Leptospirosis in Cattle

Thomas B. Hairgrove, *DVM, Diplomate, ABVP*
Haskell Veterinary Clinic, P.O. Box 723, Haskell, TX 79521

Abstract

Leptospirosis is a disease first described in the literature nearly 70 years ago. In cattle the principal serovars of *Leptospira interrograns* are *L. hardjo-bovis, L. pomona, L. canicola, L. icterhaemorrhagiae, L. grippotyphosa* and *L. szwajjizak*. *L. hardjo-bovis* is the serovar most frequently associated with reproductive wastage in the United States. Of the remaining serovars, *L. pomona* is the most significant. Many aspects of the disease remain poorly understood, e.g.variation in disease pattern and disease impact associated with different strains of the same host-maintained serovor in different management systems and different parts of the world.

This presentation focuses on *L. hardjo-bovis*. Cattle are the maintenance host and the only reservoir. In general, a disease associated with infection of the maintenance host is sub-clinical, produces low antibody titers and affects young or pregnant animals with a very rapid transmission rate from animal to animal. Maintenance host diseases can be very difficult to diagnosis. *L. hardjo-bovis* is currently diagnosed using a combination of serology and identification of leptospires in the urine. A prevalence study indicated there is a high prevalence of *L. hardjo-bovis* in the rolling plains of Texas.

Control of this disease consists of implementing biosecurity measures, use of antibiotics to clear carrier states and use of a vaccine effective for *L. hardjo-bovis*.

Introduction

Leptospirosis is a zoonotic disease caused by members of the genus leptospira. In cattle, the principal serovars of Leptospira interrogans are, *L. hadjo-prajitno, L. pomona, L. canicola, L. icterhaemorrhagiae,* and *L. szwajjizak*. Leptospira *borgpetersenii serovar hardjo* type *hardjo-bovis* and *L. kirschneri* serovar *grippotyphos* are pathogenic leptospiras associated with disease in cattle. *L. hardjo-bovis* is the serovar most frequently associated with abortion in the United States. Of the remaining serovars, *L. ponoma* is the most significant.[1] Our perception of leptospirosis as a disease has undergone significant changes in recent years and there is confusion within the profession. Many aspects of the disease remain poorly understood e.g. variations in disease pattern and disease impact associated with different strains of the same host-maintained serovar in different management systems and in different parts of the world.[2]

This presentation will focus on *L. hardjo-bovis*. Cattle are the maintenance host for *l. hardjo-bovis* and are the only reservoir. *L. hardjo-bovis* is an important cause of abortion in cattle and the commonest leptospiral infection in man.[5] Serovar *L. hardjo-bovis* is the most common serovar of cattle in the UK, Australia, New Zealand and North America.[5]

Two major genotypes of *L. hardjo* are found in cattle and sheep—*L. hardjo- bovis* and *L.hardjo- prajitno*. *L. hardjo- bovis* appears to be a better adapted parasite then *L. hardjo-prajitno*. It is excreted in much larger numbers in cattle urine and is the strain found in most countries. In cattle leptospira may persist for a mean period of 36 days (10 – 118 days), with the highest excretion rate in the first half of the period. Prolonged shedding is observed with *L. hardjo* (mean 215 days);[1] Sometimes shedding may persist for life.[3]

Epidemiology

Leptospirosis is found world-wide, most commonly in warm climates. The epidemiology of leptospirosis is potentially very complicated because animals can be infected by any of the pathogenic serovars. There are a small number of serovars endemic in any particular region, and each serovar tends to be maintained in specific maintenance hosts. An animal may be infected by serovars maintained by its own species (maintenance host infection) or serovars maintained by other animal species (incidental infection) present in the area.

The relative importance of these incidental infections is determined by the opportunities for contact and transmission of leptospires from other species to the target host provided by prevailing social, management and environmental factors.[3] Host adapted (maintenance or reservoir) and non-host adapted (accidental or incidental) leptospirosis is dependent on response of each species to a particular serovar. Serovar Hardjo infection in cattle (cattle are the maintenance host) appears to be largely independent of rainfall and cattle and sheep management.

In general, a disease associated with infection of the maintenance host is sub-clinical, produces low antibody titers and affects young or pregnant animals with

a very rapid transmission rate from animal to animal. Maintenance host diseases can be very difficult to diagnose.[2] Incidental hosts are not important reservoirs for infection and transmission from them is low. Indirect transmission plays a greater role in the transmission of incidental infections. It occurs through the exposure to a contaminated environment and a management system that facilitates close contact between carrier and susceptible animals. The optimum conditions for survival outside the host are warm moist conditions (optimum around 82° F; 28° C) and a ph close to neutral. It can persist in water saturated soil for as long as 183 days, but only 30 minutes when the soil is air dried.[5] Survival is brief in temperatures less than 48° F; (10° C) or more then 93° F (34° C). In West Texas I have observed a higher incidence of *L. pomona* on cattle grazing irrigated fields. There is a high incidence of feral swine in these areas, and swine are the maintenance host for *L. ponoma*. The optimum environment for survival of *L. ponoma* outside the host and the presence of swine which are shedding the leptospires in their urine would account for the higher incidence of incidental infection in West Texas herds. Practitioners in Texas have observed high titers to *L. bratislava* in some herds. While this has not been my experience swine are the maintenance host for *L. bratislava* and in areas where feral swine are prevalent that serovar would need to be ruled out. Transmission of infection among maintenance hosts is efficient and the incidence of infection is relatively high. Direct transmission can occur among animals via infected urine, post abortion uterine discharge, or milk. The infection can be transmitted by the venereal or transplacental route. Environments favorable to the survival of leptospirosis are much less important in the epidemiology of host-maintained leptospires. There is some debate as to immuno suppression being important in the spread of maintenance host infections, but it would be important in the spread of incidental infection. The major factors for maintaining infection in a herd are persistently infected carriers and a regular supply of susceptible animals.

Pathogensis

Infection of susceptible animals occurs through the mucous membranes of the eyes, mouth, nose, vagina, penis and through abraded or water softened skin. There can be rapid introduction into the host. Leptospires introduced in the conjunctiva of a guinea pig can be recovered in the blood in as little time as 15 to 20 minutes.[1] Pathogenic leptospires are found extracellularly between cells of the liver and kidney. Leptospirosis can occur as an acute and severe disease due to septicemia, with evidence of endotoxemia, such as hemorrhages, hepatitis and meningitis, as a moderately severe disease, or as a chronic disease characterized by abortion, stillbirth and infertility. *L. hardjo-bovis* causes endemic rather than sporadic abortions. In cattle, *L. hardjo-bovis* can cause infection in sexually mature, lactating or pregnant females. Infection occurs in the pregnant uterus and lactating gland resulting in abortion, stillbirth or birth of premature and weak infected calves. Infected but clinically normal calves can be born. *L. hardjo-bovis* is associated with a prolonged renal carrier state and may be associated with chronic renal disease. Leptospires which reach the proximal renal tubules, genital tract and mammary gland appear to be protected from circulating antibodies.[5] Leptospires persist and multiply in these sites. The level of serum antibody commonly declines to undetectable levels in persistently infected animals, making a diagnosis sometimes difficult and frustrating.

Clinical Signs

Primary clinical signs of *L. hardjo-bovis* infection in cattle are reproductive wastage, with abortions, stillbirths and weak calves. Unfortunately, most of the time there is no previous clinical evidence of disease in the herd until the onset of reproductive wasting.

In my practice, we have typically observed a low pregnancy rate, as low as 75 % ,in replacement heifers that have reached target breeding weight, have been fed a proper trace mineral supplement and have been on a thorough vaccination program. The first herd in which we diagnosed *L. Hardjo-bovis* infection exhibited 25% wastage due to weak calf syndrome and abortions. This herd is a well managed herd that had experienced bovine viral diarrhea virus (BVDV) infection in past years. In spite of adequate control of the BVD problem through excellent biosecurity measures and an excellent vaccination program, we still noted a large number of weak, premature calves. Samples were collected from dams and calves to detect infectious agents of the reproductive tract, and to evaluate their trace mineral status. Results were negative for active infection by infectious bovine rhinotracheitis virus, BVDV, Cache Valley virus, *Campylobacter fetus, Brucella abortus* and *Neospora caninum.* Concentration of copper and zinc were within normal ranges in serum and liver samples obtained by biopsy. Serum titers for multiple serovars of *Leptospria interrogans* were negative or at insignificant levels. To further investigate the possibility of a leptospira problem, serum and urine samples were collected from dams of two weak calves and sent to Dr. Carole Bolin at Michigan State University. Serology was negative but leptospira organisms were identified in the urine by fluorescent antibody testing. When clinical signs consistent with leptospirosis are present and serology is negative, an identification of leptospira or-

ganisms in properly collected urine is necessary for the diagnosis of *L. hardjo-bovis*.

We have since diagnosed *L. hardjo-bovis* in eight other herds, some experiencing weak calf syndrome, but all exhibiting excessive infertility in first calf heifers. Losses are greatest in younger females the year that infection is diagnosed, and appears to decline in subsequent years.

A milk-drop syndrome is reported in dairy cattle, affecting up to 50% of the cows at one time. There is a sudden onset of fever, anorexia, immobility and agalactia. The milk is yellow to orange and may contain clots. The udder is flabby, there is no heat or pain, and all four quarters are affected.[5] A decline in milk production can last for 2 to 8 weeks. There are reports of mastitis in the literature, but this does not appear to be a consistent finding. One beef herd in our practice where *L. hardjo-bovis* was diagnosed experienced a 10% level of mastitis in cows with their first and second calf. In previous years they had experienced about a 1% level of mastitis in the same age group.

Diagnosis

Incidental infections are usually diagnosed using clinical signs of disease and serology. There is no need for paired serology as used in diagnosing most infectious diseases. If there are clinical signs of leptospirosis and one serovar shows a highly elevated titer (800 or greater), one could diagnose the disease.

The diagnosis of maintenance host infections is much more difficult, as adults do not show clinical signs of disease. The diagnosis is usually based on laboratory findings. Recommended tissues to send to the laboratory are fetus and placenta (hopefully fresh with minimum autolysis), kidney, liver and thoracsic fluid from the fetus, and urine and serum from dam. Laboratory tests fall into two categories, tests for the demonstration of leptospires and tests for antibody. Most tests for the demonstration of leptospires in the urine or tissue are not serovar specific. The only definitive test that is serovar specific is culture, which is expensive and time consuming. The current testing protocol to diagnose *L. hardjo-bovis* relies on serology and the demonstration of the organism in urine. If the animal is negative on serology for all serovars and leptospires are found in the urine a presumptive diagnosis of *L. hardjo-bovis* is made. There has been some discussion about the possibility of the organism found in the urine being a non-pathogenic leptospire. Leptospira organisms found in the urine of animals with clinical syndromes typical of leptospirosis are considered to be pathogenic. There is a possibility of the urine specimen being contaminated with water containing a non-pathogenic leptospire, therefore attention to detail in taking the urine sample and laboratory handling are important. Current tests include darkfield microscopy, immuno-florescence, culture, histopathology with special stains, and polymerase chain reaction (PCR) assay. Immuno-florescence and PCR are most widely used. The current popular protocol involves collection of urine from cattle after injecting furosemide. Furosemide increases the glomeralar filtration rate, flushes more leptospires into the urine and produces dilute urine which enhances survival of the organism. I have submitted several samples to be tested by both PCR and FA and have found FA to be a more accurate test.

Control and Prevention

I recommend a control program to the majority of my clients because the prevalence of *L. hardjo-bovis* in the rolling plains of west Texas appears to be high. We participated in a prevalence study in which we were asked to submit 10 herds, of which four were randomly chosen to collect urine and serum. Three of the four herds were infected. We had diagnosed *L. hardjo-bovis* in two larger herds and several smaller herds prior to the study.

A control program is centered on elimination of the carrier state and vaccination to prevent new infections. The carrier state can be eliminated by treatment with long-acting oxytetracycline at the standard recommended dose. There is good evidence to show that the carrier state in females can be eliminated but there is some question concerning bulls. New infections are prevented by a vaccination program, consisting of primer and booster doses the first year, and then annual boosters. Vaccination with the current multivalent vaccines on the market in this country does not appear to provide protection against *L. hardjo-bovis*. A new monovalent *L. hardjo-bovis* effective for preventing infection has recently been introduced in the United States.[2] One should not recommend vaccinating bulls that may be exported or go to semen collecting stations. The current vaccine does cause a humoral response which could confuse serology results needed for export testing. I recommend treating breeding females (especially first and second calf heifers) with oxytetracyline when administering the first vaccination. I also recommend vaccinating all calves at branding and administering oxytetracyline at that time to eliminate the carrier state.

Prevention is accomplished by the above practices plus biosecurity. Biosecurity consists of strongly recommending that clients purchase replacement animals from herds on a good vaccination and management program, treatment of all purchased animals with long acting oxytetracycline and implementation of a vaccination program.

The first description of bovine leptospirosis in the medical literature is contained in a report of spirochetal jaundice of cattle in Russia in 1935.[4] *L. pomona* was thought for decades to be the serovar most frequently associated with bovine leptospirosis. As practitioners, we assumed the vaccines were effective if given frequently. The concept of host maintenance infection is difficult for both clients and veterinarians alike to understand and there are still unanswered questions. This is a disease that is sometimes difficult and expensive to diagnose. The Standard Performance Analysis (SPA) data for the Rolling Plains of Texas indicates only about 83% of exposed cows wean a calf. Could some of this loss be due to *L. hardjo-bovis*?

Footnotes

[a]Bolin, C.A. April 6, 2004 Dallas, Texas.
[b]Spirovac, Pfizer Animal Health, Pfizer Inc, New York, NY 10017

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