and vagina of the ewe and rams up to year old, usually as a result of infection of skin would case by fighting. Occasional out breaks have occurred in sheep after vaccination against enterotoxaemia. Ewes exposed to infection at shearing develop typical lesion but ewes traded with penicilling are un affected except that the present ewe in the letter group shown distended abdomens, weakness and recumbence due to edema and gas formation in cattle mainly transmited through injection of spores [9].

Risk factor: Animal risk factor: True blackleg is usually thought as disease of cattle and occasionally sheep but out breaks of the disease has been recorded in deer and in one case in a horse. In cattle the disease is largely confined to young stock between the age of 6 month and 2 years. In the field the disease appears to occur most frequently in rapidly growing cattle on a high plane of nutrition. Elevation of the nutritional status of sheep by increased protein feeding increases their susceptibility to blackleg. In sheep there is no restriction to age group [10].

Environmental risk factor: Typical blackleg of cattle has seasonal incidence with most cases occurring in the warm month of the year. The highest incidence may vary from spring to autumn, depending probably on when calves reach the susceptible age group some outbreak of blackleg in cattle have occurred following excavation of soil which suggests that disturbance in soil may expose and activate latent spores [11].

Economic importance: Financial impact assessment the total cost (C) of a disease is calculated as the sum of production loss (L) (both direct and indirect) and expenditures (E) incurred to control the disease (Otte and Chilonda 2000). Mathematically, C=L+E. The annual financial losses due to blackleg from farmer's perspective were calculated as the sum of the values of the annual production losses due to mortality and morbidity and the costs for treatment and vaccination. The economic model by Bennett and Ijpelaar (2005), and (Kivaria et al., 2007) as cited in (Gari et al., 2011) was adopted for the estimation of financial loss attributed to blackleg with slight modification in some of the variables in the equation to best fit to blackleg.

Pathogenesis

C. chauvoei present in contaminated pastures are ingested and undergo one or more replication cycles in the intestine before being absorbed through the intestinal mucosa to the bloodstream and/or being excreted in feces [12]. Once absorbed, the spores are distributed to multiple tissues, including skeletal and cardiac muscle. Transport is assumed to be mostly hematogenous, although lymphatics may also play a role in dissemination of spores. Once the spores of *C. chauvoei* reach the muscle, they are phagocytized by resident macrophages, and can survive in the cytoplasm of those cells for long periods of time without affecting the host.

But when anaerobic conditions are created in areas where the spores are present, most frequently related to trauma and associated hemorrhage and necrosis, those latent spores germinate, proliferate,

and release toxins that produce the clinical manifestations and lesions of blackleg [13]. It is important to distinguish this pathogenesis from the so-called exogenous mechanism of gas gangrene, a disease produced 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 [14].

Transient trauma or ischemia of the muscle favors the germination of the spores and secretion of cytolytic toxins that cause necrosis of vascular endothelia (edema, hemorrhage) and myofibers. The toxins are absorbed into the animal's bloodstream which makes the animal acutely sick and causes rapid death. Clostridial proliferation yield gas which appears as bubbles between the muscle bundiles [15].

Diagnosis

Although a presumptive diagnosis of blackleg is usually based on clinical history, signs, and gross and histologic changes, a final diagnosis requires detection of *C. chauvoei* in affected tissues. This can be achieved by culture, PCR9 [16] and/or immunodetection methods, including FAT and IHC.1 Decisions on which tests to use are often based on availability at local diagnostic laboratories.

The primary differential diagnosis for blackleg is gas gangrene, also known as malignant edema, a clostridial cellulitis and sometimes also myositis, associated with wound contamination. This disease may be produced by one or more of several clostridial species, including *C. septicum*, *C. chauvoei*, *C. perfringens*, *C. sordellii*, and *C. novyi*. Gross microscopic lesions of blackleg and malignant edema can be difficult to differentiate when thedisease is restricted to skeletal muscle, especially in cases in which wounds are not readily evident. Additional tests are often needed for distinction [17].

Clinical signs: Blackleg is typically an acute or subacute disease, with many animals dying suddenly or rarely surviving more than 36 h after the onset of clinical disease. Occasionally chronic cases may occur. In cases in which clinical signs are observed, they include one or more of the following: lethargy, anorexia, reluctance to move, lameness, and recumbency. When superficial muscles are affected, swelling and crepitus are evident [18]. Cases with cardiac lesions have similar clinical signs, in addition to increased diffuse lung sounds and dyspnea. Signs of congestive heart failure such as jugular vein distension and brisket edema have rarely been reported.

Laboratory tests: Sporulated gram positive rodes can be demonstrated in smears of infected tissues and identified with immunofluorescent. Ground muscle in soil is cultured on blood agar plates which are incubated anaerobically. Because of the possible presence of swarming a septicemia early subcultures should be attempted from some plates with others last for 48hr identification by immunofluorescence or biochemical [19].

Isolation and culture: The organisms may in cases be isolated in pure culture directly from the tissues infected. Growth in culture Medias quite dependent up on the presence on carbohydrates for the best growth soda content beyond neutrality is advisable. Body fluids or tissues except as they may act as reducing agents or contain carbohydrate don't increase suitability of media containing the. The colonies are spherical or somewhat irregular, which microscopic radiator. The isolation and identification of the causal organisms from muscle lesion is difficult because of the rapidity with clostridia invade the tissues from gastro intestinal tract after death and of certain bacteriological species such as *Cl, chauvoei* and *CI. Novyi* [9].

Differential diagnosis

Anthrax: Anthrax is a hemorrhagic 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 kidn ckleg, is very similar to blackleg. In fact, the similarity is so close that often a diagnosis can be made only when the specific bacteria are identified in the laboratory.

Malignant edema: Malignant edema does differ from blackleg in some respects. It is caused by bacteria called *Clostridium septicum*. It is more common in older animals and is more likely to occur during the winter months than blackleg.

cases the disease however, demonstration or ehrlichia bodies in the endothelium cells of blood capillaries in the brain and Jugular vein, demonstrated presence of the Amblyomma ticks.

Snake bites: Snakebites was also one of the diseases we should have to differentiate from blackleg, since it's a sudden death with lack of clinical signs and occasional per pharyngeal and brisket oedema, can be confused with sub-acute disease .Bites on the muzzle, head and neck are more likely acute systemic sign and death. It is possible to see the marks from the bite and often local swelling tissue discoloration and hemorrhae at the sight or the bite are suggestive of snake bite poisinig [20-27].

Prevention and treatment

Treatment Treatment of affected animals with penicillin and surgical debridement of the lesion, including fasciotomy, is indicated if the animal is not moribund. Recovery rates are low because of the extensive nature of the lesions. Large doses (44,000 IU/kg BW) should be administered, commencing with crystalline penicillin intravenously and followed by longer-acting preparations. According to [21-36] treatment of clostridial myositis is rarely successful due to the rapid course. Antimicrobials (drug of choice procaine penicillin) around affected tissues, aggressive surgical debridement to allow aeration along with supportive treatment can be of value. Majority of cases show poor prognosis.

Blackleg can be prevented by vaccination, a procedure that is an important component of the health management of many cattle-producing operations [7]. Annual or bi-annual boosters are recommended after that, until 2 y of age another likely candidate for vaccine production is the neuraminidase of *C. chauvoei*. It is expected that a combination of this antigen with one or more polypeptides derived from the toxins produced by *C. chauvoei* will have a potentiated protective effect against blackleg [22-41].

Conclusion and Recommendation

Blackleg is one of the major bacterial infections of cattle with great economic losses in many parts of Ethiopia. Vaccination did not involve majority of cattle. Extreme caution should be taken when working with dead or blackleg suspected animal. Epidemiology of blackleg did not done well in all Regions of Ethiopia. Generally, blackleg is an endemic disease and one of the bottleneck diseases in livestock of the country.

Therefore based on the above conclusions, the following recommendations are forwarded:

- A regular and strategic vaccination should be given.
- Carcass disposal should be done carefully.
- Blackleg suspected cases should be treated early.
- Further research should be done on Epidemiology of Blackleg disease in Somali and Gambela Regions.

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