Anaplasmosis

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Anaplasmosis is a vector-borne infectious, hemolytic, rickettsial disease of cattle, sheep, goats, and other wild ruminants. In Cattle the most common etiological agent is Anaplasma marginale, while cattle also are affected with Anaplasma caudatum, which may result in severe disease and Anaplasma centrale generally resulting in mild disease.[1] An anemia results from extra-vascular hemolysis when parasitized red blood cells (RBC's) membranes are altered and recognized by the reticuloendothelial system. These RBC's are removed and finally destroyed.[2] Accordingly, antibodies that develop against the altered cell membrane can cause destruction of uninfected erythrocytes.

The severity of anaplasmosis depends on the species involved and age of animal. Young calves seem to have an innate resistance to the disease while the acute form generally occurs in cattle from 1 to 3 years. In cattle over 3 years, the peracute or most severe form, with rapid onset and death, predominates. Animals that survive anaplasmosis can become carriers for life and act as a reservoir of infection for susceptible animals.[2] Economic losses from anaplasmosis include abortions, death, weight and gain losses, decreased milk production, bull infertility, and treatment expense.[3,2]

Epidemiology

Today, anaplasmosis can be found nearly worldwide and occurs in many areas of the United States. It is most prevalent in the South and southwestern U.S. but also is present across the Midwest in scattered pockets having no set pattern. Anaplasmosis can be transmitted by several different methods, however it is not contagious.

A transfer of blood must take place from an infected animal to an animal that is susceptible if infection is to occur. The disease commonly occurs during the warm months when arthropod vectors, both biological and mechanical are abundant.[4] Ticks are the most important biological vector with the Dermocentor species implicated in most cases in the United States. A biological vector implies that the etiological agent is passed between different stages of the tick; for example, from the larval to nymph stage and then to the adult stage. Mechanical transmission can occur with other vectors such as blood sucking flies (horse and stable flies) and mosquitoes. Mechanical transmission in this manner must occur rather quickly, and as a result, is more important in operations with closely confined cattle. Accordingly, humans can be a mechanical vector by using anaplasma-contaminated equipment such as scalpels, needles, and tatoo equipment on susceptible animals.[2]

Pathogenesis and Clinical Signs

Anaplasmosis generally is categorized into four stages: incubation, developmental, convalescent, and carrier. The incubation period is the time from which the organism is introduced into the suscep-

tible animal until 1 percent of RBC's become infected.[6] The length of this stage seem to vary directly with the number of organisms the animal is exposed to and no clinical signs will be seen at this time.[6] During the incubation period, the packed cell volume (PCV) will remain constant, as RBC's will be produced at the same rate they are destroyed.[2]

The developmental stage and clinical onset of anaplasmosis is determined by the incubation period, which can be from 15 to 45 days depending on the animal. Clinical signs begin to appear as RBC production drops and more erythrocytes are parasitized and destroyed. Studies indicate that some animals show signs at as little as 10 percent loss of RBC's, while others indicate a 65 percent loss before onset of clinical signs, with a mean somewhere between 35 percent and 50 percent.[1,2,3] Erythrocyte count, PCV, and hemoglobin values will be severely reduced following the developmental period.[1]

As the anemia becomes more severe, the acutely infected animals lose condition rapidly. Icterus, weight loss, dehydration, constipation with hard dry feces shaded green, dark yellow urine, and progressive respiratory signs may become evident. Moreover, aggressive behavior, abortion of pregnant animals, and death due to hypoxia may occur. An animal that survives a bout with anaplasmosis requires a convalescent period of up to three months, with hematologic parameters (blood values) returning to normal. Often, these clinically recovered animals become carriers for life, and act as a reservoir for the disease.[1,2]

Necropsy of deceased animals will generally show a markedly anemic and jaundiced animal with thin and watery blood. The spleen usually is enlarged and soft with prominent follicles, while the liver appears mottled and yellow-orange in appearance.[1] The gall bladder often is distended and the bile appears thick brown or green. Regional hepatic and mediastinal lymph nodes appear brown.[1]

Diagnosis

Upon observation of clinical signs the diagnosis is confirmed by the presence of marginal bodies on a Giemsa, Wright's, of Diff-Q stain. A. marginale and A. caudale appear as dark staining blue-purple bodies 0.3(m to .1(m in length, along the margins of the erythrocyte. A. centrale inclusion bodies will be more centrally located and can be differentiated in this way.[1,2] Persistently infected cattle can be identified by serological tests using complement fixation, card agglutination, enzyme linked immunosorbent assay (ELIZA), or DNA-based tests.[5]

Treatment

Treatment of sick animals is difficult, as clinical signs may not appear until the disease has progressed to the acute stage. Sick animals in the acute stage can be hard to handle and stress must be kept at a minimum to prevent animals dying from hypoxia.

A multifaceted approach to treatment may be necessary and depends on the stage of the disease and when treatment is considered.[1] Tetracyclines are the drug of choice for treating and controlling anaplasmosis. When the percentage of infected erythrocytes is less than 15 percent (about halfway into the developmental stage), a single parenteral injection of oxytetracycline (OTC) can be effective in reducing the severity of the disease.[6] If the OTC stops the increase in infected RBC's before it reaches a critical level, the animal has a good chance of survival. Blood transfusion, electrolyte solutions, and hematinic drugs may be of some benefit in treating the disease at this stage.

The effectiveness of oxytetracycline is reduced when the animal has more than 15 percent of its RBC's parasitized. Recovery will depend on the animals natural ability to produce erythrocytes from bone marrow in sufficient numbers to compensate for the loss.[6] As the developmental stage progress and the convalescent stage begins, frequently the best treatment is no treatment at all. One reason for this is the increased risk of sudden death due to anoxia, from the animal becoming stressed when treated. The second reason is OTC do little or nothing to change the outcome of the disease at this stage.[6] Finally, erythropoietic-stimulating drugs do not have time to work, and a blood transfusion with enough blood to be beneficial could overload an anoxia weakened heart.

Prevention

Prevention and control of anaplasmosis can be difficult and requires planning. Each farm or ranch is different, therefore the same program will not apply to all places. The following procedures should be considered.

1. Control insect vectors.

Absolute control of all insect vector is impossible. Select economic control programs, such as periodic

spraying that reduces insect populations or removing stagnant water that host insects involved in transmission.

2. Follow strict sanitation procedures during vaccination or surgery. It is difficult and time consuming to change needles after every vaccination, but is very important in herds with anaplasmosis.

3. Test the herd and remove carriers. This type of program necessitates bleeding each animal, identifying carriers, and removal of the carriers. An alternative to disposing of carrier animals may be to separate carrier animals and clean animals. Creating two separate herds provides that no susceptible animals are in the 100 percent carrier herd.

4. Vaccination in endemic areas may be beneficial. The use of an experimental killed organism product produced by Louisiana State University is available with the permission of your state veterinarian and the USDA.

5. Chlortetracycline (CTC) can be fed during the vector season to effectively prevent transmission to susceptible animals. CTC can be consumed at the rate of 1.1 mg/kg(.5mg/lb) body weight daily, and may be administered using medicated salt-mineral mixes or medicated feed blocks.[6]

6. Do not sell animals before recovery, condemnation may result as icterus conditions do occur.

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