Bovine Leptospirosis

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Bovine Leptospirosis

Richard Larsen, B.S.*

Bovine leptospirosis is indeed a broad subject. One could unquestionably delve deeper into most every aspect than this review does. The author felt the need for an article written toward the needs of a potential practitioner, hence the review.

GENERAL

Leptospires are highly motile, filamentous bodies which appear beaded due to twisting. An axial filament runs through the bacterium, both ends of which are bent. They measure 8-12 microns in length and .1-.2 microns in thickness.

Different species of leptospires cannot be distinguished morphologically or metabolically. They fail to ferment test media. Leptospires can be classified into the various species only by their antigenic structure using the agglutination lysis test. Presence of 10% or more of the serum titer following adsorption with known heterologous serum strains indicates the strain belongs to a different species. (15)

Cultivation can be made in liquid, semi-solid, and solid media containing 10-15% serum. Temperatures of 28-29°C are best, since temperatures above 30°C activate and kill the leptospires. They are quite sensitive to acid or base pH and have been found difficult to grow in the laboratory. (15)

Heat, sunlight, desiccation, disinfectants, acids, and bases easily destroy leptospires. Milk will lyse leptospires, accounting possibly for the difficulty noted in attempts to isolate them from the milk of infected cows. They have been known to survive up to six hours in urine. Leptospires, however, survive quite a range of environments. Moist and moderate temperatures are best, such as surface water and water saturated soil. (15)

ETIOLOGY

Bovine leptospirosis may be due to Leptospira pomona, L. canicola, L. hardjo, or L. grippotyphosa according to isolations which have been made. (15) (47) (10) (9) L. pomona, however, is responsible for about 98% of the leptospirosis in cattle and swine. (18) L. sejroe and L. icterohemorrhagiae have been found serologically. The causative agents of bovine leptospirosis show no different growth requirements, resistance or structure than those included above for the genus Leptospira.

EPIZOOTIOLOGY

The following are results of serological surveys taken around the United States for L. pomona infection. (18) (31) (32)

<table>
<thead>
<tr>
<th>State</th>
<th>% Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alabama</td>
<td>7.00</td>
</tr>
<tr>
<td>Indiana</td>
<td>7.40</td>
</tr>
<tr>
<td>Illinois</td>
<td>11.20</td>
</tr>
<tr>
<td>Maryland</td>
<td>25.00</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>.83</td>
</tr>
<tr>
<td>Ohio</td>
<td>4.10</td>
</tr>
<tr>
<td>Washington</td>
<td>12.00</td>
</tr>
<tr>
<td>Wyoming</td>
<td>.20</td>
</tr>
</tbody>
</table>

* Mr. Larsen is a senior in the College of Veterinary Medicine.
Beef cattle seem to be more severely affected, their mortality rate being higher than that of dairy cattle. (25) Most outbreaks are seen in late summer and fall, disappearing with the freezing of ponds and insects as winter approaches. Infections may be seen any time of the year, however. All ages and either sex are equally susceptible.

Infected wild animals are an important reservoir for leptospirosis in cattle and vice-versa. (25) At this time no accurate conclusions can be drawn about the relative importance of the various wild animal species as regards their part in the incidence of bovine leptospirosis. Deer in one Wisconsin area showed a high titer for *L. pomona* as did the cattle in the same area.

**TRANSMISSION**

The usual method of transmission of the leptospires is via the urine. They can survive up to six hours in urine. (15) Moist soil, ponds or slow moving streams with a temperature of 22° C.—can keep them viable for several weeks. (37)

Coition and artificial insemination can spread the disease from an infected bull to a susceptible female. (34) Aborted fetuses from a known infected animal can be a source of infection although most attempts at isolation of the leptospires fail in such cases. (15)

**SYMPTOMS**

The disease may roughly be divided into three types on the basis of symptoms shown:

**Acute type:**
This type is most often seen in calves, but may be seen in adults. They show a high temperature, prostration, diarrhea, dehydration, marked hemoglobinuria, anemia, bile pigments in the urine and marked icterus. The course is rapid and the mortality high. (35)

**Subacute type:**
Anorexia, a rapid decrease in milk production to a point where the udder feels as if the animal is not lactating and slight temperature elevation is seen. Slight hemoglobinuria occurs, often accompanied by marked albuminuria.

**Inapparent type:**
Usually no symptoms other than a transitory feverish state are seen. It can be detected by the presence of leptospires in the urine and by a high titer serologically.

Abortion follows the acute and subacute types of infection in about 25-50% of the cases and usually in those animals which are in the last ½ of pregnancy. (15) (1)

The reduction in milk production followed by production of thick and bloody milk is due to mastitis or a severe systemic infection. It is not clear at the present time which it is. Mitchell et al reports an atypical case of mastitis due to *L. sejroe*. (24)

Stoenner et al described a cow with typical symptoms of leptospirosis which also had obvious nervous problems. The animal showed lateral head and neck retraction, cricking, salivation, and spasms of muscle groups, paddling and opisthotonos. At necropsy the brain and meninges are essentially normal. It was concluded that the symptoms were due to the systemic effects of the infection and/or a rapidly developing diffuse hepatic damage. (38)

**PATHOGENESIS**

The leptospires enter the abraded skin of the feet and legs, most frequently when wading, or enter the eroded mucous membranes of the mouth, eyes or nose. (15) It is felt they cannot enter the unbroken skin or mucous membrane. A leptospiremia develops in 4-8 days—several days before the fever, but both ending about the same time. (43) The organism can be isolated from the blood from the fever peak to the appearance of agglutinins. (15)

Antibodies can be detected an average of 10 days following infection. They reach a peak by the second to the third week and may remain at a moderate level for months to years. (43)

During the acute infection leptospires are shed in the urine. After apparent cli-
cal recovery, infection remains localized in the kidney and chronic shedding of leptospires in the urine may occur for eleven days to three months. (43)

Bauer et al have precipitated and concentrated a haemolysin with \((NH_4)_2 SO_4\) from culture filtrates of *L. pomona*. This haemolysin caused the typical symptoms of leptospirosis in susceptible sheep. Animals with agglutinin titers were protected from the haemolysin. (2)

The exact pathogenesis of abortion following acute infection is not clear at this time. Sleight and Langham injected the concentrated haemolysin, mentioned above, into pregnant females and concluded that it did not cross the placental barrier. Abortion was blamed on a lowered oxygen carrying capacity of the blood which in turn caused placental metabolic disturbances. Stafseth caused fetal death by injecting leptospires into the pregnant uterus. The placenta was altered, but the uterus was not invaded. (36) Fennestad and Borg-Peterson suggest: 1. Abortion is due to fetal death from leptospirosis. The two to five week interval can be explained by two consecutive infections; first, in the mother, and following, in the fetus. 2. The interval between fetal death and expulsion often exceeds the survival time of leptospires, accounting for the difficulty in isolation. 3. Occasionally the fetus is able to produce antibodies and survives. (13) Possibly abortion results from more than one cause, i.e., fetal leptospirosis, alteration of the placenta and/or placental metabolic deficiencies.

Lingard et al did not find any significant reduction in fertility following *L. pomona* infection. (23)

Prominent macroscopic lesions on necropsy include icterus, petechia on the kidneys, yellow-brown liver and subepicardial hemorrhages along the coronary arteries. Microscopically in acute cases there is necrosis of kidney tubular epithelium in the cortex and in subacute cases, proliferation of undifferentiated lining cells and progressive interstitial lymphocytic infiltration. Renal carriers usually show no lesions, but the organism can be found at the cortico-medullary junction. (32)

**DIAGNOSIS**

1. The clinical signs must be considered only suggestive since so many diseases are of similar nature.

2. Demonstration of the organism.

It is possible to show leptospires by dark field examination of the blood if taken at the right time, in urine and in tissue suspensions taken fresh from fatal cases. Mistakes are easily made; therefore, it should be regarded as a tentative diagnosis.

Silver stains of tissues, especially liver, from fatal cases may show the organism.

Boylanger and Robertson have described a fluorescein-labeled antibody technique which was not better than culturing. (7) Later Coffin and Maestrone described a similar technique superior to cultures and dark field. They were able to demonstrate the leptospires in fresh and preserved urinary sediment, scrapings from the cut surface of an infected kidney and in sections from organs. (11)

3. Serological diagnosis.

The Galton and Stoenner macroscopic plate agglutination tests are useful only for screening sera. No titer is found. The microscopic agglutination lysis (AL) test using a live antigen and dark field examination of the results is a very useful test. Cabrey stressed the need for adoption of a standard method of performing the (AL) test. Different dilution schemes, antigenic strains and test readers effectively alter serum titers. (42) A capillary tube agglutination test and a milk agglutination test have been described. (43) The agglutination tests are species specific, hence only the species included in the antigen are tested for. With numerous species of leptospires that may cause disease in cattle, this may be a disadvantage.

A genus specific complement fixation test can be used but is laborious.

Cox described a hemolytic test (HL) using a leptospiral extract which was able to sensitize sheep erythrocytes to the hemolytic effects of antileptospiral serum and complement. It compared favorably to the AL test and is also genus specific.

*Iowa State University Veterinarian*
(12) Ringen conducted a similar test for leptospiral antigen in bovine urine. He isolated an antigenic substance (ESS) from the urine of an infected animal which would sensitize sheep erythrocytes to the hemolytic effects of antileptospiral serum and complement.(27)

Blendon et al, using a protein fraction of L. pomona, found a specific skin reaction when the fraction was injected subcutaneously in a sensitized guinea pig. Erythema, edema, and necrosis appeared in a few hours. He found the fraction stable and potentially useful for the diagnosis of leptospirosis.(5)

One should be cautious so as not to be misled by a positive serological finding for leptospirosis. Since a moderately high level of antibody may persist for years, a positive test result may actually mean only that the animal was once infected. Rather, use the clinical signs plus a rising antibody titer over a period of one to two weeks to make a positive diagnosis. Negative tests for other diseases may be especially helpful. Death may occur in calves before antibodies appear.

4. Bacteriological methods.(43)

These offer the most conclusive proof for the presence of leptospiroses.

Blood collected aseptically during the period of the leptospiremia is inoculated into a series of Fletcher’s and Stuart’s tubes using varying dilutions of inoculum, and incubate at 28–30° C. in a dark place. Check every 7–14 days for growth, generally allowing about six weeks.(8)

Tissue suspensions taken aseptically from the liver and the spleen should be suspended into a 10% solution in Stuart’s medium and inoculated, using a dilution technique, into tubes of Fletcher’s and Stuart’s medium. Incubate as described above.

Urine may be used. Collect aseptically by bladder tap, dilute 1:10 in Stuart’s medium, make dilution of this inoculum and inoculate tubes of Fletcher’s and Stuart’s medium. Incubate as above.

Leptospires are more sensitive to pH than most bacteria. Extreme care must be taken, water and soap must not change the pH.

Indirect isolation of leptospires using guinea pigs or chinchillas may be helpful. The animals should be inoculated, watched for a temperature rise, have blood cultured on days 4, 6, and 8 and be killed and have tissue collected at 21 days for the culture.

TREATMENT

Tetracycline antibiotics in the feed will prevent the disease, reduce abortion, prevent leptospiruria in the cattle and aid in prevention of complication from retained placental membranes. A toxemia secondary to retained placental membranes is quite common.(25)

Dihydrostreptomycin is probably the best drug to eliminate the carrier state although other antibiotics can be used for treatment, including penicillin. The sulfonamides are of no value in the treatment of leptospirosis. Treatment must be started early, before extensive kidney damage occurs.(46) (18) (9)

PREVENTION AND CONTROL

Leptospirosis is perpetuated by domestic animal carriers, wildlife, poor husbandry practices, environmental conditions, and virulence of the organism. Swine shed organisms for long periods and in great numbers, therefore remain the number one carrier of bovine leptospirosis. Arthropods may spread the organism from one animal to another.(25)

A system of control and prevention may include one or more of the following: (35) (46) (4) (20)

1. Serologically test the whole herd. Immunize the negative once a year and treat the positives with streptomycin, 5 mg/lb b.i.d. for three days to eliminate the carrier state.
2. Isolate the positives until treatment is complete.
3. Treat with broad spectrum antibiotics and isolate clinical and exposed cases.
4. Purchase only negative animals and vaccinate them two weeks before adding to the herd.
5. Control surface waters to prevent contamination. Drain or limit access.
7. Reduce stress on shipped animals, i.e., use tranquilizers and antibiotics be-
fore shipping, provide good nutrition and sufficient vitamin A.

8. Promote good sanitary practices.

**IMMUNITY**

Immunity following clinical disease may last up to three years or more. Calves born to these recovered animals are solidly immune for one to two months.

(20) Their titer and that of the colostrum will often be three to ten times higher than the mother’s humeral antibody.(46)

There is considerable variation in the antigenicity of present day bacterins and the ability to provide protection more or less parallels the antigenicity.(28) Vaccination with *L. pomona* bacterin does not raise the blood titer significantly although protection is afforded. Vaccinated animals do not produce as great a titer after exposure or challenge as do non-vaccinated. Also, vaccination does not prevent sub-clinical infections and leptospiruria.(20)

Laurie states that vaccination will arrest many natural infections before excretion of leptospires occur.(22)

Bacterins will produce a satisfactory immunity in ten to fourteen days. Kenzy et al., Kenzy and Gilliespie, and Keisel et al report the use of a living egg passage (EP) attenuated strain of *L. pomona* which will produce agglutinins in four days and protect from challenge up to fifty-four months. Kenzy states it is three times more effective in reducing abortion than bacterins. No evidence of transmission under experimental conditions was found.

Anaphylactic reactions following repeated yearly *L. pomona* vaccinations are commonly seen by practitioners. At present there seems to be no agreement as to its cause or prevention. Schuchardt et al (1961) implicated rabbit serum as the cause of anaphylaxis. A rabbit serum-free bacterin did not produce anaphylaxis in guinea pigs or in over 2,000 cattle. Bechenhaus (1962) produced a refined concentrated *L. pomona* bacterin which did not cause anaphylactic reactions in laboratory and field trials while standard bacterins did. Morter et al (1962) produced acute anaphylaxis with serum-free bacterins, thus strongly suggesting that the cellular components were the cause of anaphylaxis. Perhaps further research is necessary to clarify the anaphylaxis problem subsequent to the *L. pomona* immunization.

**PUBLIC HEALTH**

All pathogenic leptospires apparently pose a public health hazard. (47) Just what role the bovine plays in human infection is not clear. Paul et al reported on two surveys made. Two of a group of eighty-six and one in a group of 155 humans exposed to infected animals had a titer for *L. pomona*.

Steel et al states that no human cases have resulted from drinking milk from infected cows. Whole milk is leptosporidical, therefore there is no danger to humans.

(18) We must admit there is danger to the veterinarian. The organisms causing disease in the bovine species are pathogenic for man. These facts should promote our caution.

**BOVINE LEPTOSPIROSIS**

Selected References 1959-1963


Transmissible Gastroenteritis

Jeptha F. Randolph*

INTRODUCTION:

The occurrence of transmissible gastroenteritis (TGE) appears to be worldwide wherever swine are raised. This disease has been reported in Denmark, (17) England, (12, 13, 14) Germany, (20) Japan, (25) Canada, (17, 24) Holland, (17) and the United States. (9) During the early 1930's reports began to appear in the literature regarding a disease condition in baby pigs which caused an extremely high rate of mortality. Since most deaths occurred on about the third day after birth, the condition was known as "three-day pig disease." (26) In 1937, an outbreak was reported in central Minnesota involving 23 herds of swine where death losses were unusually heavy in pigs less than ten days of age. The etiology was not known but some farmers believed that a toxic product must be present in the sow's milk due to the fact that vomition was a fairly constant symptom. (26)

The story of TGE as a specific disease entity actually began in 1946 when two veterinarians (9) reported sporadic outbreaks of a disease characterized by diarr-

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