

## Leptospirosis in Animal and its Public Health Implications: A Review

Waktole Yadeta, Bashahun G. Michael and Nejash Abdela

School of Veterinary Medicine, College of Agriculture and Veterinary Medicine,  
Jimma University, P.O. Box 307, Jimma, Ethiopia

**Abstract:** Leptospirosis is a globally important zoonotic disease caused by the pathogenic Gram negative bacteria of the genus *Leptospira*. The disease occurs in nearly all mammalian species. It is more common in the tropical regions because of the longer survival of the organism in the environment and frequent exposure of animals and humans to contaminated environments. This manuscript reviews the likely impact of leptospirosis on animal health and its public health implications. The disease is highly zoonotic and transmitted from animal to human through contact with urine from infected animal or through ingestion of contaminated feed and water by *Leptospira*. Human infection of Leptospirosis varies from asymptomatic to severe and usually has biphasic illness. Rodents are the major reservoir of infection. Occupations with high risk of infection in human, host preference to cause acute or chronic condition of the disease, resistance to bactericidal action of complement and neutrophils and long survival of the organism in the environment are some of risk factors of the disease. The clinical signs of leptospirosis do not vary greatly with the species of animals. Laboratory tests used for the detection of the disease are microscopic evaluation, culture, molecular method, serology and animal inoculation. Dihydrostreptomycin, tetracycline, penicillin, ampicillin, doxycycline, streptomycin and erythromycin are common antibiotics used to treat the disease. Sanitary measures, vaccination, quarantine and rodent control are the most important control measures of the disease. Except few researches, Occurrence of leptospirosis in Ethiopia is not well documented so, more research should be conducted on prevalence of leptospirosis in Ethiopia.

**Key words:** Leptospirosis • Zoonosis • *Leptospira* • Rodents

### INTRODUCTION

Leptospirosis is a globally important zoonotic disease caused by the pathogenic Gram negative bacteria of the genus *Leptospira* [1]. In human it was estimated that more than 500,000 cases of severe leptospirosis are reported each year, with case fatality rate exceeding 10%. In farm animals it is major cause of reproductive loss [2]. *L. interrogans* is a pathogenic species that cause leptospirosis while *L. biflexa* is nonpathogenic [3]. *Leptospira* have characteristic hooked ends and are tightly coiled with approximately 18 coils per cell [4]. Leptospire are aerobic, catalase and oxidase positive and the most commonly used media for culturing are Fletcher's semisolid medium, liquid Korthof's medium and Ellinghausen-McCullough-Johnson-Harris medium abbreviated as EMJH [5]. Leptospirosis occurs particularly in tropical and subtropical region,

where environmental conditions favor the survival of the organism and transmission of the disease [6]. It affects virtually all mammals and is transmitted directly or indirectly depending upon the immediate source of infection [7].

Occupations with high risk of infection in human, host preference to cause acute or chronic condition of the disease, resistance to bactericidal action of complement and neutrophils and long survival of the organism in the environment are some of risk factors of the disease [8]. The disease is characterized by a broad range of clinical signs which can be presented as acute, sub-acute or chronic. Clinical signs of acute or sub-acute disease are observed in the leptospiremic phase and characterized by septicemia and hemorrhagic syndrome while clinical signs related to chronic infections in livestock are usually associated with reproductive losses [9]. Diagnosis of leptospirosis depends on the samples available and

temporal stage of the illness. Laboratory tests used for the detection leptospirae are microscopic evaluation, culture, molecular method, serology and animal inoculation [10].

Leptospirosis can be treated by antibiotics such as tetracycline, penicillin, ampicillin, doxycycline, streptomycin and erythromycin [11], while Prevention is characterized by sanitary control and decrease in the risk of infection occurring due to contact with contaminated environments, infected wild animals as well as with synanthropic animals and rodents [2]. Control measures of leptospirosis are aimed at limiting the occurrence of clinical disease based on integrated actions in several links of the transmission chain [12]. Although leptospirosis is common disease in tropical region with significant zoonosis and mortality in both animals and humans it is not well recognized and there is paucity of well documented information, Therefore, the objectives of this paper were to review impact of leptospirosis on animal health and its public health implications.

### Literature Review

**Etiology:** Leptospirosis is caused by pathogenic spirochaetes of genus *Leptospira*, occurring in nearly all the mammalian species [13]. Genus *Leptospira* is classified under Order Spirochaetales, Family Leptospiraceae, Class Spirochaetes and it is divided into two species: *L. interrogans*, comprising all pathogenic strains and *L. biflexa*, comprising the saprophytic strains isolated from the environment [3]. *L. interrogans* contain over 212 serovars arranged into 23 serogroups. Common serovars of *L. interrogans* are serovar Pomona, Canicola, Bratislava, Grippotyphosa, Hardjo and Icterohaemorrhagiae [8].

In humans Leptospirosis is known by various names like Weil's disease, Pretibial fever, Fort Bragg fever, Pea picker's fever, rice field fever, sugar cane cutter fever, swine herder's disease [14]. The morphology of leptospirae is unique among spirochetes in that they have characteristic hooked ends and are tightly coiled [4]. It is 0.1  $\mu\text{m}$  wide and 6–20  $\mu\text{m}$  long. In tissue and within phagocytes, organisms can assume a spherical or granular appearance. Their narrow helical form allows *Leptospira* to burrow into tissue. *Leptospira* have two periplasmic flagella, one attached sub terminally at each end that extend toward the cell's center without overlapping. Although the flagella lie inside the spirochete's outer membrane, they are integral to cell shape and motility [15].

*Leptospira* are an obligate aerobe with an optimum growth temperature of 28-30°C and PH 7.2-7.6 [16, 17]. The media used for isolation and cultivation of leptospirae

can be liquid or solid enriched with rabbit serum or bovine serum albumin. These most commonly used media are Fletcher's semisolid medium, Korthof's liquid medium and Ellinghausen-McCullough-Johnson-Harris medium, However no growth occurs on blood agar and other routine [2]. Antibiotics are usually added in these media when the specimen such as urine, is likely to be contaminated with other bacteria. These media contain supplements of long chain fatty acids, ammonium salts and vitamins B1 and B12, of which all are essential for successful growth [3]. The growth is often slow, with the periods of 3-4 weeks required after inoculation and round colonies of 1-3 mm in diameter develop. *Leptospira* is oxidase and catalase positive and many have lipase activity and some produce urease [7].

### Epidemiology

**Geographical Distribution:** Leptospirosis has worldwide distribution due to the large spectrum of mammalian hosts that harbor and excrete the agent from their renal tubules [18]. According to Guerrant *et al.* [19] although leptospirosis is one of the wide spread zoonoses in the world, it is more common in the tropical regions, because of the longer survival of leptospirae in the environment and frequent exposure of animals and human to contaminated environments [19]. China, Southeast Asia, Africa, South and Central America have immense areas where the disease is endemic. Serovars of *L. interohaemorrhagiae*, *L. canicola*, *L. pomona*, *L. hardjo* and *L. gripotyphosa* occur in all continents except Antarctica and outbreaks in animals and humans have been reported following natural disaster such as flooding and hurricane [20].

### Hosts

**Maintenance Hosts:** An animal infected with a host-adapted serovar of the organism is a maintenance or reservoir host. Each serovar is adapted to a particular maintenance host, although they may cause disease in any mammalian species. A serovar behaves differently within its maintenance host species and incidental or accidental hosts [8]. The disease is maintained in nature by chronic infection of the renal tubules of these maintenance hosts [21].

Maintenance host is characterized by a high susceptibility to infection, endemic transmission within the host species, relatively low pathogenicity for its host, tendency to cause chronic rather than acute disease, producing insidious economic loss through reproductive losses, persistence of the serovar in the kidney and sometimes the genital tract, low antibody response to

infection and low efficacy of vaccination in prevention of infection. Examples of this relationship are serovar Bratislava in swine and serovar hardjo bovis in cattle [8]. The primary reservoir hosts for most *Leptospira* serovars are wild mammals, particularly rodents and Reservoir hosts among domestic animals includes cattle, dogs, sheep and pigs and they may act as carriers for several months (temporary carrier) while rodents usually remain carrier throughout their life (permanent carrier). Rodents are therefore considered as the major reservoir of infection [2].

**Accidental (Incidental) Hosts:** Exposure of susceptible animals to non-host-adapted serovars results in accidental or incidental disease. Incidental host is characterized by relatively low susceptibility to infection but high pathogenicity for the host, a tendency to cause acute and severe rather than chronic disease. An example of this relationship is infection by serovar Pomona in cattle which is pig adapted serovar [8]. Humans are incidental hosts for *Leptospira* species [18].

Table 1: Maintenance and incidental hosts for important serovars of *L. interrogans*

Serovar	Maintenance host	Incidental host
<i>L. Bratislava</i>	Pig	Horse, dog
<i>L. Canicola</i>	dog	Pig, cattle
<i>L. graphityphosa</i>	rodent	Cattle, pig, horse, dog
<i>L. hardiso</i>	cattle	human
<i>L. interohemorrhagie</i>	Brown cat	Domestic animals and human
<i>L. Pomona</i>	Pig, cattle	Sheep, horse, dogs

Source: [5].

**Source of Infection and Modes of Transmission:**

The main sources of infection of the disease are urine of infected or carrier animals, contaminated surface water, mud, feed, soil, aborted fetuses and uterine discharges [21]. From these sources the organism enters the body via mucous membranes of the eyes, nose, vagina, or abraded skin [22]. The modes of transmission of leptospirosis are often categorized as direct or indirect depending upon the immediate source of infection. When the immediate source of infection is animal tissue, body fluids, urine, Transplacental, or venereal the transmission is termed as direct. When the immediate source of infection is an environment contaminated with urine of carrier animals, the transmission is termed as indirect [7]. Faine *et al.* [23] has reported sexual transmission of *Leptospira* by mating in rats, pigs and dogs. Infected milk and semen of an infected bull may contain leptospirae, so transmission through milk and natural breeding or artificial insemination

can occur but it is uncommon [5]. Transmission to human is through direct or indirect contact of mucous membranes or abraded skin with urine from infected animals or contaminated freshwater surfaces including mud or water in lakes, rivers and streams. Ingestion or inhalation of contaminated water or aerosols may also result in infection [24]. Transmission between humans is very rare and it occurs through blood transfusion, organ transplantation, breast feeding and sexual intercourse [21].

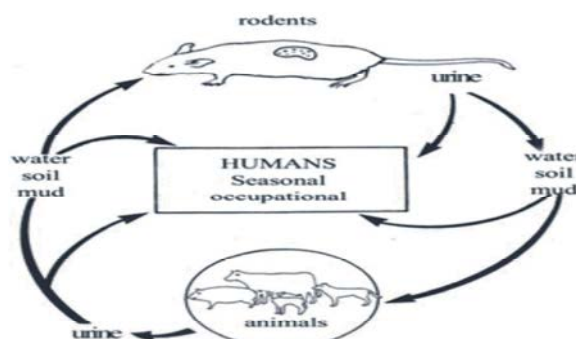


Fig. 2: Transmission cycle of leptospirosis  
Source: PIRP, [25]

**Risk Factors**

**Host and Management Risk Factors:** Animals of all age groups can be affected by leptospirosis, but young animals are affected more often and with higher morbidity [8]. Although leptospirosis virtually occur in all mammalian species, it occur commonly in cattle, sheep, goats, dogs, horses and pigs, but illness seems to be rare in cats [21]. Certain management factors that pose risks of infection are infected animal introduced into herds, co-grazing or common grazing with infected ones, access to contaminated water supplies such as streams, rivers, flood or drainage water and purchasing or loan of infected male animals for natural insemination [8].

**Pathogen Risk Factors:** Virulent *Leptospirae* resist the bactericidal action of complement and neutrophils in non-immune hosts but are rapidly killed by either mechanism in the presence of specific epithelial and endothelial antibody [5]. The ability of *Leptospira* to invade Vero cells and to reduce apoptosis in macrophages was correlated with virulence; nevertheless the organism must penetrate host epithelial and endothelial cell barriers for both hematogenous spread and localization in target organs, such as liver and kidney [26, Revise Ref. S. No. as the previous one is 24??]. A cytotoxic glycol lipoprotein fraction is shown to inhibit hosts ATPase with

the activity ascribed to the presence of long chain fatty acid. *L. Pomona* in cattle causes intravascular haemolysis due to hemolytic exotoxin [27].

**Pathogenesis:** The bacterium enters the body via intact mucous membranes (mouth, nose, eyes, vagina) or a skin with lesions and scratches [17, 28]. Through lymphatic vessels from the infection site the leptospire enter the bloodstream [23]. In the bloodstream the bacteria will multiply and spread to organs such as the kidneys, spleen, central nervous system, liver, eyes and reproductive organs [28]. There are three possible pathways after the systemic circulation. If the animal has a high and adequate antibody titer the body will be cleared from leptospire and no clinical signs can be seen. Animal with a moderate antibody can present with a mild or short leptospiremia followed by mild clinical signs. The leptospire are then eliminated through the kidneys and after the elimination the animal will not continue to shed leptospire. If the animal has a low or absent antibody titer there will be a multiplication of leptospire in the bloodstream [28].

The endothelium will be damaged which can cause ischemia in different organs such as the kidneys (renal tubular necrosis), liver (hepatocellular damage) or lungs [17]. Neutrophils and thrombocytes are stimulated by LPS in the outer membrane of the leptospire and this contributes to inflammation and coagulatory abnormalities [28]. The LPS can contribute to the renal and hepatic damage. Meningitis can develop if the leptospire enter the nervous system or cerebral spinal fluid in the acute phase of the disease. If bacteria persist despite the antibody response, then immune-complex-mediated meningitis can occur. When this phenomenon occurs in the eyes it causes uveitis [23].

The incubation period of leptospirosis depends on dose, infectious strain and host but is averagely between 7-14 days [29]. According to Levett [21] antibodies become detectable 5-7 days after infection. It takes about two weeks for the leptospire to reach the proximal tubular cells and the tubular lumen in the kidneys [30]. In the best case scenario, the antibodies will clear the blood and tissues from leptospire. The bacteria can also become eliminated from the kidneys and no leptospire will thus be shed in the urine. In some animals, despite an increased antibody titer, the bacteria can replicate and persist in the renal tubular cells. This may result in chronic shedding of leptospire in the urine for days to months, even years [23, 28, 30].

#### **Clinical Signs of Leptospirosis in Animals:**

Leptospirosis is characterized by a broad range of clinical symptoms in livestock with minor difference between species affected: acute, subacute or chronic. Clinical signs of acute or sub-acute disease are observed in the leptospiremic phase and it is characterized by septicaemia, high fever and anorexia, petechiation of mucosa, depression and acute hemolytic anaemia with hemoglobinuria, jaundice and pallor of the mucosa [9].

Clinical signs related to chronic infections in livestock are usually associated with reproductive losses through abortion, stillbirth, infertility and mastitis and Milk drop syndrome. Abortion usually occurs during the last trimester of pregnancy. Infertility and milk drop occurs only in pregnant or lactating cows because *Leptospira* organisms prefer pregnant uterus and lactating mammary gland to proliferate [8]. Sudden drop in milk production may affect up to 50% of cows at one time and 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 1-14 days [5].

Infections in goats and sheep can be severe or subclinical and may manifest as reproductive problems such as infertility, abortion and stillbirth [31]. In various studies anorexia, lethargy and vomiting were the three most common clinical signs in dogs with leptospirosis. Weight loss, polyuria, diarrhea, abdominal or lumbar pain, musculoskeletal pain and dehydration were also common [32]. The clinical features of equine leptospirosis are essentially similar to those observed in other animals, such as cattle, with low-grade fever, listlessness and anorexia the most common presentation in milder disease. In more severe forms of disease a range of typical signs may occur, including conjunctival suffusion, jaundice, anaemia, petechial hemorrhages on the mucosa and general depression. Renal failure may also occur, especially in foals. Infection of pregnant mares can result in placentitis, abortion and stillbirths [33].

**Necropsy Findings:** Leptospirosis is characterized by the development of vasculitis, endothelial damage and inflammatory infiltrates composed of monocytes, plasma cells, histiocytes and neutrophils. On gross examination, petechial hemorrhages are common and organs are often discolored due to the degree of icterus [21]. The histopathology is most marked in the liver, kidneys, heart and lungs but other organs may also be affected according to the severity of the individual infection. The overall structure of the liver is not

significantly disrupted, but intrahepatic cholestasis, Hypertrophy and hyperplasia of Kupffer cells is evident while in the kidneys, interstitial nephritis is the major finding accompanied by an intense cellular infiltration composed of neutrophils and monocytes. Leptospire can be seen within the renal tubules [8].

By electron microscopy, the tubular cell brush borders are denuded, the tubular basement membrane is thickened and tubular cells exhibit mitochondrial depletion (In addition, minor changes are seen in the glomeruli), suggesting an anatomical basis for proteinuria in leptospirosis. Pathological findings in the heart include interstitial myocarditis with infiltration of predominantly lymphocytes and plasma cells, petechial hemorrhages (particularly in the epicardium), mononuclear infiltration in the epicardium, pericardial effusions and coronary arteritis. In the lungs, pulmonary congestion and hemorrhage are common while perivascular cuffing and vascular lesions in the meninges are observed in the brain [21].

**Diagnosis:** Laboratory tests are necessary to confirm the diagnosis of clinically suspected leptospirosis due to its varied symptoms, Laboratory analysis depends on the samples available and temporal stage of the illness. Various laboratory tests described for the detection of *Leptospira* are microscopic evaluation, culture, molecular method, serology and animal inoculation [10].

**Microscopic Evaluation:** Leptospire may be seen on microscopic evaluation of blood, urine, CSF and peritoneal or pleural exudate during the first 10 days of the infection [21]. Dark field microscopy is required as the leptospire are very small [34], however more than 10000 organisms/ml are required to be able to see them. This method is insensitive and has a low specificity. When choosing a body fluid to analyze it is important to consider the pathogenesis and at what stage it is possible to detect the leptospire. For example, blood can only be used in the acute stage of the disease [21]. Dark field microscopy must be followed by serology or cultural diagnostic methods if it is desirable to specify the serovar. The leptospire can be seen with light microscopy if using either Geimsa stain or silver impregnation on air-dried smears [30]. Immunofluorescence is also possible to confirm the causative agent [21].

**Culture:** *Leptospira* organisms could be isolated from body fluids, mainly urine. Nevertheless, tissue from dead animals is giving a greater opportunity of a successful isolation, if target tissue is not autolysed. Such target

tissue is kidney, liver, lungs and brain. If the agent is suspect for abortions, isolation could be attempted from non autolysed abortion materials or tissue samples from a freshly aborted fetus. Isolation of the microorganism from fetal tissue (kidney, liver, lungs) confirms maternal infection [17]. Isolation requires expensive and properly prepared and kept culture media. Inoculated media are incubated at 28-30° C for several weeks or months. Cultures are incubated in dark and quite environment. Time of incubation depends on the serovar. Serovars such as Pomona and grippotyphosa require the least time incubation up to 10 day. Regardless of time required for isolation, the inoculated culture media must be protected from contamination, thus require the addition of antimicrobial agents selected to inhibit growth of contaminants [18].

**Molecular Method:** Polymerase Chain Reaction (PCR) assays has been developed but is still not commonly used even though this method has high sensitivity [17]. Serum, urine, aqueous humor and tissues from autopsy have been used for PCR. PCR involves the enzymatic amplification of target DNA sequences specific to the organism Through a series of polymerizations which is carried out by heat stable DNA polymerase enzymes using primers which are short DNA fragments and they bind specifically to the sequence of interest. The amplified DNA produced by this reaction is visualized on agarose gel electrophoresis [21]. Modern methods such as fragment length polymorphism (FLP), pulse field gel electrophoresis (PFGE) and other methods are currently being assessed [10].

#### Serology

**Microscopic Agglutination Test:** MAT is the standard method for the serological diagnosis of leptospirosis [21]. To execute MAT leptospire are grown in liquid media and used alive. Serum is mixed with this live liquid grown leptospire in order to test for agglutination. Agglutination indicates that the serum contains anti-*Leptospira* antibodies [29]. As the leptospire are thin and small, dark field microscopy is used to evaluate the agglutination. Agglutinating antibodies are most frequently IgM and IgG to a lesser extent. IgM concentrations fluctuate according to the presence of the organism [30]. MAT has a high sensitivity and specificity but the difficulties are that live cultures of different serovars are necessary to carry out the method. It is also necessary that a trained person works with the samples and evaluates the result [17].

**ELISA:** Enzyme linked immuno sorbent assay (ELISA) test of leptospirosis can be performed either by using commercial kits or within house produced antigen. A broadly reactive so-called genus-specific antigen is generally used to detect IgM and sometimes also IgG antibodies against *Leptospira* antigen. The presence of IgM antibodies indicates current or recent leptospirosis [20]. Common commercially-available *Leptospira* IgM ELISA is used to serologically detect acute leptospiral infections in patient serum samples. This ELISA works on the principle that any *Leptospira* IgM antibodies present in patient serum will bind to the *Leptospira* antigen attached to the polystyrene surface of the micro wells. Residual serum is removed from these micro wells by washing with 1% buffer (provided in the kit). The peroxidase-conjugated anti-human IgM is there after added to the wells and the plate is re-incubated allowing for the bound antigen antibody complexes to bind to the conjugate. Wells are washed again and a colorless substrate system, tetramethylbenzidine hydrogen peroxide is added. The substrate is hydrolyzed and the chromogen turns blue. The TMB turns yellow once the reaction is stopped using phosphoric acid. Color development indicates the presence of IgM antibodies to *Leptospira* in the serum sample [35].

**Animal Inoculation:** Laboratory animals are useful for isolating the organisms from contaminated material and for maintaining recent isolates and may be used to recover a single serotype from a mixed culture. Young animals preferably weanlings should be used which must be free from endemic leptospiral infection; guinea pigs, hamsters, gerbils, young rabbits, Swiss white mice, albino American deer mice and 1-3-day old chicks may be used. The material should be inoculated intraperitoneally through one of the lower quadrants of the abdominal wall. The animals should be examined twice daily and a drop of peritoneal fluid can be examined with dark field microscopy for active leptospire from the third to the seventh day [3]. On the death of the animal hemorrhagic lesions with spirochetes are found in many organs [5].

#### **Public Health Implication of Leptospirosis**

**Risk Factors in Human:** Leptospirosis affects risk groups that are exposed to animal reservoirs or contaminated environments, such as abattoir and sewage workers, salver workers, coal mines, plumbers, farm workers, veterinarians, pet shop owners, abattoir workers, meat handlers, military personnel, slaughter house workers and workers in fishing industry [36]. Recreational

activities that increase the risk of leptospirosis are gardening and water sports such as canoeing, swimming and white water rafting residents of some urban areas [8]. According to Pavli and Maltezou [37]. Men are more frequently diagnosed with leptospirosis compared with women and this has been traditionally attributed to the over representation of men in high-risk occupations.

**Incidence and Mortality:** The precise incidence of leptospirosis remains unknown due to the lack of awareness and systematic investigation for this disease worldwide. Estimated annual incidence rates range from 0.02 of 100,000 to 1 of 100,000 persons in temperate areas and from 10 of 100,000 to 100 of 100,000 persons in humid tropics [20]. During outbreaks and in high-risk exposure groups, incidence may reach 100 of 100,000 persons [37]. According to Farrelly *et al.* [38] the mortality varies with the form and is higher in the elderly, icteric form is rarely fatal, occurs in 5-10% of all patients and has an overall mortality rate of 5-15% and a 54% case fatality rate in severe cases with myocardial involvement. Most patients with kidney failure, hepatic disease or anterior uveitis eventually recover with full kidney or liver functions and vision.

#### **Incidence and Prevalence of Leptospirosis in Ethiopia:**

So far, few documented information concerning the occurrence of leptospirosis in animals in Ethiopia, climatologic, socioeconomic and other factors are highly favorable for the occurrence and spread of the disease in the country. In Ethiopia leptospirosis has been reported to occur in domestic animals [39] working in Ethiopia, found incidences of 91.2% in horses, 70.7% in cows, 57.1% in pigs, 47.3% in goats, 43.4% in sheep, 15.4% in camels and 8.3% in dogs. In human Eshetu *et al.* [40] reported from a total of 59 febrile patients attending the outpatient of oromia region, Wonji Hospital, 47.46% of the patients were positive for leptospirosis and the occurrence of the disease was more common in males than females. According to Tsegay *et al.* [41] a total of 184 out of 418 horses had antibody titres of 1:100 or greater to at least one of 16 serovars, demonstrating the presence of 16 serovars of *Leptospira* species in Central and Southern Ethiopian horses. This means, 44% of the sampled horses were seropositive to at least one serovar.

**Treatment:** The primary aim of treatment is to control the infection before irreparable damage to the liver and kidneys occurs [21]. Treatment with dihydrostreptomycin 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 septicemia has subsided. The secondary aim of treatment is to control the leptospiruria of carrier' animals and render them safe to remain in the group [8]. Other Antibiotics used to treat leptospirosis include tetracycline, penicillin, ampicillin, doxycycline, streptomycin and the erythromycin. The efficacy of treatment may depend on the serovar. Fluid therapy, blood transfusion and other supportive care may also be necessary [30]. These supportive treatments depend on the animal and its needs and if the animal is severely affected and in shock it will need fluid therapy [21].

Fluid therapy is necessary for small animals with fluid losses due to diarrhea and vomiting. Fluids are chosen according to electrolyte imbalances [30]. Blood transfusions are indicated as treatment for the hemolytic anemia in acute leptospirosis in cattle. The clinical indications for a blood transfusion include obvious pallor of the mucous membranes, weakness and tachycardia [8]. In beef herds further abortions may be prevented by vaccination and treatment of all animals with antibiotics and in dairy cattle, only infected animals are usually treated due to the potential loss of milk sales [42]. The primary treatment for equine recurrent uveitis in horses is anti-inflammatory drugs such as corticosteroids and medications to decrease discomfort like topical atropine, Surgery and other therapies may also be used [8].

**Prevention and Control:** Understanding the epidemiological features of leptospirosis is a critical step in designing interventions for reducing the risk of the disease transmission [21]. Intervention strategies can target many points in the transmission cycle of leptospirosis. Although little can be done in wild animals, leptospirosis in domestic animals can be controlled through vaccination, Prophylactic treatment of exposed animals with antibiotics, quarantine newly introduced animals of whatever the species for at least 4 weeks, Rodent control, regular serological testing, improved environmental hygiene, separating young animals from adults and safe artificial insemination [13].

Occupational hygiene, taking care of animal bite, vaccination, drinking clean water, early treatment, prophylactic therapy, acquisition of information for people coming to high risk areas, is fundamental for preventing human leptospirosis [20]. In herds, the disease is usually introduced by an infected animal, through the

environment or by contact with other infected animals in mixed pasture. Animal reposition must be selected according to the non-reactivity of herds to leptospirosis. Available Vaccines of leptospirosis for domestic animals can decrease the severity of the disease although it cannot prevent the infection completely because the immunity is serovar-specific and vaccines protect only against serovars included in the immunogens [12].

## CONCLUSIONS

Leptospirosis is a bacterial zoonosis caused by spirochetes of genus *Leptospira* of the order Spirochaetales. The disease infects both animals and human worldwide. In human it is commonly called Weil's disease. It occurs most commonly in tropical regions. In Ethiopia it has been reported in both human and domestic animals. The main sources of infection are urine of infected or carrier animals and environment contaminated with urine of these animals. In human the disease is associated with occupations exposing to animal reservoirs and contaminated environment. Transmission modes of this disease are through direct or indirect contact of mucous membranes or abraded skin with urine from infected animals or contaminated freshwater surfaces including mud or water in lakes, rivers and streams. Ingestion or inhalation of contaminated water or aerosols may also result in infection. Leptospirosis is easily treatable in early case of infection by antibiotics. Laboratory tests described for confirmation of the disease are microscopic evaluation, culture, molecular method, serology and animal inoculation. Sanitary measures, vaccination, quarantine and rodent control are the most important control measures in domestic animals. In Human it can be controlled by reducing its prevalence in wild and domestic animals.

Based on the above conclusion the following points are forwarded as recommendations:

- More researches should be conducted on prevalence/incidence of leptospirosis in Ethiopia since very few findings are documented currently.
- Communities at risk should wear protective gear when exposed to animal reservoirs and contaminated environment.
- Government should involve in funding the research to be done on the status of leptospirosis in Ethiopia.
- Proper control measures and public awareness should be made in endemic areas

## REFERENCES

1. Bharti, A.R., J.E. Nally, J.N. Ricaldi, M.A. Matthias, M.M. Diaz and M.A. Lovett, 2003. Leptospirosis, a zoonotic disease of global importance. *The Lancet Infectious Diseases*, 3: 757-771.
2. Tilahun, Z., D. Reta and K. Simenew, 2013. Global epidemiological overview of leptospirosis. *International Journal of Microbiology Research*, 4(1): 09-15.
3. Sharma, M. and A. Yadav, 2008. Leptospirosis: epidemiology, diagnosis and control, *Journal of Infectious Disease and Antimicrobial Agents*, 25(2): 93-103.
4. Doern, G.V., 2000. Detection of selected fastidious bacteria, *Journal of Clinical Infectious Diseases*, 30: 166-173.
5. Fentahun, T. and M. Alemayehu, 2012. Leptospirosis and its public health significance, a review, *European Journal of Applied Sciences*, 4(6): 238-44.
6. Pappas, G., G.P. Papadimitriou, V. Siozopoulou, L. Christou and N. Akritidis, 2008. The globalization of leptospirosis, worldwide incidence trends, *International Journal of Infectious Diseases*, 12(4): 351-357.
7. Sehgal, S.C., 2006. Epidemiological patterns of leptospirosis. *Indian journal of medical microbiology*, 24(4): 310-311.
8. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and P.D. Constable, 2006. *Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs and goats*, London, Saunders, Elsevier Health Sciences, pp: 1094-1110.
9. Petrakovsky, J., A. Bianchi, H. Fisun, P. Nájera-Aguilar and M.M. Pereira, 2014. Animal Leptospirosis in Latin America and the Caribbean Countries, Reported Outbreaks and Literature Review (2002-2014), *International journal of Environmental Research and Public Health*, 11(10): 10770-10789.
10. Ahmad, S.N., S. Shah and F.M. Ahmad, 2005. Laboratory diagnosis of leptospirosis. *Journal of Post graduate Medical*, 51: 195-200.
11. Heymann, D., 2004. *Control of Communicable Diseases, Manual*, American Public Health Association, 18<sup>th</sup> ed., pp: 306-309.
12. Lucheis, S.B. and Jr. Ferreira, 2011. Ovine leptospirosis in Brazil, *The Journal of Venomous Animals and Toxins including Tropical Diseases*, 17(4): 394-405.
13. Dhanze, H., M. Kumar, Suman and B.G. Mane, 2013. Epidemiology of leptospirosis, An Indian perspective, *Journal of food borne and zoonotic diseases*, 1(1): 6-13.
14. Mohit, Bhatia, B.L. and Umapathy, 2015. Desiphering leptospirosis-a diagnostic mystery, an insight, *International Journal of Medical Research and Health Sciences*, 4(3): 693-701.
15. Plank, R. and D. Dean, 2000. Overview of the epidemiology, microbiology and pathogenesis of *Leptospira* spp. in humans, *Journal of Microbes and Infection*, 2(10): 1265-1276.
16. Palmer, M.F. and W.J. Zochowski, 2000. Survival of leptospires in commercial blood culture systems revisited, *Journal of Clinical Pathology*, 53: 713-714.
17. Adler, B. and A.P. Moctezuma, 2010. *Leptospira* and Leptospirosis, *Journal of Veterinary Microbiology*, 140: 287-296.
18. Ko, A.I., C. Goarant and M. Picardeau, 2009. *Leptospira*, the dawn of the molecular genetics era for an emerging zoonotic pathogen, *Journal of Nature Reviews Microbiology*, 7: 736-47.
19. Guerrant, R.L., D.H. Walker and P.F. Weller, 2006. *Tropical infectious diseases: principles, pathogens and practice*, 2<sup>nd</sup> ed. Philadelphia, PA. Elsevier, pp: 105-134.
20. WHO (World Health Organization), 2003. *Human leptospirosis: guidance for diagnosis, surveillance and control*. pp: 1-107, Malta.
21. Levett, P.N., 2001. Leptospirosis, *Clinical Microbiology Reviews*, *Journal of American society of microbiology*, 14(2): 296-326.
22. Thayaparan, S., I.D. Robertson, A. Fairuz, L. Suut and M.T. Abdullah, 2013. Leptospirosis, an emerging zoonotic disease in Malaysia, *Malaysian Journal of Pathology*, 35(2): 123-32.
23. Faine, S., B. Adler, C. Bolin and P. Perolat, 1999. *Leptospira and Leptospirosis*, 2<sup>nd</sup> edn, medisci, Melbourne, Australia.
24. Meites, E., M.T. Jay, S. Deresinski, W.J. Shieh, S.R. Zaki, L. Tompkins and D.S. Smith, 2004. Reemerging leptospirosis, California. *Emerg Infect Dis*, 10(3): 406-12.
25. Saif, A.N., 2013. The Detection of *Burkholderia* spp. and pathogenic *Leptospira* spp. in South Africa. PhD Thesis, university of the Witwatersrand, pp: 96.
26. Merien, F.G., Baranton and P. Perolat, 1997. Invasion of verocells and induction of apoptosis in macrophages by pathogenic *Leptospira* spp. are correlated with virulence, *Journal of Infectious Immunology*, 65: 729-738.



27. Craig, E., J.E. Greene, A.B. Sykes, Cathy and K. Hartmann, 2006. Infectious disease of the dog and cat, 3<sup>rd</sup>edn, Canada, Saunders, pp: 402-417.
28. Langston, C.E. and K.J. Heuter, 2003. Leptospirosis, A re-emerging zoonotic disease, Veterinary Clinics of North America, Journal of Small Animal Practice, 33(4): 791-807.
29. Sykes, J.E., K. Hartmann, K.F. Lunn, G.E. Moore, R.A. Stoddard and R.E. Goldstein, 2011. 2010 ACVIM small animal consensus statement on leptospirosis: diagnosis, epidemiology, treatment and prevention, Journal of Veterinary Internal Medicine, 25(1): 1-13.
30. Greene, C.E., J.E. Sykes, G.E. Moore, R.E. Goldstein and R.D. Schultz, 2006. Chapter 42, Leptospirosis, Infectious Diseases of the dog and cat, 4<sup>th</sup> ed, St Louis, Saunders, Elsevier, pp: 431-447.
31. Zacarias, F.G.D.S., S.A. Vasconcellos, E.K. Anzai, N. Giraldo, J.C.D. Freitas and R. Hartskeerl, 2008. Isolation of leptospiraserovars Canicola and Copenhageni from cattle urine in the state of Parana, Brazil. Brazilian, Journal of Microbiology, 39(4): 744-748.
32. Greenlee, J.J., C.A. Bolin, D.P. Alt, N.F. Cheville and C.B. Andreasen, 2004. Clinical and pathologic comparison of acute leptospirosis in dogs caused by two strains of *Leptospira kirschneri* serovar grippotyphosa. American journal of veterinary research, 65(8): 1100-1107.
33. Verma, A., B. Stevenson and B. Adler, 2013. Leptospirosis in horses. Journal of Veterinary microbiology, 167(1): 61-66.
34. Zuerner, R.L., 2010. Genus *Leptospira*, Bergey's Manual of Systematic Bacteriology, 2<sup>nd</sup>ed, 4: 232-242, Springer, New York.
35. Winslow, W.E., D.J. Merry, M.L. Pirc and P.L. Devine, 1997. Evaluation of a commercial enzyme-linked immunosorbent assay for detection of immunoglobulin M antibody in diagnosis of human leptospiral infection. Journal of clinical microbiology, 35(8): 1938-1942.
36. Katz, A.R., V.E. Ansdell, P.V. Effler, C.R. Middleton and D.M. Sasaki, 2002. Leptospirosis in Hawaii, 1974-1998, Epidemiologic analysis of 353 laboratory-confirmed cases, American Journal of Tropical Medicine Hygiene, 66: 61.
37. Pavli, Androula and Helena, C. Maltezou, 2008. Travel-Acquired Leptospirosis, Journal of Travel Medicine, 15(6): 447-453.
38. Farrelly, H.E., B. Adler and S. Faine, 1987. Oponic monoclonal antibodies against lipopolysaccharide antigens of *Leptospira interrogans* serovar hardjo. Journal of medical microbiology, 23(1): 1-7.
39. Moch, R.W., E.E. Ebner, I.S. Barsoum and B.A.M. Botros, 1975. Leptospirosis in Ethiopia, A serological survey in domestic and wild animals, Journal of Tropical medicine and Hygiene, 78(2): 38-42.
40. Eshetu, Y., K. Simone, M. Tsehaynesh, W. Dawit, N. Bethelehem, G. Neway, D. Belachew, J. Eduard and Sanders, 2004. Human leptospirosis in Ethiopia, a pilot study in Wonji, Ethiopian Journal of Health Developmet, 18(1): 48-51.
41. Tsegay, K., A.D. Potts, N. Akililu, C. Lötter and B. Gummow, 2016. Circulating serovars of *Leptospira* in cart horses of central and southern Ethiopia and associated risk factors. Journal of Preventive Veterinary Medicine, 125: 106-115.
42. Griffith, M.E., D.R. Hospenthal and C.K. Myrray, 2006. Antimicrobial therapy of leptospirosis, journal of Current Opinion on Infectious Diseases, 19: 533-537.