

A Review of Bovine Leptospirosis

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Abstract: Leptospirosis is caused by bacteria of genus *Leptospira*. It is a potentially zoonotic disease and affects humans and animals and has worldwide occurrence. The source of infection is an infected animal which contaminates the premises by infective urine, aborted fetuses and uterine discharges. Risk factors like animal risk factors and environmental and management factors affect the relationship between host and agent. It has major economic impact in farm animals which is associated with abortion, stillbirth and birth of weak neonates, with a high death rate. The clinical signs may be acute, subacute, or chronic and is usually associated with two serovars, Pomona or Hardjo. The most prominent post mortem findings with leptospirosis are anemia and icterus. A variety of serological tests can be used for diagnosis of leptospirosis such as ELISA and Immunofluorescence; however, the microscopic agglutination test is still the most widely used as reference method in the determination of antibody titer. Many antimicrobial drugs are effective for the treatment. Control methods are useful to avoid and eradicate this disease, which includes biosecurity and biocontainment, occupational hygiene and immunization.

Key words: Cattle • Leptospirosis • Risk Factor • Zoonosis

INTRODUCTION

Leptospirosis is a worldwide zoonotic disease of domestic animals and wildlife. It is caused by a spirochete bacteria classified under the *Leptospira*, of which there are approximately 17 species. The term *Leptospira interrogans* is used to describe the broad group of pathogenic leptospires associated with animal hosts. Serovars are primary grouping below the species designations [1].

The same disease processes are seen in all animals although some species are more resistant to acute infections. Infections may be asymptomatic or cause various signs, including fever, icterus, haemoglobinuria, renal failure, infertility, abortion and death. After acute infection, leptospires frequently localize in the kidneys or reproductive organs and are shed in the urine, sometimes in large numbers for months or years, especially with host adapted serovars. Because the organisms survive in surface waters, such as swamps and rivers, for extended periods, the disease is often waterborne. The organism survives well in mud and moist soil, such river banks, floods frequently result in an increase of disease outbreaks [2]. Infection is commonly acquired by contact

of skin or mucus membranes with urine and to a lesser extent, by intake of urine-contaminated feed or water. Ingestion of infected animals and venereal transmission can also be routes of infection. Infections can be readily established via the conjunctiva, vaginal mucosa, or skin abrasions. Disease outbreaks in small herds are often self-limiting. However, control of enzootic infections in large herds generally requires immunization, chemotherapy and fencing the herd from surface waters and limiting contact with rodents and others wildlife [1].

The microscopic agglutination test (MAT) is the commonly used serological test for diagnosis. It measures both IgM and IgG antibodies. An enzyme linked immunosorbent assay (ELISA) measures IgM and IgG is helpful in distinguishing titers due to natural infections from those due to vaccination. Demonstration of leptospires in urine or tissues is helpful in diagnosis. Older techniques like dark field microscopy of urine are not sensitive or specific. Newer tests include fluorescent antibody techniques and polymerase chain reaction (PCR) [3]. Definitive confirmation of leptospirosis is made by isolation of the organism from urine or tissues of infected animals. However, because leptospires are not easily cultured, isolation is not usually performed in clinical

cases. Humans are susceptible for all pathogenic serovars found in domestic animals and transmission from wildlife generally occurs after contact with tissues of infected animals or surface waters contaminated by urine from infected animals. Because of these origins, the disease in humans often is occupationally or recreationally related [4].

Therefore the aims of this review are to provide updated information on leptospirosis in cattle and to aware the public on its zoonotic importance.

Leptospirosis in Cattle

Etiology and Characteristics: *Leptospira* species belongs to the family Leptospiraceae. Members of this species are motile helical bacteria with hook shaped ends [5], obligate aerobes and use fatty acids or alcohols and carbon as energy source [2]. Although cytochemically Gram negative, they do not stain well with conventional bacteriological dyes and are usually visualized using dark-field microscopy. Formerly leptospire were differentiated by serological reactions and two species were recognized, *L. interrogans* containing pathogens and *L. biflexa* containing saprophytes. Leptospiral species (genospecies) are now classified by DNA homology and within each species, various serovars are recognized on the basis of serological reactions [6].

Currently more than 250 serovars in 23 serogroups are defined. Differentiation between serovars, formerly serotypes, belonging to a particular serogroup is by cross-agglutination tests. Two strains are considered different if, after cross-absorption with adequate amounts of heterologous antigen, 10% or more of the heterologous titer regularly remains in either of the two antisera. Because this system is subjective, the restriction endonuclease analyses (REA) of leptospiral DNA is used as a genotyping taxonomic tool [7] which takes less time and is less labor consuming than cross agglutination absorption and gives highly reproducible results.

Epidemiology

Occurrence: Leptospire are worldwide, the infection occurs in approximately 160 mammalian species [8]. Although found worldwide, some serovars appear to have a limited geographical distribution. In addition, most serovars are associated with a particular host species, their maintenance host. Most leptospiral infections are subclinical [6] which is more common than clinical disease. *L. Pomona* is the commonest infection in all farm animals but its international distribution is unpredictable; it had not been present in the United Kingdom until recent

years and then only sporadically. *L. Canicola*, *L. haemorrhagiae*, *L. hyos*, *L. grippotyphosa*, *L. sejroae*, *L. hebdomadis* and *L. australis* are serovars that infect cattle. Serological survey of cattle in the African continent revealed evidence of antibodies against numerous leptospiral serovars. In West Africa serosurvey of dairy herds revealed 45% of cattle were positive to one or more serovars, which probably represented natural infection because vaccination had not been, practiced [9].

In general outbreaks are caused by exposure to water contaminated by the urine of infected animals. Several occupational groups are particularly at risk, such as workers in rice fields, sugar cane plantations, mines, sewer systems and slaughter houses, as well as animal care takers, veterinarians and members of the military [10].

Methods of Transmission: The source of infection is an infected animal which contaminates pasture, drinking water and feed by infective urine, aborted fetuses and uterine discharges. All of the leptospiral types are transmitted within and between species in this way. A viable infected neonate can harbor the infection for several weeks after birth. The semen of an infected bull may contain leptospirae and transmission by natural breeding or artificial insemination can occur but is uncommon [11]. *L. hardjo* is excreted from the genital tract of aborting cows for as long as 8 days after abortion or calving and is detectable in oviduct and uterus for up to 90 days after experimental infection and in naturally infected cows. Infection of the genital tract may indicate the possibility of sexual transmission [12].

Leptospiuria: Urine is the chief source of contamination because animals, even after clinical recovery, may shed leptospirae in the urine for long periods. All animals which have recovered from infection may intermittently shed organisms in the urines and act as “carriers”.

Wildlife as Source of Infection: Although survey of the incidence of leptospirosis in wildlife has been conducted and the pathogenic effects of *L. Pomona* on some species, particularly deer and skunks have been determined, the significance of wildlife as a source of infection for domestic animals is uncertain. Variable rates of seroprevalence to leptospire have been documented in white-tailed deer, pronghorns, red deer and elk [13]. There is also a report in which insect vectors, for example ticks, transmit and disseminate the micro-organism among cattle, or from, for example, hedgehogs to cattle. It is likely that flies would act as mechanical vectors by

picking up the micro-organisms from urine or other infected material and depositing it on exposed mucosa or skin wound [14].

Risk Factors

Animal Risk Factors: The epidemiology of leptospirosis is most easily understood by classifying the disease in to two broad categories: host-adapted and non-host-adapted leptospirosis. An animal infected with a host-adapted serovars of the organism, is a 'maintenance' or 'reservoir' host. Exposure of susceptible animals to non-host-adapted serovars results in accidental or incidental disease. Each serovars is adapted to a particular maintenance host, although they may cause disease in any mammalian species. Cattle are maintenance hosts for *L. borgpetersenii* serovar *hardjo* [15].

Environmental and Managerial Risk Factors: Survival of the organism in the environment depends largely upon variations in the soil and water conditions in the contaminated area. The organism is susceptible to drying and $P^H < 6$ or > 8 is inhibitory. Ambient temperatures lower than 7-10 °C (44-50 °F) or higher than 34-36 °C (93-96 °F) are determined to its survival. Low urinary P^H in cattle feed with brewer's grains may inactivate leptospirosis in animal leptospirosis [16].

Ground surface moisture and water are the important factors governing the persistence of the organism in bedding or soil, it can persist as long as 183 days in water saturated soil but survives for only 30 minutes when the soil is air dried. In soil under average conditions, survival is likely to be at least 47 days for *L. pomona*. Contamination of the environment and capacity of the organism to survive for longer periods under favorable conditions of dampness may result in a high incidence of the disease on heavily irrigated pasture, in areas of high rainfall and temperate climate, in field with drinking water supplies in the form of easily contaminated surface bonds and in marshy fields and muddy paddocks or feedlots. *L. hardjo* antibodies have a high prevalence through all rainfall areas, but *L. pomona* is much more common in low rainfall areas in Australia [17].

Certain management risk factors have been identified which pose risk of *L. hardjo* infection being introduced in to dairy herds such as: purchase of infected cattle, co-grazing or common grazing with infected sheep, purchase of loan of an infected bull and access of cattle to contaminated water supplies such as streams, rivers, flood and drainage water [18].

Economic Importance: Leptospirosis is the major economic loss in farm animals. The majority of leptospiral infections is subclinical and associated with fetal infections causing abortion, still births, infertility and increased culling rate causing major economic loss. Epidemics of agalactiae in dairy cattle, 'the milk drop syndrome', are associated with infection with *L. hardjo*. Exposure of non-vaccinated dairy cows to *L. hardjo* can be associated with a subsequent reduction in fertility, as indicated by a greater time from calving to conception and a high number of breeding per conception [19]. The cost of various control strategies and financial losses associated with disease are also considered [17].

Zoonotic Implication: In the past decades, leptospirosis has emerged as a globally important infectious disease in human medicine. It is an important zoonotic disease worldwide [20].

It is uncommon in developing countries but the incidence is increasing in travelers returning from endemic countries. The epidemiology has undergone major changes, with a shift away from the traditional occupational disease in developed countries, to a disease associated with recreational exposure [4].

It is now recognized as an emerging, potentially epidemic disease associated with excess rainfall in tropical settings, representing a significant public health hazard. Mortality in humans with leptospirosis remains significant because of delay in diagnosis due to lack of diagnostic infrastructure and adequate clinical suspicion when patients are presented for medical diagnosis and care. Pulmonary hemorrhage is increasingly being recognized as a major, often lethal, manifestation of leptospirosis in humans, the pathogenesis of which is unclear.

Leptospirosis is an important zoonosis and is an occupational hazard to butchers, farmers and veterinarians [21]. Human infection is most likely to occur by contamination with infected urine or uterine contents. Although leptospirae may be present in cow's milk for a few days at the peak of fever in acute cases, the bacteria do not survive for long in the milk and are destroyed by pasteurization. However, farm workers who milk cows are highly susceptible to serovars *hardjo* infections and one New Zealand survey found 34% of milkers were seropositive, mostly to *hardjo*, but a high proportion also to *Pomona*. The risk of transmission of leptospirosis from dairy cattle infected with *L. hardjo* to dairy workers in Israel was low [22].

Veterinary students may be exposed to leptospirosis by taking courses in food inspection and technology on farm clinical experience, contact with pets especially carnivore and contact with animal traders. In humans the disease causes a wide range of symptoms and some infected persons may have no symptoms at all. Symptoms of leptospirosis include high fever, severe headache, chills, muscle aches and vomiting and may include jaundice, red eyes, abdominal pain, diarrhea, or a rash. If the disease is not treated the patient could develop kidney damage, meningitis, liver failure and respiratory distress. The disease can be prevented through appropriate hygiene, sanitation and husbandry [23].

Pathogenesis and Pathogenicity: Leptospire invade tissues through moist, softened skin or through mucous membranes; motility may aid tissue invasion. They have the ability to bind to epithelial cells and attach to the constituents of the extracellular matrix through an active process involving surface proteins. Pathogenic leptospire are found extracellular between cells of the liver and kidney. Release of lymphokines such as TNF-alpha from monocytes through the endotoxic activity of the leptospiral LPS may be an important virulence mechanism. Induction of TNF-alpha release may help explain the damage to endothelial cells with resultant haemorrhage seen in severe leptospirosis [24]. There is evidence that leptospiral chemotaxis for haemoglobin may be involved in the initiation of infection. Leptospire can evade phagocytes in the blood stream, possibly by inducing macrophage apoptosis [25]. Leptospirosis can occur as an acute and severe disease due to septicemia with evidence of endotoxaemia such as haemorrhages, hepatitis, nephritis, meningitis as a subacute; moderately severe disease with nephritis, hepatitis,agalactia and meningitis, or as a chronic disease characterized by abortion, still birth and infertility. In the occult forms there is no clinical illness. Variations between serotypes of *L. interrogans* in their pathogenicity also affect the nature of the signs which appear. For example, in *L. pomona* infections, intravascular hemolysis and interstitial nephritis are important parts of the disease. The pathogenesis of the disease associated with *L. pomona* is set out as follows:

Acute Form: After penetration of the skin or mucosa, the organisms multiply in the liver and migrate to and can be isolated from, the peripheral blood for several days until

the accompanying fever subsides. At this time serum antibodies begin to appear and organisms can be found in the urine [24].

Septicaemia, Capillary Damage, Hemolysis and Interstitial Nephritis: During the early periods of septicaemia, hemolysin may be produced to cause overt haemoglobinuria as a result of extensive intravascular hemolysis. This is an unlikely event in adult cattle, but is common in young calves. If the animal survives this phase of the disease, localization of the infection may occur in the kidney. Hemolysis depends on the presence of a serovar which produces hemolysin. Capillary damage is common to all serovars and during the septicaemic phase, petechial haemorrhages in mucosae are common. Vascular injury also occurs in the kidney and if the hemolysis is severe, anemic anoxia and haemoglobinuric nephrosis may occur. There is some evidence that leptospire produce a lipopolysaccharide endotoxin which may exacerbate the vascular lesions [26].

Abortion: Following systemic invasion, abortion may occur due to death, with or without placental degeneration. Abortion usually occurs, several weeks after septicaemia because of the time required to produce the changes in the fetus, which is usually autolysed at birth. Abortion occurs most commonly in the second half of pregnancy, due probably to the greater ease of invasion of the placenta at this stage, but may occur at any time from 4 months on. Although abortion occurs commonly in cattle after either the acute or the subacute form of the disease, abortion without prior clinical illness also occurs in a less extent; this may be due to degenerative changes in the placental epithelium [27].

Subacute and Occult Form: In the subacute form, the pathogenesis is similar to that of the acute septicaemic form, except that the reaction is less severe. It occurs in all species, but is the common form in adult cattle. Occult cases, with no clinical illness but with rising antibody titers, are common. These are difficult to explain but may be associated with strains of varying pathogenicity. But with leptospirosis, characteristically, differences between groups may be associated with prior immune status, environmental conditions, or number of carriers in relation to severity of exposure [24].

Clinical Findings: The clinical findings in leptospirosis are similar in each animal species and do not vary greatly with the species of *Leptospira* except that infection with *icterohaemorrhagiae* usually causes a severe septicaemia. The incubation period varies between 1-2 weeks [2]. Leptospirosis in cattle may be acute, subacute, or chronic and is usually associated with *Pomona* or *Hardjo*.

Acute Leptospirosis Associated with *L. Pomona*: Calves up to one month old are more susceptible to acute leptospirosis. The disease is manifested by septicaemia, with high fever (40.5-41.5 °C; 105-107 °F), anorexia, depression and acute haemolytic anemia with haemoglobinuria, jaundice and pallor of the mucosae [24]. Because of the anemia, tachycardia, loud heart sounds and more readily palpable apex bear are present: dyspnea is also prominent. The case fatality rate is high and if recovery occurs, convalescence is prolonged. In adult cattle, abortion due to the systemic reaction may occur at the acute stage of the disease. Milk production markedly decreased and the secretion is red-coloured or contains blood clots and the udder is limp and soft. After the acute course of leptospirosis, petechiae are found on the epicardium and the lymph nodes [28].

Subacute Leptospirosis Associated with *L. Pomona*: The subacute form of leptospirosis differs from the acute form only in degree. Similar clinical findings are observed in a number of affected animals but not all of the findings are present in the same animals. The fever is milder (39-40.5 °C; 102-105 °F) and depression, anorexia, dyspnea and haemoglobinuria are common but jaundice may or may not be present. Abortion usually occurs 3-4 weeks later. One of the characteristic findings is the marked drop in milk production and appearance of blood stained or yellow orange, thick milk in all four quarters without apparent physical change in the udder [27].

Chronic Leptospirosis Associated with *L. Pomona*: The clinical findings in the chronic form of leptospirosis are mild and may be restricted to abortion. Severe 'storms' of abortion occur most commonly in groups of cattle which are at the same stage of pregnancy when they are exposed to infection. The abortions usually occur during the last trimester of pregnancy. Apart from the abortions, there is no depression of reproductive efficiency in cattle affected by leptospirosis. Many animals in the group develop positive agglutination titers without clinical illness. There

are occasional reports of leptospiral meningitis in cattle. Incoordination, excessive salivation, conjunctival and muscular rigidity are the common signs [24].

Leptospirosis Associated with *L. Hardjo*: Infertility and milk drop syndrome occurs only in pregnant or lactating cows because the organism is restricted to proliferation in the pregnant uterus and the lactating mammary gland. 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 equally affected. The sudden drop in milk production may affect up to 50% of cows at one time and cause a precipitate fall in the herds milk yield. The decline may last for up to 8 weeks but individual cow's milk production will return to normal within 10-14 days because humoral protective antibodies are developed [2]. In some cases, there is no evidence of mastitis, no change in consistency of the milk and no changes in the udder of affected cause, but leptospiruria may be present in up to 30% of affected cows. In endemically infected dairy herds, there may be no relationship between seropositive and seronegative cows in different lactations, or at different stages of lactation and total lactation milk yield [29].

The herd fertility status incorporating the first service conception rate, the number of services per conception for cases conceiving, the calving-to-conception interval and the culling rate usually reveal a low reproductive performance, especially during the years of the diagnosis. Exposure of non-vaccinated dairy cows to *L. hardjo* can be associated with a subsequent reduction in fertility as indicated by a greater time from calving to conception and a higher number of breeding's per conception [19]. Abortion may occur several weeks or months after infection and may also occur as the only evidence of the disease; in some areas or circumstances it is the principal manifestation of leptospirosis due to *L. hardjo* and the principal cause of abortion in cattle [24].

Necropsy Findings: Acute bovine leptospirosis is characterized by anemia, jaundice, haemoglobinuria and subserosal haemorrhages. There may be ulcers and haemorrhages in the abomasal mucosa. Pulmonary edema and emphysema are also common in this species. Histologically, there is focal or diffuse interstitial nephritis, centrilobular hepatic necrosis and in some cases, vascular lesions in the meninges and brain in subacute to chronic infection [2].

In the later stages, the characteristic finding is a progressive interstitial nephritis manifested by small, white, cortical foci which are initially raised but become slightly depressed as the lesion ages. Many clinically normal cattle presented to abattoirs have these lesions, which may represent sequel to episodes of bacteremia from a variety of pathogens and should not be considered pathognomonic for leptospirosis [30].

Clinical Pathology

General Consideration: Laboratory procedures used in the diagnosis include culture or detection of leptospires in blood or body fluids and detection and measurement of antibody in blood and body fluids and cervico-vaginal mucus [31]. Culture of leptospires is laborious and can take up to 2 months and *L. hardjo* is particularly fastidious in its cultural requirements. Serological and microbiological detection of chronically infected animal is difficult, as is the confirmation of leptospirosis as a direct cause of reproductive losses on a herd. A positive diagnosis of leptospirosis in individual animals is often difficult because of the variation in the nature of the disease, the rapidity with which the organism dies in specimens once they are collected and their transient appearance in various tissues. During the septicaemia stage, leptospirae are present only in the blood and there may be laboratory evidence of acute hemolytic anemia and increased erythrocyte fragility and often haemoglobinuria [24].

If abortion occurs, the kidney, lung and pleural fluid of the aborted fetuses should be examined for the presence of the organism, but even in a fresh fetus the positive identification of leptospirae in lesions is not an easy task. Serological testing at the time of abortion is often unreliable because the acute titers have already peaked and are declining. In the stages immediately after the subsidence of fever, antibodies begin to develop and the leptospirae disappear from the blood and appear in the urine. The leptospiruria is accompanied by albuminuria of varying degrees and persists for varying lengths of time [2].

The diagnosis of leptospirosis is much easier on a herd basis than in a single animal because in an infected herd, some animals are certain to have high titers and chances of demonstrating or isolating the organism in urine or milk are increased with samples being taken from many animals. On the other hand, in a single animal,

depending on when the infection occurred, the titer may have declined to a low level and be difficult to interpret [32].

Serological and Related Tests: Acute and convalescent sera taken 7-10 days apart should be submitted from each clinically affected animal, or from those with a history of abortion and sera should also be taken from 15-25% of apparently normal animals. MAT is the most commonly used serological test for the diagnosis of leptospirosis. In animals which survive infection, acute leptospirosis can readily be diagnosed on the bases of demonstrating a rising antibody titer in acute and convalescent sera [31].

Although paired sera are normally considered necessary so that a rise in titer can be detected, in cases of bovine abortion or still birth, infection may have occurred 1-4 weeks before the abortion by which time the MAT titers may be declining. Following infection, the IgM class of antibodies are first to appear followed by IgG antibodies, which persist for longer than IgM antibodies. The MAT detects both IgM and IgG antibodies. The MAT is particularly useful in diagnosis of disease associated with incidental, non-host-adapted serovars or acute disease associated with host-adapted serovars. It is less useful in diagnosis of chronic disease in maintenance hosts since antibody response to infection may be negligible in chronic infections or may persist from subclinical infections. The ELISA test is much more accurate than others and has many advantages from the point of view of laboratory practice. It can be specific for IgM antibodies or IgG antibodies. A positive IgM-specific ELISA results can therefore indicate that infection occurred within the previous month [33].

Demonstration or culture of organism or antibody: A number of tests are available to detect leptospires or leptospirae DNA in tissues or body fluids. Of all the laboratory diagnostic tests for leptospirosis, the examination of urine samples for the organism probably offers the best opportunity to demonstrate the presence of infection. Following natural infection with *L. hardjo*, cattle may shed leptospires in the urine for between 28 and 40 weeks; following experimental infection, shedding occurs for about 26-32 weeks. After the initial infection, large numbers of leptospires are shed in the urine for several weeks and thereafter there is a progressive decline in the numbers shed, which may be associated with sharp increase in urinary anti-leptospiral IgG and IgA antibody levels [34].

Detection of organism in urine: Fluorescent staining of antibody in urine a fast and accurate diagnostic method for detecting the presence of *Leptospira* and for identifying serotypes. An ELISA has been used to detect specific antibody to *L. hardjo* in the cervico-vaginal mucus as early as 2 weeks after natural or experimental infection and may reach high levels after 8 weeks [29].

Detection of organism in tissue: DNA probe and PCR-leptospire can be detected in tissues using a DNA genomic probe and DNA-based techniques will probably provide rapid and sensitive diagnostic techniques that are serovar and genotype specific. The DNA probe was shown to be much more sensitive than other techniques in detecting the genotype *hardjo-bovis* [35].

Detection of Organism in Semen: A PCR assay has been developed to detect pathogenic leptospire in the semen and urine of infected bulls [3].

Differential Diagnosis: Acute leptospirosis must be differentiated from those diseases causing hemolytic anemia with or without haemoglobinuria. They include: Babesiosis; anaplasmosis; post parturient haemoglobinuria; bacillary haemoglobinuria, etc. Chronic leptospirosis causing abortion must be differentiated from all other causes of abortion in cattle [32].

Treatment: The primary aim of treatment is to control the infection before irreparable damage to the liver and kidneys occur. Treatment with dihydrostreptomycin preferably, or one of the oxytetracyclines, as soon as possible after signs appear is recommended. The results of treatment are often disappointing because in most instances animals are presented for treatment only when the septicaemia has subsided. The secondary aim of treatment is to control the leptospiruria of 'carrier' animals and render them safe to remain in the group [24].

Parenteral antimicrobials for infections due to *L. Pomona* include dihydrostreptomycin (12 mg/kg BW IM twice daily for 3 days) is effective in the treatment of the systemic infection. For the elimination of leptospiruria in cattle, a single dose of dihydrostreptomycin (25 mg/kg BW IM) is recommended [36]. In an outbreak in cattle the simultaneous treatments of all animals with dihydrostreptomycin at 25 mg/kg BW IM and vaccination has been successful in preventing new cases and abortion when pregnant cattle are involved. Oxytetracycline, tilmicosin and ceftiofur are also effective for resolving leptospirosis in cattle [37].

Control

Biosecurity and Biocontainment: In an individual farm leptospirosis can be eradicated or controlled by vaccination. Annual revaccination and regular serological testing for new infections, combined with controlling the source of new infections, will usually successfully control further outbreaks. The major risk is the introduction of carrier animals of any species, or by reintroduction of the infection by rodents or other wildlife. It is because of this risk that most programs aim at containment rather than eradication. The first step in control is to identify the original source of infection and to interrupt transmission [38].

Source of infection include clinically affected animals, wildlife, aborted fetuses, placentas, carrier animals, dogs and cats and environmental sources such as water supplies. Education about leptospirosis is an effective method for reducing its incidence and its effects [39]. Groups to which educational efforts should be directed include professionals in human and veterinary medicine and public health, primary human and animal health care practitioners, wildlife and conservation scientists, water and sewage engineers and planners, health administration and educators and not least, the public at risk. Bull's destined for artificial insemination centers must be free of antibody to *L. hardjo*, *L. grippityphosa*, *L. Canicola*, *L. Pomona* and *L. icterohaemorrhagiae* [40].

Eradication: Detection and elimination of carrier animals presents some difficulties. Positive reactors to the MAT do not necessarily void infective urine and determine their status as carriers necessitates repeated examination of the urine for the organism. New stock brought on to clean premises must be held in isolation for 2 weeks and should be given a single parenteral treatment with dihydrostreptomycin in order to eliminate a possible renal carrier state [24].

If eradication is attempted and completed, introduced animals should be required to pass a serological test on two occasions at least 2 weeks apart before allowing them to enter the herd. Occupational hygiene and control of clinical disease by immunization are also important for prevention and control of leptospirosis [24].

CONCLUSIONS

Leptospirosis is an infectious disease of animals and humans, its incidence increases in the rainy season. Potential sources of infection are carrier animals which

shed the leptospire in the urine. It is easily transmitted from animal to animal and animal to human by contact with potentially contaminated urine and also by contaminated aborted fetuses and discharges. The disease causes major economic losses especially in dairy and beef farms worldwide. Peoples affected are most professionals and recreational related which are in close contact with animals and contaminated water sources. For both human and animal leptospirosis, extension services should include on addressing the impacts of risk factors for the occurrence of leptospirosis. Furthermore, interdisciplinary collaboration and joint ventures among medical, veterinary and public health professionals is of paramount importance to control this disease.

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