Anaplasmosis in Iowa

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In 2010 we saw an increased number of cases of Anaplasmosis from submissions to the Iowa State University Veterinary Diagnostic Laboratory (ISU-VDL). Although the disease is not enzootic in Iowa it is observed regularly. Environmental conditions in 2010 may have enhanced the spread of anaplasmosis within the state.

Anaplasmosis is caused by a protaozoan parasite (*Anaplasma marginale*) that is spread by ticks and biting insects. Additionally, it can be transmitted by needles or surgical equipment. Once the parasite reaches the blood stream in infects red blood cells (RBCs) where they multiply. Anemia results from a phagocytosis of the RBCs. Anemic animals will develop icterus (jaundice) but not hemoglobinuria (red urine) because RBC destruction occurs from phagocytosis in the spleen rather than RBC lysis in the blood vessels.

Clinical Signs

Clinical signs of anaplasmosis are associated with anemia. In acute anaplasmosis cattle will be febrile and anemic with an increased heart and respiratory rates. Cattle may also have muscle weakness, inappetence and depression. Mucus membranes will be pale. Icterus develops several days after onset of symptoms. Peracute anaplasomosis can be seen in highly susceptible animals such as adult purebred animals or high-producing dairy cows. These cattle can succumb to the infection within hours. Chronic anaplasomosis may follow an acute infection for up to 3 months of poor response. Young animals will show less severe clinical signs, due to a more responsive production of RBCs. Abortions are common in pregnant cattle with acute anaplasmosis. Bulls may show a temporary infertility which could be important in a fall breeding program.

Diagnosis

Diagnosis during the acute stage of the disease is usually based on clinical signs, presence of anemia and microscopic examination of a stained blood smear. In cattle the *A. marginale* organisms are located on the periphery of the RBC. However, in some acute infections the organism is not visible on a blood smear because all infected cells were removed from circulation and immature erythrocytes are usually not infected. Serology for *A. marginale* can be useful to aid in diagnosis in these cases.

Necropsy examination of cattle that die from anaplasmosis reveals either pale and anemic or icteric depending on stage of disease. An enlarged spleen and a swollen liver with enlarged gall bladder are also present. Urine will NOT be discolored with hemoglobin.

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Submission samples include blood smear or EDTA blood tube sample, Serum sample or clotted blood sample for serology. Tissues to submit include liver, spleen, kidney.

Treatment

Tetracycline is the drug of choice for clinical anaplasmosis. A single dose of long-acting 200 mg/ml oxytetracycline is usually sufficient. General supportive care is also important for anemic animals. Blood transfusions are of limited benefit. Large volume is needed because of the severe anemia and because the erythrophagocytosis system is activated transfused RBC are removed from circulation in 24-48 hours.

Control

Typically, cases of anaplasmosis increase in late summer and fall as insect vectors increase. Therefore, control of vectors is key to preventing anaplasmosis. If necessary herd treatment with oxytetracycline injection every 3 to 4 weeks during high risk times may be necessary will prevent clinical disease but animals can become carriers. Another option is oral administration of chlortetracycline at 1.1 mg/kg daily. To remove carrier state cattle need to treated with long acting oxytetracycline every 3 days for 4 treatments or 2 mg chlortetracycline per pound body weight orally per day for 50 days. Cattle will become serologically negative 3-4 months after the carrier state has been removed.