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Bovine Leptospirosis and Its Control

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Leptospirosis is a disease of cattle which varies from an inapparent to an acute fulminating disease in some cattle. Most livestock owners only associate leptospirosis with abortions and stillbirths. However, as with many diseases, the overt signs vary with age and physiologic state of the affected animals and the pathogenicity of the agent.

In the United States, six serotypes, *pomona, hardjo, szwajizak, grippotyphosa, canicola,* and *icterohaemorrhagiae* have been isolated from cattle (3, 6, 11, 22, 24, 28). All belong to separate serogroups except *hardjo* and *szwajizak* which are members of the hebdomadis serogroup and, therefore, are difficult to distinguish between serologically. Several other serotyps have been detected serologically only in U.S. cattle. Serologic testing involving reports from 20 diagnostic laboratories and 66,22 sera from 18 states gave reactor rates of 7.2% for *hardjo* end 6.5% for *pomona* (7). These two serotypes are apparently transmitted directly from cow to cow in most cases while *grippotyphosa, canicola,* and *icterohaemorrhagiae,* which are of lower incidence (0.7% to 1.4%) are extensions of infections from wildlife (2,21). However, once the less common serotypes become established in a herd, they can become a major herd disease problem (11,28).

Leptospirosis is caused by long filamentous spirochetes which move rapidly in liquid media, and enter the body through breaks in the skin or through intact mucous membranes. Transmission can occur during breeding (25). After an incubation period of four to ten days when the organisms are multiplying rapidly, the bacteremia occurs which becomes apparent by a rise in body termperature of one to four degrees in many animals. At the acute stage, depression, anorexia, hemoglobinuria, anemia, and jaundice can occur, and agalactiae may be present in lactating animals (10,16,28). The involvement of the mammary gland results in yellow clotted milk but no swelling of the udder as with most bacterial mastitis infections. The reaction in the mammary gland is suggestive of an endotoxin reaction (1). Usually the temperature returns to normal in 12 to 48 hours but may persist several days. Leptospires which are present in most tissues during the acute stage, multiply in the greatest numbers in liver, spleen, kidney, and brain tissue. Signs of encephalitis, incoordinated gait, may or may not occur at this time even though leptospires are often present in the brain.

Agglutinins usually can be detected in the serum several days after the onset of the acute signs. The antibody titers rise rapidly in a period of one to two weeks and then remain at a significant level, MA titers of 1:100 or greater, for several days to many weeks or months (12). Leptospires are shed from a few weeks to three or four months in the cattle urine. Organisms may persist in kidney and brain tissue beyond the shedding period, but can only be demonstrated by culturing the tissue. In some cases, leptospiruria also persists after the agglutinins are no longer detected in the blood.

Abortion, stillbirths, and weak calves are a common response when pregnant cattle are infected in the last half of the gestation period (20). The fetus becomes infected during the acute stage but abortions and stillbirths occur one to four weeks later after the calf dies and the fetus is expelled. The death of the fetus is apparently due directly to either an acute infection or possibly indirectly from endotoxins. At the time of the abortion or stillbirths, antibody titers may be detectable in the fetus if the fetus survives an adequate period for an immune response (9).

Calves infected late in the gestation period are usually born with a bacteremia and appear weak. Weak calves may die during the first week or survive but gain weight slowly. Some calves infected at birth appear normal but shed leptospires in the urine for several weeks (28).

Calves nursing cows with significant agglutination titers will absorb large amounts of the leptospiral antibody from the colostrum (5,12,23). Many nursing calves retain detectable agglutinins for three to four

months and a few as long as five or six months (5).

Infertility, as expressed by repeat breeding, has been commonly associated with *hardjo* infections and has been reported in *szwajizak* infections (6). This sign may be the only evidence of chronic leptospirosis and is an indication of an endemic infection in the herd. A serologic test of the entire adult herd should be conducted to evaluate the possible relationship of leptospirosis to an infertility problem (12).

Diagnosis of clinical leptospirosis is difficult due to the varied symptomatology and occurrence of inapparent infections. Clinical signs are suggestive in many cases but not conclusive so laboratory confirmation is an important procedure. Serologic tests which usually consist of either the plate agglutination test or microscopic agglutination test are the most common laboratory procedures. As agglutinins appear early and often persist for a few weeks to several years, it may be difficult to assess the relationship of the agglutination reaction in a few animals with an outbreak of disease. Collection of serum from a representative herd sample of 10% or more animals provides a more reliable approach. All animals showing subacute or chronic signs should have agglutinins present; animals in the acute stage would be negative.

Conclusive proof of an infection is dependent upon isolation of the serotype in laboratory media or in inoculated laboratory animals (8). Urine is the most reliable source of infection and aborted or stillborn fetus the least (24). However, isolations have occasionally been made from aborted feti and milk collected during acute signs. The laboratory animal of choice is the weanling hamster.

Determination of the leptospiral status of a serologically positive bull is difficult. It is unlikely leptospires are shed either in the semen beyond the acute stage of the disease or from urine longer than three months. Semen collected and handled by freezing and adding of antibiotics should eliminate active leptospires. The semen can be further evaluated by the inoculation of five or six weanling hamsters with semen and examination of the hamsters three weeks later for antibodies or leptospires in the urine.

As four of the six leptospiral serotypes from U.S. cattle also infect other domestic animals and wildlife, control by eradication and isolation is only possible in herds with complete confinement programs. The closed-herd practice will reduce exposure from domestic animals but will not exclude wildlife contacts. Therefore, a more practical approach is to vaccinate the cattle against the common serotypes in the area. In the United States, *pomona, hardjo* and *grippotyphosa* which are quite widely distributed and *canicola* and *icterohaemorrhagiae* to a lesser extent have all been isolated from cattle. Vaccination as a herd preventative procedure should start with the calves at four to six months of age, followed by revaccination at one year and revaccination yearly thereafter (13). With this procedure a herd immunity is established which should abort any infections resulting from outside exposure. Vaccination with a bacterin produces an antibody response involving both IgM and IgG classes of antibodies (18,19). However, the IgM antibody response, which can be measured by agglutination tests, is minimal and of short duration following vaccination. The IgG response, which can be measured by the hamster protection or growth inhibition tests, is substantial and persists for six to 14 months (14,15,27). Revaccination primarily enhances the IgG response (15).

Control of acute outbreaks by treatment with an antibiotic and vaccination is advisable if diagnosis is made early (7). In dairy herds, treatment presents a problem as the most effective antibiotic is dihydrostreptomycin which requires a greater withdrawal period than the tetracyclines (26). Generally treatment in a dairy herd outbreak should be limited to only the cattle which have had clinical signs of leptospirosis. Infusion of the udder with antibiotics during acute leptospirosis does not change the course of the disease.

In beef cattle herds, a leptospiral outbreak can best be controlled by treatment of all the contact animals with dihydrostreptomycin (25 mg per kilo) and vaccination with the appropriate serotype bacterin (26).

Experience has shown it is worthwhile to vaccinate the entire herd with a bacterin of the serotype involved (4,13,29). Future control of leptospirosis looks promising due to recent studies on the evaluation of bacterins. Present bacterins administered annually and used properly effectively prevent the serious signs and lesions associated with leptospirosis. Limited infections can occur in vaccinated animals, and a temporary shedding of leptospires may occur (2,17). However, in a vaccinated herd, the disease is self-limiting without associated losses (13). Studies are in progress to further assess the efficiency of leptospiral bacterins and to attempt to develop more effective products. The development of multiple serotype bacterins has been recommended, as they will provide protection against several serotypes and an increase in antibodies to common antigens (6). A bacterin containing *canicola* and *icterohaemorrhagiae* antigens has been available for several years. Recently, a product containing *pomona, hardjo* and *grippotyphosa* has become available (7).

Summary

Bovine leptospirosis is an important infectious disease of U.S. cattle. The disease causes both acute and chronic signs. The delayed effects of the disease, abortion and stillbirths are the most common forms recognized although acute signs which are less dramatic may go undiagnosed.

Diagnosis is often dependent upon laboratory techniques as leptospirosis is so varied in its host response. Serologic tests are available at most diagnostic laboratories, but the spectrum of antigens

available vary considerably.

Control is primarily limited to antibiotic therapy and vaccination. Early diagnosis followed by treatment is effective in limiting the extent of outbreaks.

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Differential Diagnosis, Treatment and Prevention of Diarrhea in Brood Cows and Yearlings

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This presentation should be titled "Diarrhea, a Symptom of Many Diseases."

Diarrhea by definition is an abnormal frequency and liquidity of the feces. I feel that we are all guilty of making the mistake of confusing diseases and symptoms.

The problem of diagnosing diseases characterized by diarrhea in cattle is more complicated than in other classes of animals because the forestomachs add another dimension to consider in determining the reason that a cow or a herd of cattle has diarrhea.

Forestomach diseases as a primary cause of diarrhea will not be considered in great depth today because the topic has been well covered at previous meetings of the AABP and due to the time allowed today, we will concentrate on diagnostic problems that occur with diseases of the alimentary tract beyond the abomasum. Inflammations of the stomach are called gastritis and many veterinary texts use the term when describing inflammatory conditions of the rumen, reticulum, omasum and abomasum. This paper will use the term gastritis to mean inflammation of the abomasum only.

Enteritis refers to inflammation of the small intestines and dysentery refers to inflammation of the large intestines. The term "dysentery" usually infers