Pathology in Practice

History

A 10-month-old 322-kg (708-lb) Angus heifer had a 2-day history of signs of depression and anorexia; during the 2-day period, it was frequently observed to remain isolated and to be reluctant to move or recumbent. The heifer had been administered a clostridial bacterin-toxoid vaccine against diseases caused by 7 clostridial species or types approximately 3 months previously. A similar heifer was found dead in the pasture 10 days prior to evaluation of this heifer but was not necropsied.

Clinical and Gross Findings

The heifer was examined at the Oklahoma State University Veterinary Teaching Hospital. On physical examination, the heifer had signs of depression and was febrile (rectal temperature, 40.8°C [105.5°F]) and 5% dehydrated; slight jugular vein distension was noticeable, and mild brisket edema was present. Auscultation examination revealed increased diffuse lung sounds, an abnormal breathing pattern, and a pericardial friction rub that was heard only on the left side. Ultrasonographically, the amount of pericardial fluid appeared to be greater than that considered typical in a healthy bovid.

The heifer suddenly died while being examined and was submitted for necropsy to the Oklahoma Animal Disease Diagnostic Laboratory. The necropsy was commenced immediately. The heifer had been in excellent nutritional condition, but important gross lesions were detected in the thoracic cavity. The pleural cavity contained approximately 300 mL of serosanguineous fluid and abundant, confluent mats of fibrin. The adjacent portion of a lung has prominent pulmonary edema of interlobular septa. In panel B, notice the multifocal, variably positioned, deep red foci of myocardial necrosis. In panels A and B, bar and scale = 2 and 1 cm, respectively.

Figure 1—Photographs of the thoracic cavity (left lateral perspective; A) and a cross section of the left ventricle (including the left ventricular free wall and interventricular septum; B) in an Angus heifer that died suddenly while being examined because of a 2-day history of signs of depression, anorexia, tendency to remain isolated from other animals in the herd, and reluctance to move or recumbency. In panel A, the prosector has a gloved finger in the opened pericardial sac, which contains serosanguineous fluid and abundant, confluent mats of fibrin. The adjacent portion of a lung has prominent pulmonary edema of interlobular septa. In panel B, notice the multifocal, variably positioned, deep red foci of myocardial necrosis. In panels A and B, bar and scale = 2 and 1 cm, respectively.
**Histopathologic Findings**

All skeletal muscles (including tongue and diaphragm) were sectioned and examined grossly, and sections of cardiac tissue were examined microscopically.

The normal myocardial and epicardial architecture was markedly yet partially disrupted by extensive cardiac myocyte necrosis, infiltrates of large numbers of degenerate and nondegenerate neutrophils, fibrin deposition, and hemorrhage. Epicardial adipose tissue was necrotic and infiltrated by degenerate neutrophils admixed with an abundant amount of fibrin. Blood vessels within the epicardium and myocardium contained fibrin thrombi, and perivascular neutrophilic inflammation was often severe. There were several loci of interstitial fluid, hemorrhage, and clear spaces separating myofibers and myofiber bundles (Figure 2); some clear spaces possibly represented early gas bubble formation. Cardiac myocyte necrosis and inflammation were characterized by myofiber fragmentation, myofiber hyalinization, basophilic nuclear fragmentation, and neutrophil infiltrates (Figure 3). Multifocally, cardiac myocytes contained few 100- to 200-µm-diameter, 200- to 300-µm-long, membrane-bound parasitic cysts without associated inflammation. The cysts contained many curvilinear 10- to 20-µm-long zoites (the morphologic characteristics of which were consistent with *Sarcocystis* spp). Examination of skeletal muscle tissues did not reveal a focus of skeletal muscle necrosis.

The myocarditis and epicarditis were accompanied by severe fibrinosuppurative pericarditis. Sections of fresh myocardium underwent fluorescent antibody testing for *Clostridium chauvoei*, *Clostridium novyi*, *Clostridium septicum*, and *Clostridium sordellii*; results indicated that the tissue was positive for *C chauvoei* and negative for *C novyi*, *C septicum*, and *C sordellii*.

**Morphologic Diagnosis**

Severe necrosuppurative myocarditis and fibrinosuppurative epicarditis (consistent with *C chauvoei* infection [clostridial cardiac myonecrosis, also known as visceral blackleg disease]).

**Comments**

In the heifer of this report, myocarditis and epicarditis were accompanied by severe fibrinosuppurative pericarditis and results of fluorescent antibody testing indicated that myocardial tissue was positive for *C chauvoei*, the etiologic agent of blackleg. Specifically, the lesion pattern specified a form of the disease that is sometimes called visceral blackleg, which we determined to be the cause of death.

Blackleg, caused by the gram-positive, anaerobic, spore-forming rod *C chauvoei*, is a worldwide disease of ruminants and a common cause of sudden death in cattle. Blackleg is most commonly a disease of cattle 9 to 24 months of age but can occur in older adult cattle. Principally a disease of pastured cattle,
it typically develops during summer months. Pathogenesis of the disease begins with ingestion of spores from the soil and their translocation across the intestinal mucosa, most likely via macrophages. Spores residing in phagocytic cells are then distributed widely to other tissues, including muscle. Germination of the spores, which generates vegetative cells, is thought to occur when an insult results in muscle damage or creation of an otherwise hypoxic environment. Under such conditions, vegetative cells produce alpha, beta, gamma, and delta toxins, which cause muscle necrosis, hemolysis, and terminal septicemia. The classic form of blackleg causes variably sized, deep red to black foci of hemolysis, and terminal septicemia. The classic form of such conditions, vegetative cells known as sarcocysts, are considered an intriguing but unproven possible cause of signs of septicemia or acute heart failure in bovids. When performing a gross necropsy on affected cattle, careful examination of the heart and identification of cardic myonecrosis with serofibrinous to fibrinosuppurative pericarditis could lead to a tentative diagnosis of visceral blackleg. Confirmatory testing can be performed at veterinary diagnostic laboratories; the mainstay of such assessments is fluorescent antibody testing for Clostridium chauvoei and other clostridial species. Appropriate vaccination and treatment regimens can be instituted for the remainder of the herd. Multivalent clostridial bacterin toxoids are both inexpensive and efficacious in the prevention of the disease and should be administered in accordance with the product label. Treatment of clinical cases is often unrewarding, but administration of penicillin and supportive measures such as fluid therapy and administration of NSAIDs may be of benefit. Anecdotally, many practitioners advocate chemoprophylactic treatment with penicillin in at-risk herds while awaiting a protective vaccine response; this practice has not been rigorously examined or subjected to any formal cost-benefit analysis. Finally, although a comprehensive vaccination history for the heifer of this report and the herd from which it came could not be definitively reconstructed, the best available interview evidence indicated that the clostridial vaccine received was only the primary vaccination; no booster vaccination was administered. This is perhaps the reason that this heifer developed blackleg despite a history of some vaccination. Other explanations could include vaccine failure or faulty vaccine administration.

**References**