

# Prevalence of cardiac lesions in cases of bovine clostridial myositis

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## Research note

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# Abstract

## Objective

The aim of this retrospective study was to determine the prevalence of cardiac lesions in cases of clostridial myositis. Clostridial myositis (blackleg) is a common cause of death in cattle and is caused by the bacterium *Clostridium chauvoei*. The characteristic lesions include hemorrhage, necrosis, edema, and emphysema within skeletal muscle and, less commonly, the heart.

## Results

Of the 37 cases of blackleg identified, 26 animals (70.3%) had cardiac lesions, including 4 (10.8%) which had only cardiac involvement without skeletal muscle lesions. Cardiac lesions were characterized as necrotizing myocarditis (n=21), fibrinous to fibrinosuppurative pericarditis, epicarditis, or endocarditis (n=24), or both (n=19). These data demonstrate that, in cases of bovine clostridial myositis, cardiac lesions are common, while heart involvement in the absence of skeletal muscle lesions is uncommon.

## Introduction

Clostridial myositis, or blackleg, is caused by the bacterium *Clostridium chauvoei*.<sup>[1–5]</sup> Affected cattle are typically pastured and are between 6 and 24 months of age.<sup>[3]</sup> Ingested spores of *C. chauvoei* from contaminated soil are transported across the intestinal mucosa, presumably via M cells, and then disseminated to tissues, including striated muscle, where they are phagocytized by resident macrophages and survive intracellularly without deleterious effects.<sup>[3,6]</sup> In the face of reduced oxygen tension, usually following muscle damage, there is spore germination.<sup>[5]</sup> The now active bacteria proliferate and produce exotoxins, which result in tissue damage, systemic illness, and death.<sup>[5]</sup> A flagellum provides mobility and facilitates the spread of the organism.<sup>[2]</sup> Known toxins include a pore-forming hemolysin (toxin A or CctA), a cholesterol-dependent cytolysin (chauveolysin), a neuraminidase, and others.<sup>[2,4,6]</sup> Toxins diffuse into the tissues, resulting in necrotizing myositis.<sup>[6]</sup>

Although affected animals are typically found dead, some exhibit lameness or muscle swelling prior to dying. Since there are no effective treatments, <sup>[5]</sup> death typically occurs within 24–26 hours of the onset of clinical signs.<sup>[3]</sup>

At necropsy, affected muscles are dark red to black (necrosis and hemorrhage) with separation of muscle fibers by emphysema and edema; they may smell of spoiled butter (rancid) from the production of butyric acid.<sup>[1–6]</sup> Similarly, the microscopic changes are primarily necrosis and hemorrhage with edema, emphysema, and varying degrees of inflammation.<sup>[1,2,5]</sup>

Associated lesions include fibrinohemorrhagic pleuritis, pericarditis, and mural or valvular endocarditis. If present, valvular endocarditis typically affects the right atrioventricular valve.<sup>[7]</sup>

In some cases, necrotizing lesions similar to those seen in skeletal muscle occur in the heart.[1<sup>-3</sup>,5,7-10] The prevalence of cardiac lesions in cattle with blackleg in California was recently reported to be 69%.[1] This study aimed to determine the prevalence of cardiac lesions in cases of blackleg in Tennessee.

## Materials And Methods

The University of Tennessee Veterinary Medical Center database was searched for cattle with a confirmed diagnosis of blackleg necropsied between 2004 and 2018. Necropsy reports were reviewed for relevant gross and microscopic findings. When available, histology slides of skeletal muscle and/or the heart were examined to confirm the presence of supportive lesions.

## Results And Discussion

Thirty-seven cases of blackleg were identified. The presence of *Clostridium chauvoei* in the lesions was confirmed by immunohistochemistry [1] in 25 cases (67.6%), by culture and immunofluorescence in 11 cases (29.7%), and by polymerase chain reaction in 1 case (2.7%). In 32 cases (86.5%), affected skeletal muscle (n=6), heart (n=6), or both (n=20) were examined microscopically, and, when present, the lesions were consistent with blackleg in all of these cases. The remaining 5 cases were diagnosed based on the gross findings in conjunction with culture and immunofluorescence.

The affected muscles were grouped as limbs (any), cervical, thoracic (shoulder, pectorals), lumbar (hypaxial and epaxials), pelvic (gluteals), head, tongue, and diaphragm. Of the 37 cases, 33 (89.2%) had skeletal muscle involvement. Most animals (43.2%) had only 1 skeletal muscle group affected, 12 (32.4%) had 2 groups affected, 3 (8.1%) had 3 groups affected, and 1 (2.7%) had 4 groups affected. The limbs were most commonly affected, with 19 affected animals (51.4%). The involvement of other skeletal muscle groups included thoracic (n=12, 32.4%), neck (n=10, 27.0%), pelvic (n=4, 10.8%), tongue (n=3, 8.1%), diaphragm (n=3, 8.1%), lumbar (n=2, 5.4%), head (n=1, 2.7%).

Cardiac lesions were found in 26 of the 37 animals (70.3%); no cardiac lesions were identified in the remaining 11 cases. Of the 26 cases with cardiac lesions, 24 cases had gross lesions, while 2 cases were identified only by microscopic examination. Four animals (10.8%) had cardiac lesions without skeletal muscle involvement. Of the 26 animals with cardiac lesions, 2 (7.7%) had only necrotizing myocarditis; 5 (19.2%) had only fibrinous or fibrinosuppurative pericarditis, epicarditis, or endocarditis; and 19 (73.0%) had a combination of myocarditis and pericarditis, epicarditis, or endocarditis (Table 1, Figures 1, 2).

**Table 1.** Prevalence of cardiac lesions and proportional morbidity of bovine clostridial myositis due to cardiac lesions

<b>Cardiac lesions</b>	<b>Number</b>	<b>Prevalence (%)</b>	<b>Proportional morbidity due to a cardiac lesion (%)</b>
Necrotizing myocarditis only	2	5.4	7.7
Fibrinous to fibrinosuppurative peri-, epi-, or endocarditis only	5	13.5	19.2
Both necrotizing myocarditis and fibrinous to fibrinosuppurative peri-, epi-, or endocarditis	19	51.4	73.1
No cardiac lesions found	11	29.7	N/A
<b>Total</b>	<b>37</b>	<b>100</b>	<b>100</b>

The anatomic localization of the cardiac lesions was not always specified in the necropsy reports or able to be determined microscopically. Based on the available data, cardiac myositis primarily involved the left ventricle, whereas pericarditis, epicarditis, or endocarditis more commonly affected the right side of the heart.

In 6 cases (23.1% of cardiac associated cases; 16.2% of all cases), valvular endocarditis was noted grossly or microscopically. In 4 cases, it affected the pulmonic valve; in 2 of those cases, the left atrioventricular valve was also affected.

The 70.3% prevalence of cardiac lesions in Tennessee cattle with blackleg is similar to the 69% that was recently reported in California.[1] Of the 37 animals with blackleg, only 11 (29.7%) did not have heart involvement; 4 (10.8%) had involvement of only the heart; and 22 (59.5%) cases had involvement of the heart in addition to the skeletal muscle. Of the 26 cases with heart involvement, 92.3% had a fibrinous to fibrinosuppurative pericarditis, epicarditis, or endocarditis, and 80.8% had a myocarditis. The pericarditis, epicarditis, or endocarditis tended to involve the right side of the heart, while myocarditis tended to involve the left ventricle. Less commonly, there was valvular endocarditis, which most commonly involved the pulmonic valve. The presence of pulmonic valvular endocarditis is in conflict to previous literature indicating that the right atrioventricular valve is most commonly affected;[3] given the rarity of valvular endocarditis in cases of blackleg, the significance of this discrepancy is unclear.

The pathogenesis of clostridial myositis has been recently and thoroughly reviewed.[1,2,7] Cardiac lesions may be the result of hematogenous dissemination of the bacteria and/or toxins to the heart. In rare cases where there is only myocarditis, determining the inciting cause for the decreased oxygen tension required to cause vegetation of the spores is challenging. Some have proposed that concurrent

toxicities (ionophore, gossypol) or nutritional deficiencies (Vitamin A, selenium) may precipitate cardiac hypoxia, but to date, supporting evidence of these theories is lacking.[10] Stress of handling and increased cortisol may also allow for germination of latent spores.[10] Interestingly, outbreaks of clostridial myocarditis have been associated with high rainfall in cattle[9,10] and lambs.[7] The wet conditions may create an anaerobic environment in the soil, which favors the proliferation of *C. chauvoei*, or may facilitate the dissemination of spores.[2] It is unclear, however, how the wet conditions would favor activation of the spores in the heart. Given the nature of this study, it was not possible to make associations with weather conditions.

Involvement of the heart in cases of blackleg is common; however, clostridial myocarditis in the absence of skeletal muscle lesions is uncommon.

## Limitations

This is a retrospective study, so correlation of the data with other factors (weather conditions) was not possible.

## Declarations

### ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable

### CONSENT FOR PUBLICATION

Not applicable

### AVAILABILITY OF DATA AND MATERIALS

The dataset supporting the conclusions of this article is available on Dropbox:  
<https://www.dropbox.com/s/f2ltwk19ys8w6ic/blackleg%20data%20%2012-14-20%20without%20owner%20names.xlsx?dl=0>

### COMPETING INTERESTS

None

### FUNDING

None

### AUTHORS' CONTRIBUTIONS

KN designed the project, made diagnoses and mentored CC. CC helped identify the cases and organize the data. CO performed the statistical analyses. FU and his lab performed the immunohistochemistry and he assessed the slides for immunoreactivity to confirm the diagnosis for many cases.

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## Abbreviations

HE Hematoxylin and eosin

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# Figures

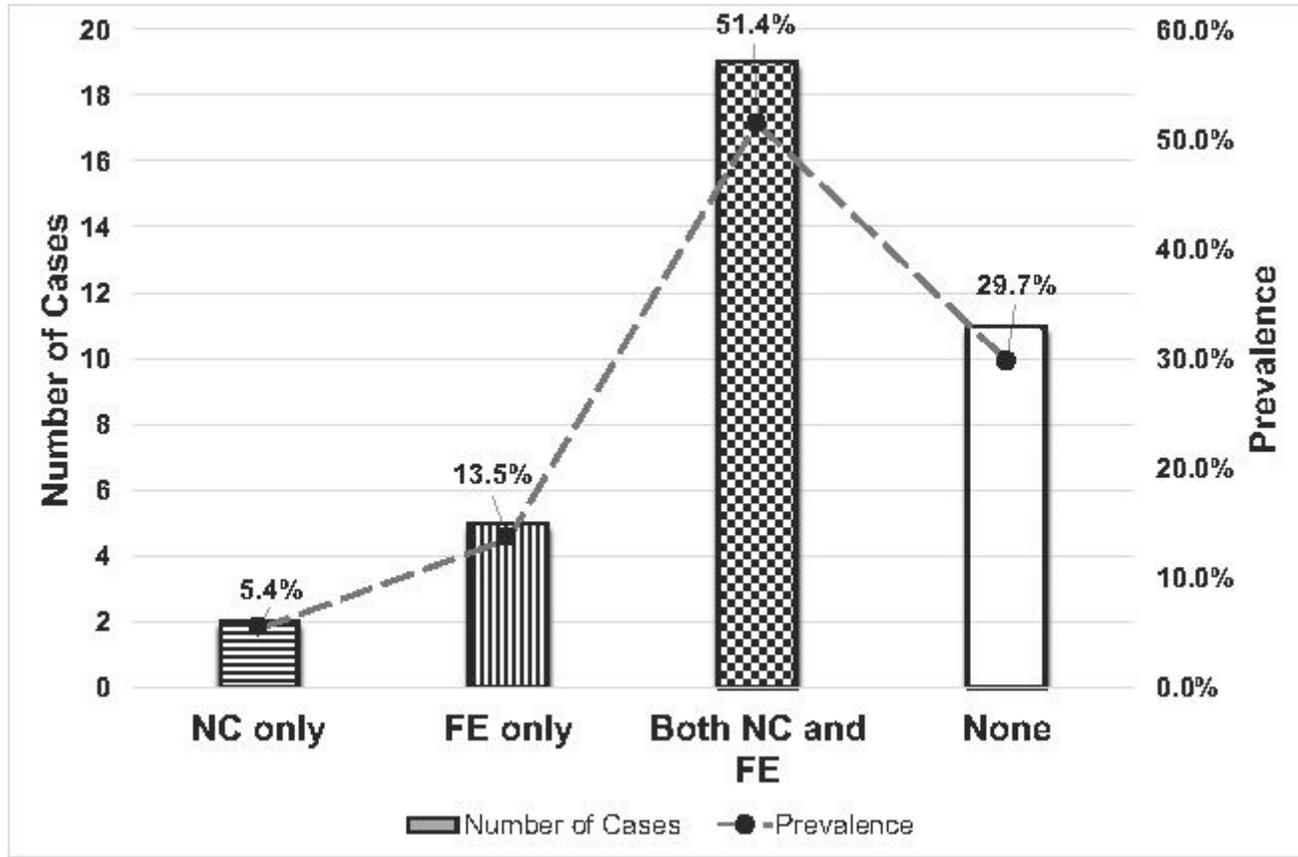
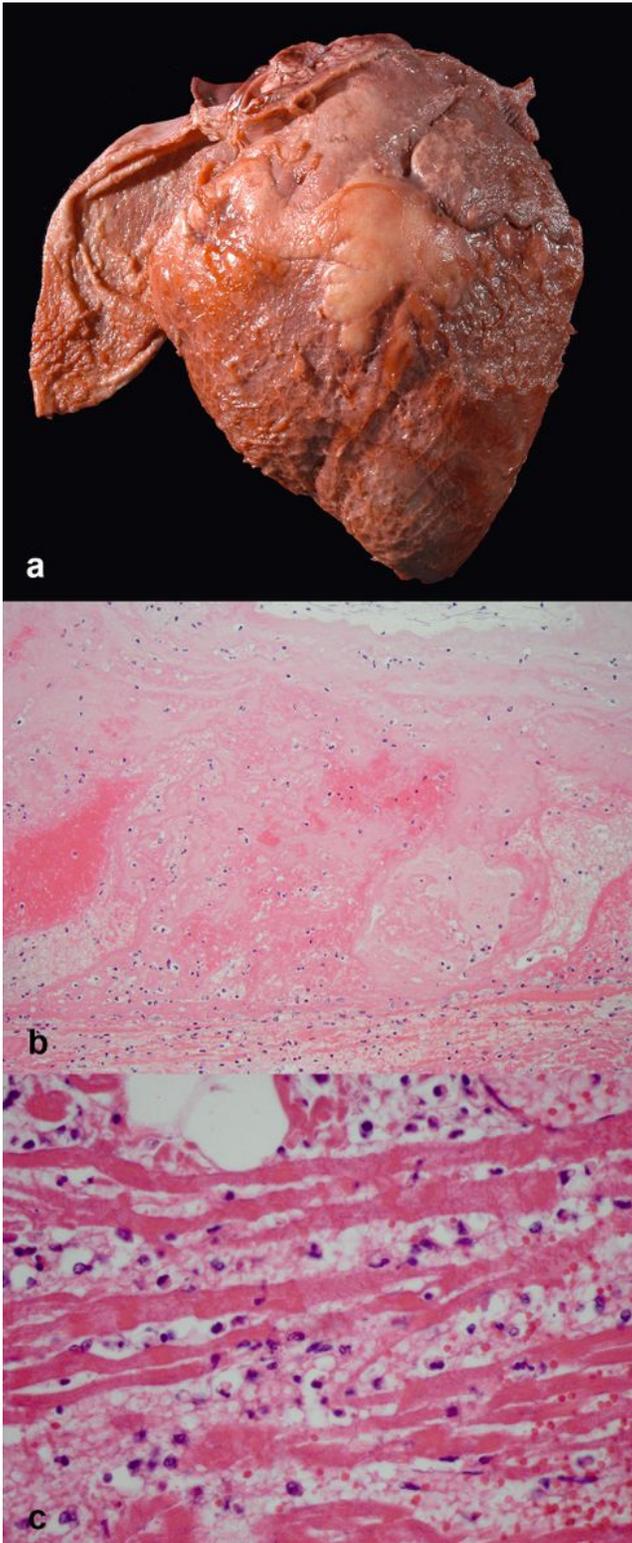


Figure 1

Number and prevalence of cardiac lesions in cases of bovine clostridial myositis



**Figure 2**

Heart lesions associated with clostridial myositis, bovine. a. The epicardium and attached pericardium are covered in fibrin. b. The endocardium is markedly expanded by fibrin, hemorrhage, and neutrophils. Bacterial rods are present superficially. Hematoxylin and eosin (HE). c. There is fragmentation and necrosis of the cardiomyocytes, infiltration by neutrophils, and expansion of the interstitium by edema, emphysema, fibrin, and hemorrhage. HE.