

Review

Review on Leptospirosis and its Public Health Significance

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ABSTRACT

Leptospirosis is the most widespread and most prevalent zoonotic disease caused by *Leptospira* bacteria this is pathogenic Gram-negative bacteria, the disease leptospirosis a zoonotic disease of global importance. It caused by various serovars of *leptospira interrogans* that belong to the genus *Leptospira*. Besides *leptospira interrogans* is the most serovars like the disease occurs in nearly all mammalian species. The highest burden in low-income both found in urban and in rural environments. Rodents are known as the main reservoir animals, However, all animals are reservoir include human so leptospirosis is more common in the tropical regions rather than subtropics because of the longer survival of the organism in the environment and frequent exposure of animals and humans to contaminated environments. Direct transmission to humans by broken skin and mucous membranes exposed to the urine of infected animals. While the clinical signs of leptospirosis do not vary greatly with the species of animals include laboratory tests. Control and prevention is sanitary measures, vaccination, quarantine and rodent control are the most important control measures of the disease, require elimination of the carrier state, maintenance of environmental condition and immunization of the animal, tropical areas where warm humid conditions and alkaline or neutral soil helps *leptospira* survive better.

Keywords

Leptospirosis; Zoonosis; *Leptospira*; Rodent.

INTRODUCTION

Leptospirosis has a global impact on human health and is considered to be burdening the world economy. More than 60,000 people die due to leptospirosis annually and nearly one million are reported to be affected.¹ During the past several years, a large number of leptospirosis outbreaks have shook many parts of the world. In addition to Indian sub-continent Oceania, Caribbean and Latin America are considered endemic to leptospirosis.²

Leptospirosis, classified as a neglected tropical disease, is caused by a pathogenic spirochete bacterium of the genus *Leptospira*.³ Besides *leptospira* are classified in to two *leptospira interrogans* are pathogenic species that cause Leptospirosis whereas *L. biflexa* is non-pathogenic.⁴ Although leptospirosis is one of the world most widely spread bacterial disease caused by the genes, which are classified as a direct anthroponosis, it affects humans and a wide range of animals. Leptospirosis traditionally has to be considered as rural based diseases however, urban epidemic associated with severe form of the disease is also reported to occur annually resulting in significant mortality.⁵

Pathogenic *Leptospira* lives in the kidneys of natural hosts predominantly mammals, and are excreted with the urine into the environment where they survive for up to several months depending on favorable conditions. Like, infection occurs by contact with infected animals, their urine or *via* urine contaminated environment (mostly mud and water). The infection route is through open skin and mucous membranes. Leptospirosis is commonly diagnosed in several animal species a large number of sylvatic and domestic animals serve as reservoir of *Leptospira*. Human infections caused by *L. interrogans* of which there are over 200 known Pathogenic serovars and several modes of transmission are involved in leptospirosis.⁶

Human infection is caused by recreational exposure to water contaminated with *Leptospira* and therefore humans are considered to be accidental hosts whereas animals are considered reservoir hosts of *Leptospira*, a large number of clinical manifestations are associated with leptospirosis. The diagnosis of leptospirosis is based on the availability of the sample and the temporal stage of the disease. Laboratory tests used for the detection of *leptospira* are

microscope evaluation, culture, molecular methods, serology and animal inoculation.⁷

Treatments of Leptospirosis by antibiotics such as tetracycline, penicillin, ampicillin, doxycycline, streptomycin and erythromycin.⁸ The risk of infection occurring due to interaction with contaminated environments, infected wild animals as well as with synanthropic animals and rodents,⁹ control measures of leptospirosis are aimed at limiting the occurrence of clinical disease based on integrated actions in clinical disease based on integrated actions in several links of the transmission chain.¹⁰ Therefore, the objectives of this paper were to review on Leptospirosis and its Public Health Significance that affects Domestic Animals and Humans.

LITERATURE REVIEW

Etiology

Leptospirosis is caused by pathogenic spirochetes of genus *Leptospira* occurring in almost all the mammalian species,¹¹ *Leptospirae* is unique among spirochetes like Leptospirosis is caused by spirochetes from the genus *Leptospira* currently contains 20 species containing nine pathogenic, six saprophytic, and five intermediate species.¹² Like Leptospirae are mobile, their bodies are small diameter requiring the use of dark field microscopy or phase contrast for observation.

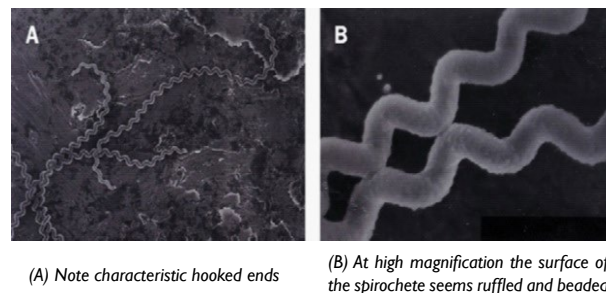
These bacteria are aerobic, do not resist drought or hypertonicity, however, they support alkalization to pH 7.8. Leptospirae are thin, obligate aerobe, fine spiral shaped organisms with hooked ends having two or more axial filaments that are responsible for the motion of the spirochete, and visualized under dark field microscopy, the epidemiology of most serovars is poorly studied but certain serovars have been linked strongly to specific animal reservoirs⁶ some of the serotypes are present globally, while others are confined to certain place.

Leptospirosis is an infectious disease caused by *L. interrogans* complex, which has over 20 serogroups and more than 200 serovars. Dog, and pigs act as carriers for several months (temporary carriers) while rodent usually remain carrier throughout their life (permanent carrier). Rodents are therefore considered as the major reservoir of infection. Leptospirae are excreted in the urine of the animals and they affect man when he is exposed to urine of infected animals, directly or indirectly,¹³ some of the serotypes are present globally, while others are confined to certain areas. Commonly known serovars are *L. interrogans* serovars pomona (swine), *L. interrogans* Bratislava (swine), *L. interrogans* Canicola (dogs), *L. interrogans* hardiso (cattle), and Icterohemorrhagiae and *L. graphityphosa* (rats).¹⁴

The bacteria are highly motility thin, flexible and filamentous, made up of fine spirals with hook-shaped ends.¹⁵ It is 0.1 µm wide and 6-20 µm long. In tissue and inside phagocytes, the organism will assume a spherical or granular look. Their narrow helical type permits *Leptospira* to burrow into a tissue. *Leptospira* have two periplasmic flagella, one attached sub terminally at each end that extends toward the cell center without overlapping, although the

flagella lie inside the spirochaete outer membrane, they are integral to cell form and motility,¹⁶ the organism is sensitive to common disinfectants and antiseptic, and easily killed at 60 °C in 10 seconds. The case fatality range from three to over 50% (Figure 1).¹⁷

Figure 1. High-Resolution Scanning Electron Micrograph of *Leptospira Interrogans* Serovar Icterohemorrhagiae



The *Leptospira* genome has two circular chromosomes and the genome is larger compared with the genomes of the other spirochetes. This indicates that *Leptospira* species can live in a diverse environment like animal hosts and freely in the environment.¹⁸

They grow in simple media enriched with vitamins B2 and B12, the media used for isolation and cultivation of *Leptospira* are liquid or solid enriched with rabbit serum or bovine albumin. Fletcher's semisolid medium, Korthof's liquid medium and Ellinghausen-McCullough-Johnson-Harris medium are the most commonly used media. On the other hand, no growth occurs on blood agar and other routines and some produce urease.¹⁹ Leptospirae are motile and aerobic, cannot survive dry environment, heat, acids and basics disinfectants but can sustain alkali pH up to pH 7.8.²⁰ Although the bacteria can only be visualized using a dark field or phase contrast microscope they are gram negative in nature, gram's staining is not done for visualization of the bacteria. Like silver impregnation, staining, immune peroxidase staining or immunofluorescence should be done to observe these bacteria.²¹

Leptospirosis is aerobic in nature, the bacteria can be cultured media enriched with vitamins like B1 and B12, long-chain fatty acids, and ammonium salts at 28-30 °C. Of course fatty acids are the sole source of carbon and are utilized by β-oxidation.²²

EPIDEMIOLOGY OF LEPTOSPIROSIS

Leptospirosis has a complex epidemiology as many animals serve as reservoir of infections, widely distributed geographically and occurs mostly in tropical, subtropical and temperate zones.²³ Disease is recorded in both sexes with predominance in young adult males, and in urban and rural settings, it is an important occupational zoonosis of agriculture workers, sewer workers, mine workers, slaughterhouse employees, butchers, dairy farmers, veterinarians, animal handlers, kennel attendants, sanitary workers, construction workers, military personnel's and fishermen.⁶ To occupationally exposed teams, urban slum dwellers in areas with poor sanitation are communities at significantly high-risk.²⁴ Infection-related to outdoor

recreational exposure, international travel, especially to endemic areas in the tropics and flooding is increasing.

Most communities in such areas collect water from natural water bodies such as rivers, streams, or underground aquifers, and then store this water for long periods of time are reservoir. It is globally being recorded as an epidemic threat in developing countries in and around the rainy season. In urban setting, flooding of roads during heavy rains expose the persons to *Leptospira* infection. However, in rural areas, farmers working in rice fields are at high-risk of acquiring infection.²⁵

Host

All mammals seem to be prone to at least one of *Leptospira*. The principal reservoir hosts for most *Leptospira* serovars are wild mammals, specifically rodents. Cattle, sheep, goats, dogs and pigs are reservoir hosts among domestic animals and they act as a carrier for several months while rodents usual remain carriers throughout their life. Thus, rodents are considered the main reservoir of infection,²⁶ the particular reservoir hosts differ with the serovar and the geographic region (Table 1). Leptospirosis in reservoir hosts is more probable to be asymptomatic, mild or chronic.²⁷ Most cases of leptospirosis are asymptomatic or mild. The overall case fatality rate is 1-5%. The mortality varies with the form and is higher in the elderly. The icteric form is rarely fatal. The icteric form, which occurs in 5-10% of all patients, 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.²⁸

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 of Infection and Modes of Transmission

Leptospirosis is mostly transmitted by the urine of an infected animal and by contact with skin abrasion. The type of habitats most likely to carry infective bacteria are moist and damp areas like riverbanks, muddy livestock rearing areas where there is a regular passage of wild or farm mammals.²⁹ The disease is, seasonal in temperate climates and year-round in tropical climates and directly with the amount of rainfalls, leptospirosis is reported to be transmitted *via* the semen of the infected animals.³⁰

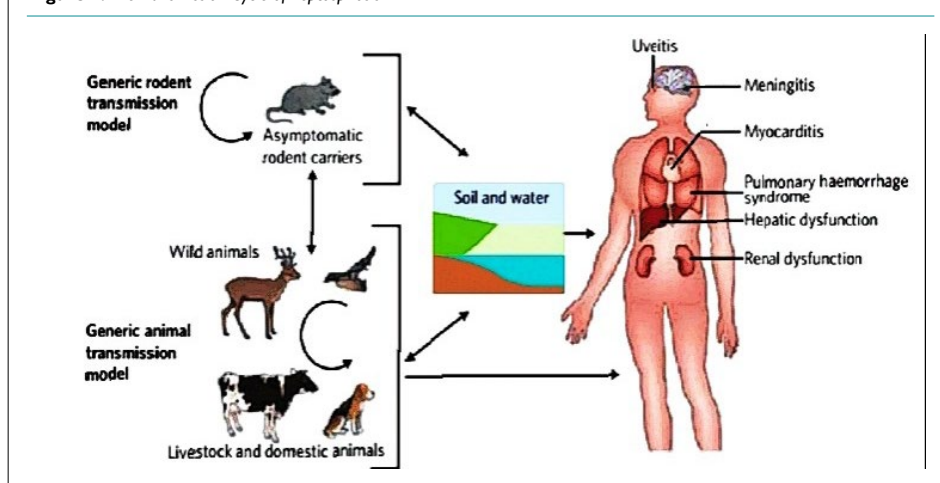
Like humans are considered to be the accidental hosts as a result of direct or indirect contact with leptospirosis infected animals. Besides animal hosts of leptospirosis may be either carrier or reservoir hosts, the latter being the primary source of infection. Although, the presence of animal carrier is considered important in leptospirosis transmission but it can occur through various environmental sources, *leptospirae* being ubiquitous in nature are found everywhere but their primary habitat is renal tubules of carrier animals. Leptospirosis has also been reported in various wild animals such as bats, possums, deer, mongoose and small insectivores.³¹

Direct transmission occurs through leptospirae from tissues, body fluids or urine of acutely infected or asymptomatic carrier animals. Infection with leptospirae is maintained within a population of natural maintenance hosts by vertical and horizontal transmission, such population of natural animal host form the infection reservoir. Leptospirae can also be transmitted from human to human by sexual intercourse, trans-placental from infected mother to the fetus and through breast milk and vision problem and developmental delay in children (Figure 2).³²

Risk Factors

Host and Management risk factors: All age groups of animals can be affected by *Leptospira* infection. However, the leptospirosis is higher in young animals with higher morbidity. Although leptospirosis nearly occurs in all mammalian species, it occurs commonly in cattle, sheep, goats, dogs, horses and pigs, but the disease appears to be rare in cats.³³ Certain management factors that pose risks of infection are diseased animals introduced into herds, com-

Figure 2. The Transmission Cycle of Leptospirosis



mon grazing with the infected one, access to contaminated water provides like streams, rivers, flood or drainage water and buying or loan of infected male animals for natural insemination.³⁴

Pathogenic risk factors: Virulent *Leptospira* resist the anti-bacteria action of complement and neutrophils in immune non-hosts, however, it is rapidly killed by either mechanism within the existing of particular epithelial and endothelial antibody,³⁵ 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 a cytotoxic glycolipoprotein fraction is showing to inhibit host ATPase with the activity ascribed to the presence of long chain fatty acid. *Leptospira Pomona* in cattle causes intravascular hemolysis due to hemolytic exotoxin.³⁶

Public Health Implication of Leptospirosis

Risk factors in human: Leptospirosis infection are occupationally risk groups that are exposed to animal reservoirs or contaminated environments, such as abattoir and sewage workers, salver workers, coalmines, plumbers, farmworkers, veterinarians, slaughter house employees, meat handlers, military personnel and employees within the fishing industry.³⁷ Recreational activities that increase the risk of *Leptospira* infection are husbandry, and water sports like canoeing, swimming and white rafting residents of some urban areas.³⁴ Men are more frequently diagnosed with leptospirosis compared with women and this has been traditionally attributed to the representation of men in high-risk occupational groups.³⁸

PATHOGENESIS

The bacteria enter the body through small abrasions, mucosa, conjunctiva and genital tracts. It involves 33 some chemotactic interactions and a transmembrane passage. The bacteria settle down in the convoluted tubules of the kidney and thus keep on shedding the pathogen in urine. The period of shedding varies from a few weeks to many months. After the bacteria reach a higher concentration in blood and tissue, there is tissue damage due to endotoxins secreted by the pathogen, hemolysis is also secreted by the bacteria and leads to damage of blood cells. Endothelium gets damaged which leads to ischemia and other complications. The exact molecular basis of virulence is yet unknown; however, the humoral response has been observed to be active in the first week of infection leading to phagocytosis by macrophages and neutrophils.³⁹

The primary lesion is damaged to the endothelium of small blood vessels leading to localized ischemia in organs, resulting in renal tubular necrosis, hepatocellular and pulmonary damage, meningitis, myositis and placentitis. Like the incubation period depends on infective dose, growth rate of organisms, their toxicity, and immunity's, the correct characterization of leptospiral pathogenicity is strengthened by using a polyvalent analytical approach that minimizes uncertainties encountered from individual tests especially when phenotypic analysis does not strictly equate with genotypic speciation.⁴⁰

The invasive capacity of *leptospirae* may be related to their pathogenicity because nonpathogenic *leptospirae* do not penetrate cells as deadly as pathogenic *leptospirae*. Damage to the endothelium of small blood vessels may result in ischemic damage to the renal parenchyma³⁶ kidney are involvement in many animal special is chronic and results in elimination of a large number of *leptospirae* in the urine. This is the main source of contamination and infection of human and other mammals. Common symptoms also include malaise, fatigue, headaches, and mild depression also found in cervical and vaginal secretion, semen, breast milk, tears, urine, it is well known for causing disease through human contact with cat feces, and have atypical lymphocytes.

CLINICAL SIGN

The clinical findings in leptospirosis are both highly variable and non-specific, depending on both; host and pathogen factors that significant proportion of infections are; asymptomatic or subclinical, and when symptoms do occur, onset is typically 2 to 30-days after exposure, with average incubation time of 7 to 12-days.^{41,42} In humans the disease is characterized by variety of symptoms, including high fever, vomiting, jaundice (yellow skin and eyes), red eyes, headache, chills, muscle aches, abdominal pain, diarrhea, rash etc. unknown many of these symptoms can be mistaken for other diseases. some infected persons may have no symptoms at all.

The time between a person exposure to a contaminated source and becoming sick is 2-days to 4-weeks. The illness usually begins abruptly with fever and other symptoms. In animal the clinical signs of leptospirosis are often related to kidney this serovar is also maintained in farmed red deer and wapiti areas with a high disease incidence in adult cattle early signs such as fever and depression, a severe pulmonary form of leptospirosis. The clinical findings in leptospirosis are similar in each animal species and do not vary greatly with the specie of *leptospirae* except that infection with *L. interohaemorrhagiae* usually causes severe septicemia.

The clinical finding in the acute form of leptospirosis is maintained by septicemia, with high fever anorexia, petechiation of mucosa depression and acute hemolytic anemia with hemoglobin urea, jaundice and pallor of the mucosa. Milk production is markedly decreased and the secretion is red colored or contains blood clot and the udder is lop and soft. the subacute form of leptospirosis differs from the acute form only in degree. Fever is mild and hemoglobinuria is common but jaundice may or may not be present. the clinical findings in the chronic form of leptospirosis are mild and may be restricted to abortion. Abortion usually occurs during the last trimester of pregnancy.³⁴

Infertility and milk drop occurs only in pregnant or lactating cows because *Leptospira* organisms require pregnant uterus and lactating mammary gland to proliferate. In humans, the disease is characterized by variety of symptoms, including high fever, vomiting, jaundice (yellow skin and eyes), red eyes, abdominal pain, diarrhea, rash many of these symptoms can be mistaken for other diseases. In addition some infected persons may have no symptoms.

Leptospirosis may Occur in Two Phases

Phase 1: The first phase (acute or septic phase) ends after 3-7-days of illness with the appearance of antibodies against *Leptospira* and the disappearance of bacteria from the bloodstream. The Patient is asymptomatic for 3-4-days until the second phase begins with another episode of fever.

Phase 2: The hallmark of the second phase is meningitis (inflammation of the membranes covering the brain) the illness lasts from a few days to 3-weeks or longer. Without treatment, recovery may take several months. Majority of the cases of the leptospirosis are mild form. The classic form of severe leptospirosis is known as Weil's disease, which is characterized by liver damage (causing jaundice), kidney failure, and bleeding. The disease affects brain also causing meningitis, encephalitis of brain tissue with same signs and symptoms; and lung affected as the most serious and life-threatening of all leptospirosis complications. The infection is often incorrectly diagnosed due to the non-specific.

DIAGNOSIS

Leptospirosis cannot be diagnosed alone on the basis of clinical manifestations but easy by laboratory diagnostic methods is required for correct diagnosis. Understanding the course of the disease, right specimen selection and the choice of diagnostic test are the essential factors that help in precise leptospirosis diagnosis, the clinical diagnosis of leptospirosis must be supported by a variety of laboratory tests such as microbiological, immunological, and molecular techniques.⁴³

Like on infection the bacteria can be found in blood and cerebrospinal fluid (CSF) for the first 7 to 10-days and then moving to the kidneys. After 7 to 10-days the microorganism can be found in fresh urine because of the large range of symptoms, it can be incorrectly diagnosed as another disease, which is early diagnostic efforts, include isolation from blood or other clinical materials through culture of pathogenic leptospires.³⁶

Diagnosis of leptospirosis is confirmed with tests such as enzyme-linked immunosorbent assay (ELISA)⁴⁴ and polymerase chain reaction (PCR).⁴⁵ Because clinical examination of leptospirosis are very non-specific and have significant confuse with a variety of other febrile illnesses, diagnostic testing because clinical manifestations of leptospirosis are very non-specific and have significant overlap with a variety of other febrile illnesses a combination of exposure history and symptoms should prompt confirmatory testing. However clinical suspicion alone may be enough to warrant empiric antibiotic treatment in many cases. In general, definitive diagnosis of leptospirosis can be made *via* either traditional microbiological methods (direct detection, culture) or (serology).²¹ *Leptospira*, like other spirochetes, stains poorly with traditional staining methods and is best visualized with dark field microscopy, however, sensitivity and specificity are both poor when examining clinical samples.⁴⁶

Culture

Leptospira from patient samples is also challenging: the organisms

typically take 1-2-weeks. to grow, but may take over a month, and special growth media is required, often necessitating advance notice to the lab. though specificity of culture is excellent sensitivity is very poor samples for culture should be collected prior to the administration of antibiotics.⁴⁷ Samples should be stored and transported at ambient temperatures since low temperatures are detrimental to pathogenic leptospires.⁴⁸ *Leptospira spp.* stain poorly with the Gram stain and are not observed by microscopy unless special stains or methods are employed. Silver staining or immunogold-silver staining is sometimes useful as an adjunct technique. Dark field microscopy can also be used to detect *Leptospira*.²¹

Serology Based Methods

The most commonly used means of confirming a diagnosis of leptospirosis and also The "gold standard" is the microscopic agglutination test (MAT), in which acute and convalescent sera from a suspected case is mixed with a panel of live antigens from different serogroups of *Leptospira* organisms and examined for agglutination.⁴² While there is some variability amongst labs/references, most commonly, a single titer of 1:100 (range is 1:100 to 1:800), or a fourfold rise in titer between acute and convalescent sera, serologically confirms the diagnosis of leptospirosis.⁴⁶

Microscopic Agglutination Test

The microscopic agglutination test (MAT) is still considered the gold standard test in diagnosing leptospirosis according to Centers for Disease Control and Prevention (CDC). Although Live *Leptospira* are to be cultured regularly and agglutination reaction is carried out to check the species and serovar of the pathogen, like it is a very laborious and expensive method and needs live bacterial antigen to test, that's why this test is only conducted in specially designed labs.^{49,50}

Several tests based on antigen-antibody agglutination are being used currently for the diagnosis of leptospirosis. It is considered to be serogroup/serovar specific when carried out on paired sera. However, microscopic agglutination test involves the treatment of patient serum sample containing leptospiral antibodies with live antigen. The agglutination between antibody and live antigen is observed under dark field microscope maximum agglutination has occurred.⁵¹

When no free leptospires are observed under the dark field microscope, maximum agglutination has occurred. The patient serum samples are continuously diluted till maximum agglutination has occurred and no free leptospirosis are available. The final serum dilution is the one at which 50% or more leptospires have undergone agglutination. Even though a number of studies have been carried out which exhibits the variable sensitivity and specificity of MAT in leptospirosis diagnosis. The sensitivity of MAT in diagnosing leptospirosis in the acute phase is considered very less.⁵²

Enzyme-Linked Immune Sorbent Assay

The family *Leptospiraceae* consists of three genera *via*, *Leptospira*,

Leptonema and Turneria. *Leptospira* consists of many pathogenic and non-pathogenic serovars characterized under it. Enzyme linked immune sorbent assay is considered to be the easy and simplest method used for leptospirosis diagnosis and also more sensitive than most of the serology based tests used for conventional diagnosis of leptospirosis.⁵³

The ELISA test is much more accurate than other tests and has much advantage from point of view of laboratory practices, it has excellent diagnostic specificity and sensitivity, convenient technical feature including automation and can be used efficiently as screening test for large number of serum samples.⁵⁴ Although, it is easy, safe and can detect IgM and IgG.⁵⁵ It can be used in humans and animals.⁵⁶ Elisa can be performed with minimal training and typically provides results in 2-4 hrs.⁵⁰ It has been recommended for the rapid diagnosis of leptospirosis in endemic areas.⁵⁷

TREATMENT

The primary aim of treatment of leptospirosis is to control the infection before irreparable damage to the liver and kidneys occurs. Treatment with antibiotics counseled as before long as doable when signs seem. The results of treatment are often disappointing because in most instances, animals are present for treatment only when the septicemia has subsided. The secondary aim of treatment is to regulate the leptospiruria of carrier animals and render them safe to stay within the group. Other antibiotics used to treat leptospirosis include tetracycline, penicillin, ampicillin, doxycycline, streptomycin and erythromycin.⁸

The efficacy of treatment may depend on the serovar. Fluid therapy, blood transfusion and other supportive care may also be necessary. These supportive treatments depend on the animal and needed if the animal is severely affected and in shock, it will need fluid therapy. In beef herds, further abortions prevented by vaccination and treatment of all animals with antibiotics and in dairy cattle, only infected animals usually treated due to the potential loss of milk sales.⁵⁸

CONTROL AND PREVENTION

Leptospirosis has been an underreported disease and there are a few reliable global incidence data as the disease is not detected in common laboratory tests. Like as the symptoms are very much similar to many other diseases that can confuse and the bacteria being destroyed by common antibiotics, Leptospirosis is very hard to detect in a population. Although the risk of leptospirosis can be greatly avoiding water contaminated with animal urine or contact with potentially infected animals. Like occupational workers should wear protective clothing or footwear. Any skin cuts should be covered with waterproof dressing while swimming in fresh water to protect against a range of infections. In pet dogs, vaccination against the bacteria protects the animal and the household, human immunization helps to provide a certain degree of protection against infection.⁵⁹

The complete elimination of the disease seems unfeasible as many species of rodents serve as reservoir of infection. Hence,

proper strategies for prevention of leptospirosis are based on awareness of leptospirosis epidemiology and transmission mechanisms. It is possible to prevent the disease by reducing exposure and implement protective measures, immunization, and pre- or post-exposure chemoprophylaxis. Housing construction that prevents entry of rodents from invading residential living areas and water sources greatly reduces risk of infection. Swimming in contaminated water, and walking barefoot in floodwater should be avoided.⁴³

In 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.²⁶

CONCLUSION AND RECOMMENDATION

Leptospirosis is the most common zoonosis of global relevance, caused by *Leptospira* interrogans, which are also the most pathogenic. Among the spirochetes, *Leptospirae* is unusual. It has an impact on both humans and animals. Although the bacteria are usually spread by infected animals' urine and skin abrasions, the bacteria enter the body through tiny abrasions, mucosa, conjunctiva, and genital tracts in animals like rats and mice. Pathogenic *Leptospira* lives in the kidneys of natural hosts, mostly mammals, and is expelled with the urine into the environment, where it can persist for several months under favorable conditions. Antibiotics are effective treatments for leptospirosis, and current leptospirosis vaccinations for domestic animals can help to reduce the disease's severity.

Therefore the scarcity of information regarding to leptospirosis in African countries, including Ethiopia. Leptospirosis is seriously contaminated urine is highly infectious for people and for susceptible animal species; therefore, contact with urine on mucous membranes or skin abrasions should be avoided. When handling infected animals or dealing with areas of contamination, protective gear should be worn such as gloves, eye protection and face masks. Although, easy to treat with most antibiotics, Leptospirosis is easy to diagnosis by microscopic, culture and serology.

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

- It is important to conduct applicable control techniques and increasing the public awareness about zoonotic transmission of leptospirosis is suggested.
- Besides, any study and control strength should be conducted by collaboration among human, animal and environmental health professions.
- 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 glove when exposed to animal reservoirs and contaminated environments.

- Government should involve in funding the research to be done on the status of leptospirosis in developing country including Ethiopia.
- Proper control measures and public awareness should be made in endemic areas.

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REFERENCES

- Mwachui MA, Crump L, Hartskeerl R, Zinsstag J, Hattendorf J. Environmental and behavioral determinants of leptospirosis transmission: A systematic review. *PLoS Neglected Tropical Diseases*. 2015; 9(9): e0003843. doi: [10.1371/journal.pntd.0003843](https://doi.org/10.1371/journal.pntd.0003843)
- Pappas G, Papadimitriou P, Siozopoulou V, Chistou L, Akritidis N. The globalization of leptospirosis; worldwide incidence trends. *Int J Infect Dis*. 2008; 12: 351-357. doi: [10.1016/j.ijid.2007.09.011](https://doi.org/10.1016/j.ijid.2007.09.011)
- Smith RM, Zochowski WJ. Leptospirosis. In: Palmer SR, Solusby L, Torgerson PR, David WGB, eds. *Zoonosis Biology, Clinical Practice, and Public Health Control*. 2nd ed. NY, USA: Oxford University; 2011.
- Robi DT. Epidemiology and zoonotic implication of leptospirosis in domestic animals in Ethiopia. *Academic Journal of Animal Diseases*. 2020; 9(1): 19-32. doi: [10.5829/idosi.ajad.2020.19.32](https://doi.org/10.5829/idosi.ajad.2020.19.32)
- Tewodros F, Makash A. Leptospirosis and its public significance: A review. *Eur J Appl Sci*. 2012; 4(6): 238-244. doi: [10.5829/idosi.ejas.2012.4.6.66162](https://doi.org/10.5829/idosi.ejas.2012.4.6.66162)
- Pal M, Hadush A. Leptospirosis: An infectious emerging waterborne zoonosis of global significance. *Air Water Borne Dis*. 2017; 6: 133. doi: [10.4172/2167-7719.1000133](https://doi.org/10.4172/2167-7719.1000133)
- Ahmad SN, Shah S, Ahmad FM. Laboratory diagnosis of leptospirosis. *J Postgrad Med*. 2005; 51: 195-200.
- Heymann DL. *Control of Communicable Diseases, Manual*. 18th ed. Washington, D.C., USA: American Public Health Association. 2004: 306-309.
- Tilahun Z, Reta D, Simenew K. Global epidemiological overview of leptospirosis. *International Journal of Microbiology Research*. 2013; 4(1): 9-15. doi: [10.5829/idosi.ijmr.2013.4.1.7134](https://doi.org/10.5829/idosi.ijmr.2013.4.1.7134)
- Lucheis SB, Ferreira RS. Ovine leptospirosis in Brazil. *Journal of Venomous Animals and Toxins including Tropical Diseases*. 2011; 17(4): 394-405. doi: [10.1590/S1678-91992011000400006](https://doi.org/10.1590/S1678-91992011000400006)
- Dhanze H, Suman MK, Mane B. Epidemiology of leptospirosis, An Indian perspective. *Journal of Food Borne and Zoonotic Diseases*. 2013; 1(1): 6-13.
- Wang Y, Zhunang X, Zhong Y, et al. Distribution of plasmids in district leptospira pathogenic species. *PLoS Negl Trop Dis*. 2015; 9(11): e0004220. doi: [10.1371/journal.pntd.0004220](https://doi.org/10.1371/journal.pntd.0004220)
- Shivakumar S. Leptospirosis-current scenario in India. In: *Medicine Update*. 2010; 18: 799-809. doi: [10.13140/2.1.4905.6968](https://doi.org/10.13140/2.1.4905.6968)
- Deanna H. Leptospirosis: A waterborne zoonotic disease of global importance. *Georgia Epidemiology Report*. 2006; 22: 8.
- Doern G. Detection of selected fastidious bacteria. *Clin Infect Dis*. 2000; 30: 166-173. doi: [10.1086/313586](https://doi.org/10.1086/313586)
- Plank R, Dean D. Overview of the epidemiology, microbiology and pathogenesis of Leptospira spp. in humans. *Microbes Infect*. 2000; 2(10): 1265-1276. doi: [10.1016/s1286-4579\(00\)01280-6](https://doi.org/10.1016/s1286-4579(00)01280-6)
- World Health Organization (WHO). Human Leptospirosis: Guidance for diagnosis, surveillance and control. 2003. Web site. <https://www.who.int/publications/i/item/human-leptospirosis-guidance-for-diagnosis-surveillance-and-control>. Accessed October 12, 2021.
- Fraser C, Norris S, Weinstock G. Complete genome sequence of Treponema pallidum, the syphilis spirochete. *Science*. 1998; 281: 375-388. doi: [10.1126/science.281.5375.375](https://doi.org/10.1126/science.281.5375.375)
- Adler B, Moctezuma A. Leptospira and leptospirosis. *Veterinary Microbiology*. 2010; 140: 287-296. doi: [10.1016/j.vetmic.2009.03.012](https://doi.org/10.1016/j.vetmic.2009.03.012)
- Johnson RC, Faine S. Leptospira. In: Krieg NR, Holt JG, eds. *Bergey's Manual of Systematic Bacteriology*. Philadelphia, USA: Williams & Wilkins; 1984; 1: 62-67.
- Ajaj EA, AL-Farwachi MI. Detection of bovine leptospirosis using different conventional laboratory tests in Nineveh province, Iraq. *J Anim Health Prod*. 2013; 1(3): 32-35. doi: [10.13140/RG.2.2.21052.69768](https://doi.org/10.13140/RG.2.2.21052.69768)
- Evangelista KV, Coburn J. Leptospira as an emerging pathogen: A review of its biology, pathogenesis and host immune responses. *Future Microbiol*. 2010; 5(9): 1413-1425. doi: [10.2217/fmb.10.102](https://doi.org/10.2217/fmb.10.102)
- Costa F, Hagen JE, Calgano J, et al. Global morbidity and mortality of leptospirosis: A systematic review. *PLoS Negl Trop Dis*. 2015; 9: e0003898. doi: [10.1371/journal.pntd.0003898](https://doi.org/10.1371/journal.pntd.0003898)

24. Abela-Ridder B, Sikkema R, Hartskeerl R. Estimating the burden of human leptospirosis. *Int J Antimicrob Agents*. 2010; 36: 5-7. doi: [10.1016/j.ijantimicag.2010.06.012](https://doi.org/10.1016/j.ijantimicag.2010.06.012)
25. Lau C, Smythe L, Craig S, Weinstein P. Climate change, flooding, urbanization and leptospirosis: Fueling the fire. *Trans R Soc Trop Med Hyg*. 2010; 104: 631-638. doi: [10.1016/j.trstmh.2010.07.002](https://doi.org/10.1016/j.trstmh.2010.07.002)
26. Waktole Y, Bashahun G, Nejash A. Leptospirosis in animal and its public health implications: A review. *World Appl Sci J*. 2016; 34(6): 845-853. doi: [10.5829/idosi.wasj.2016.34.6.103113](https://doi.org/10.5829/idosi.wasj.2016.34.6.103113)
27. Office International des Epizooties (OIE), 2005. Institute for International Cooperation in Animal Biology. Iowa State University, College of Veterinary Medicine, Web site: <http://www.cfs.ph.IA.state.edu>. Accessed October 12, 2021.
28. Farrelly HE, Adler B, Faine S. Opsonic monoclonal antibodies against lipopolysaccharide antigens of leptospira interrogans serovar hardjo. *J Med Microbiol*. 1987; 23: 1-7. doi: [10.1099/00222615-23-1-1](https://doi.org/10.1099/00222615-23-1-1)
29. Kohn B, Steinicke K, Arndt G, et al. Pulmonary abnormalities in dogs with leptospirosis. *J Vet Intern Med*. 2010; 24(6): 1277-1282. doi: [10.1111/j.1939-1676.2010.0585.x](https://doi.org/10.1111/j.1939-1676.2010.0585.x)
30. Wasiński B, Dutkiewicz J. Leptospirosis—current risk factors connected with human activity and the environment. *Ann Agric Environ Med*. 2013; 20(2): 239-244.
31. Ellis WA. Animal leptospirosis. *Curr Top Microbiol Immunol*. 2015; 387: 99-137. doi: [10.1007/978-3-662-45059-8_6](https://doi.org/10.1007/978-3-662-45059-8_6)
32. World Health Organization (WHO). Report of the first meeting of the leptospirosis burden epidemiology Reference Group, WHO Library Cataloguing-in- Publication Data. Web site: http://apps.who.int/iris/bitstream/handle/10665/44382/9789241599894_eng.pdf;jsessionid=C77D7B6387ED10A9A81F68CBE5C280D6?sequence=1. 2010: 1-34.
33. Levett PN. Systematics of leptospiraceae. In: Adler B, ed. *Leptospira and Leptospirosis*. Berlin: Springer; 2015: 11-20.
34. Radostits O, Gay C, Hinchcliff K, Constable P. *Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats*. London, Saunders: Elsevier Health Sciences; 2006: 1094-1110.
35. Fentahun T, Alemayehu M. Leptospirosis and its public health significance: A review. *Eur J Appl Sci*. 2012; 4(6): 238-244.
36. Craig SB, Graham GC, Burns MA, Dohnt MF, Smythe LD, et al. Hematological and clinical-chemistry markers in patients presenting with leptospirosis: A comparison of the findings from uncomplicated cases with those seen in the severe disease. *Ann Trop Med Parasitol*. 2009; 103: 333-341. doi: [10.1179/136485909X435058](https://doi.org/10.1179/136485909X435058)
37. Katz A, Ansdell V, Effler P, Middleton C, Sasaki D. Leptospirosis in Hawaii, 1974-1998, epidemiologic analysis of 353 laboratory-confirmed cases. *Am J Trop Med Hyg*. 2002; 66: 61-70. doi: [10.4269/ajtmh.2002.66.61](https://doi.org/10.4269/ajtmh.2002.66.61)
38. Langston, C, Heuter K. Leptospirosis, A re-emerging zoonotic disease. *Vet Clin North Am Small Anim Pract*. 2003; 33(4): 791-807. doi: [10.1016/s0195-5616\(03\)00026-3](https://doi.org/10.1016/s0195-5616(03)00026-3)
39. Mohammed H, Nozha C, Hakim K, Abdelaziz F, Reikia B. Leptospira: Morphology, classification and pathogenesis. *Journal Bacteriology Parasitology*, 2011; 2: 120. doi: [10.4172/2155-9597.1000120](https://doi.org/10.4172/2155-9597.1000120)
40. Mgone GF, Machang'u RS, Collares-Pereira M, et al. Challenges in determining the pathogenicity status of Leptospira isolates with phenotypic methods: The need for a polyvalent approach. *African Journal of Microbiology Research*. 2010; 4: 2528-2533.
41. Hirsh DC, Maclachlan NJ, Waller RL. *Veterinary Microbiology*. 2nd ed. NY, USA: Black Well; 2004: 148-152.
42. Haake DA, Levett PN. Leptospirosis in humans. *Curr Top Microbiol Immunol*. 2015; 387: 65-97. doi: [10.1007/978-3-662-45059-8-5](https://doi.org/10.1007/978-3-662-45059-8-5)
43. Pal M. *Zoo Noses*. 2nd ed. Jaipur, India: Satyam Publishers; 2007.
44. Ajaj EAB, AL-Farwachi MI. Serosurvey of leptospira interrogans serovars hardjo and pomona in cattle in nineveh province, Iraq. *Animal Health Prod and Hyg*. 2013; 2(1): 156-158.
45. Ayyadevare S, Thaden JJ, Shmookler RJ. Discrimination of primer 39-nucleotide mismatch by taq DNA polymerase during polymerase chain reaction. *Anal Biochem*. 2000; 284: 11-18. doi: [10.1006/abio.2000.4635](https://doi.org/10.1006/abio.2000.4635)
46. Schreier S, Doungchawee G, Chadsuthi S, Triampo D, Triampo W. Leptospirosis: Current situation and trends of specific laboratory tests. *Expert Rev Clinical Immunology*. 2013; 9: 263-280. doi: [10.1586/eci.12.110](https://doi.org/10.1586/eci.12.110)
47. Musso D, La Scola B. Laboratory diagnosis of leptospirosis: A challenge. *J Microbiol Immunol Infect*. 2013; 46: 245-252. doi: [10.1016/j.jmii.2013.03.001](https://doi.org/10.1016/j.jmii.2013.03.001)
48. Draghi M, Brihuega B, Benitez D, Sala J, Biotti G, Pereyra M. Leptospirosis outbreak in calves from Corrientes Province, Argentina. *Rev Argent Microbiol*. 2011; 43: 42-44. doi: [10.1590/S0325-75412011000100009](https://doi.org/10.1590/S0325-75412011000100009)
49. Mosby. *Mosby's Medical Dictionary*. 9th ed. Amsterdam, Netherlands: Elsevier Health Sciences; 2013: 697.
50. Picardeau M, Bertherat E, Janclous M, Skouloudis AN, Durski K, Hartskeerl RA. Rapid tests for diagnosis of leptospirosis: current tools and emerging technologies. *Diagn Microbiol Infect Dis*. 2014; 78(1): 1-8. doi: [10.1016/j.diagmicrobio.2013.09.012](https://doi.org/10.1016/j.diagmicrobio.2013.09.012)

51. Mulla S, Chakra borty T, Patel M, Pandya HP, Dadhaniya V, Vaghela G. Diagnosis of leptospirosis and comparison of ELISA and MAT techniques. *Indian J Pathol Microbiol.* 2006; 49: 468-470.
52. Limmathurotsakul D, Turner EL, Wuthiekanun V, et al. Fool's gold: why imperfect reference tests are undermining the evaluation of novel diagnostics: A re-evaluation of 5 diagnostic tests for leptospirosis. *Clinical Infectious Diseases.* 2012; 55: 322-331. doi: [10.1093/ cid/cis403](https://doi.org/10.1093/cid/cis403)
53. Wasiński B, Pajsek Z. Occurrence of leptospiral infections in swine population in Poland evaluated by ELISA and microscopic agglutination test. *Pol J Vet Sci.* 2010; 3: 695-699. doi: [10.2478/v10181-010-0006-3](https://doi.org/10.2478/v10181-010-0006-3)
54. Hirsh DC, Maclachlan NJ, Waller RL. *Veterinary Microbiology.* 2nd ed. NY, USA: Black Well; 2004: 148-152.
55. Chen H, Zhang Z, Halsey E, et al. Detection of *Leptospira*-specific antibodies using a recombinant antigen-based enzyme-linked immunosorbent assay. *Am J Trop Med Hyg.* 2013; 89: 1088-1094. doi: [10.4269/ajtmh.13-0041](https://doi.org/10.4269/ajtmh.13-0041)
56. Sabir M, Hatta M. Detection of IgM to leptospira agent with ELISA and leptodistick method. *Bio Celebes.* 2011; 5: 8-12.
57. Desakorn V, Wuthiekanun V, Thanachartwet V, et al. Accuracy of a commercial IgM ELISA for the diagnosis of human leptospirosis in Thailand. *Am J Trop Med Hyg.* 2012; 86: 524-527.
58. Guidugli F, Castro A, Atallah A. Antibiotics for leptospirosis. *Cochrane Database Syst Rev.* 2006; (2): CD008264. doi: [10.1002/14651858.CD008264.pub2](https://doi.org/10.1002/14651858.CD008264.pub2)
59. McBride AJA, Athanazio DA, Reis MG, Ko AI. Leptospirosis. *Curr Opin Infect Dis.* 2005; 18: 376-386. doi: [10.1097/01.qco.0000178824.05715.2c](https://doi.org/10.1097/01.qco.0000178824.05715.2c)