Review Article

LEPTOSPIROSIS – AN ENIGMATIC ZOONOSIS

IJCRR
Section: Healthcare
Sci. Journal
Impact Factor
4.016

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ABSTRACT

Leptospirosis is a zoonosis which has a global distribution. The disease is still an enigma 130 years post discovery. Leptospirosis is mainly a disease of the animals like the rodents and various other animals, wherein man is an accidental host. *Leptospira* is a spirochete, which to be visualised in a dark field microscope or in a light microscope after special staining techniques. It is a slow growing organism and requires special media like the Fletcher`s medium. Leptospirosis results when the person comes in contact with the organism either as an occupational hazard or during avocational exposure or during floods. The pathogenesis of leptospirosis is incompletely understood. Despite years of speculation, the route and mode of entry of leptospires in natural infections is not clear. The site of entry into host is through mucosal surfaces. Important portals of entry are fresh or partially healed abrasions of the skin and intact mucosa of the buccal cavity, nasal passages or conjunctiva. The disease can be seen in two forms, the mild anicteric form and the severe icteric form also known as Weil`s disease. The diagnosed leptospirosis cases are just the tip of the iceberg; many go undiagnosed due to the protean manifestations of the disease. The drug of choice for treatment is Penicillin. Vaccines are available for animals. An approved vaccine for humans is still awaited.

Key Words: Potentially fatal, Leptospirosis, Anicteric leptospirosis, *Leptospira icterohaemorrhagiae*

INTRODUCTION

Leptospirosis is recognized as the most common zoonotic infection in the world.[1] It is caused by infection with pathogenic *Leptospira* species.[2] Leptospirosis is a fairly common disease in humid and warm climates.[3] A wide spectrum of human disease is caused by leptospires, ranging from an asymptomatic subclinical infection to a life threatening multiorgan infection.[2] In man anicteric leptospirosis is an acute febrile disease with few complications.[4] The severe, potentially fatal, icteric leptospirosis also known as Weil`s syndrome is typically characterized by renal, hepatic and vascular complications.[5] Leptospirosis is often under diagnosed because of its protean clinical manifestations leading to significant morbidity and mortality.[6] The diagnosis of leptospirosis can be established by demonstration of organism and detection of antibodies.[7]

HISTORY

Over 100 years ago Adolf Weil in Heidelberg first reported the syndrome of icteric leptospirosis with renal failure.[2] In the year 1883 leptospirosis was noted to be an occupational disease of the sewer workers .[8]

The term Weil's disease was used first by Goldschmidt in 1887.[9] In 1907, Stimson stained tissues from a patient using the Levaditi technique for staining spirochetes in tissue sections, the kidneys contained spiral organisms with hooked ends which he called "Spirocheta interrogans".[10] *Leptospira icterohaemorrhagiae* was first demonstrated as the cause of Weil's disease in Japan in 1914.[11] During 1914-15 in Japan Weil's disease was common among coal miners. [10] Inada and colleagues succeeded in transmitting the infection to guinea-pigs, from the blood of which they were able to grow an organism, a spirochete which they called "*Spirocheta icterohaemorrhagiae*". [10] In the year 1916 the causative agent was identified by Inada et al in Japan. [8] The organism was recovered for the first time by Noguchi in the year 1917 from a Norway rat. [12] The name '*Leptospira*' was proposed by Noguchi in 1918. [13] In the year 1929 Taylor and Goyal reported the first case of leptospirosis in India from Andaman and Nicobar islands.[14] The two forms of leptospirosis -anicteric and icteric were recognized by Feigin and Anderson in 1975. [4]

MORPHOLOGY

Leptospires are tightly coiled spirochetes, usually 6-20um X 0.1um by length X breadth, the cells have pointed ends bent into a distinctive hook. [2] The organisms are characterized by a flexible thread like structure consisting of a large number of fine, regular spiral coils.[7] Due to their narrow diameter,

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Received: 15.06.2015 **Revised:** 05.02.2016 **Accepted:** 23.08.2016

dark-field illumination or phase contrast microscopy is ideal to visualize the leptospires.[13] Leptospires can also be visualized by electron microscopy.[15]

There is an outer envelope, surrounding a cell wall or peptidoglycan complex, wound in a helical shape.[10] Based on the morphology the free living (*Leptospira biflexa*) and parasitic leptospires (*Leptospira interrogans*) cannot be distinguished. [13]

RESISTANCE

Leptospires do not survive drying, as in dehydrated cultures, tissues or contaminated water or urine, or in the environment, consistent with their essentially aquatic nature. [10] They are relatively easily preserved by lyophilisation of cultures. [10] Leptospires survive in water and culture media for long periods. [10] Leptospires are able to survive in alkaline soils, mud, swamps, streams and rivers, organs and tissues of live or dead animals. [10]

All leptospires are sensitive to acid, at pH of 6.8 or lower, but they survive alkaline conditions of up to pH 7.8-7.9. [10]

CULTURAL CHARACTERISTICS

Leptospires require an optimum temperature of 28° C to 30° C .[2] Cultures are incubated at 28° C to 30° C in the dark for 6 weeks or longer.[12] *Leptospira* are slow growing organisms having a generation time of approximately 24 hrs at 30° C.[16] Optimum growth occurs in the pH range 7.2-7.6. [10] Under aerobic conditions growth can occur from very small numbers, in adequate media and under optimum conditions. [10] Growth is best checked by observing under dark field microscopy. [10] Leptospires grow well in tubes of semi-solid media containing 0.1-0.3% agar, following inoculation of one or more drops onto the surface or stab inoculation into the depths. [10] Growth appears after a variable period as a disc, known as Dinger`s disc, which is about 0.2-0.5 mm thick. [10]

Leptospires are fastidious in their nutritional requirement and need addition of an animal protein in the form of fresh rabbit serum or bovine serum albumin fraction V for their growth.[1] Vitamins B1 and B 12, and long chain fatty acids are the only organic compounds required for their growth. [13]

A wide variety of media have been described:

Liquid media

Media containing serum- Traditional media containing approximately eight to ten % rabbit serum e.g. Stuart`s, Korthof`s and Fletchers media. [17]

Media without serum

a) Media containing protein: a serum-free oleic-acid albumin medium, and derivatives containing tweens as the sources of fatty acids, with bovine serum albumin (BSA) as a detoxifier, such as the widely used, commercially available Ellinghausen-McCullough-Johnson-Harris medium (EMJH). [10] Low-protein media containing 0.1% or 0.01% BSA are sometimes used as a compromise to cultivate strains which cannot be adapted to protein-free media or for maintenance of strains in protein-free medium. [10]

b) Media without protein: are those in which conditions are balanced and ingredients selected or purified so as to avoid toxicity. [10] Their main application is in use for vaccines where BSA is unacceptable because of the risks of hypersensitivity or auto-immune reactions in vaccinated animals or people. [10]

Solid media

Any liquid medium can be solidified by the addition of agar. [10] The usual concentrations for semi-solid media are 0.1-0.2% agar. [10] Special selective media have been developed for the primary isolation of leptospires from animal tissues and organs, based on the premise that some leptospires could be more easily cultivated on primary inoculation if the media were more suited to their fastidious nutritional and growth requirements and the risk of contamination avoided on prolonged incubation. [10] These include 5-fluorouracil; a combination of nalidixic acid, vancomycin, and polymyxin-B-sulfate; or rifampicin. [10]

Pathogenesis

The pathogenesis of leptospirosis is incompletely understood. [18] Despite years of speculation, the route and mode of entry of leptospires in natural infections is not clear. [10] The site of entry into host is through mucosal surfaces. [19] The main modes of entry are fresh or recent abrasions of the skin and even intact mucosa of the buccal cavity, conjunctiva or nasal passages. [19] They may also enter directly into the bloodstream or lymphatics via the conjunctiva; the genital tract in some animals; the nasopharyngeal mucosa, possibly through the cribriform plate; the lungs following inhalation of aerosols; or through invasion of the placenta from mother to fetus at any stage of pregnancy in mammals.[10]

The organisms enter the blood stream where they multiply and this process is accompanied by the development of transient fever. [18] At the same time the bacteria also start acting upon other organs and further symptoms depend on the organ which is affected.[18] Leptospires take just a few days to establish in organs like liver, spleen, kidney and the pathological changes are initiated there. [19] By the time the immune system gets activated, leptospires get established in the parenchyma of the liver and spleen and in tubular region of the kidneys where they may persist. [19]

Potential virulence factors include attachment, toxin production, immune mechanisms and surface proteins Leptospiral lipopolysaccharide exhibits weak endotoxic activity but a number of serovars produce haemolysins, which may act as sphingomyelinases, phospholipases or pore forming proteins.[20]

The primary lesion in leptospirosis is disruption of the integrity of the cell membrane of the endothelial cells lining small blood vessels in all parts of the body resulting in capillary leakage and haemorrhages. [13] Widespread petechial haemorrhages are apparent in all organs and tissues, particularly the lungs, omentum and pericardium. [13] Ischaemia from damage to blood vessels in the renal cortex leads to renal tubular necrosis. [13] The resulting anatomical damage causes renal failure that can be fatal. Ischaemia results in liver cell necrosis, which leads to the characteristic icterus of severe leptospirosis. [13]

Following formation of antibodies, the leptospires are removed from all sites other than the proximal renal tubules, brain and the eye where they can persist for a period ranging from few weeks to months. [18]

Leptospires enter the cerebrospinal fluid (CSF) in the early septicaemia phase of the illness. [13] The anterior chamber of the eye is invaded by leptospires during acute infection. [13] The leptospires can persist in specific immunologically privileged sites, even after antibodies and phagocytes have cleaved the leptospires have been cleaved from all other sites. [13] The most significant site of persistence is the renal tubule. [13] Leptospires are excreted by animals regularly or intermittently for months to years, sometimes even throughout their lifespan. [13] However, humans do not remain carriers for long, and the urine is free of leptospires at the time of clinical recovery. [13]

Diagnostically significant bacteraemic phase lasts for about 1-7 days. [10] Once immunity develops, leptospires are removed from the circulation and from tissues and organs by phagocytosis, following opsonisation. [10]

Clinical features

The spectrum of human disease caused by leptospires is extremely wide, ranging from subclinical infection to a severe syndrome of multiorgan infection with high mortality .[2] As a result of its protean clinical manifestations and non specific presentations, leptospirosis has been under diagnosed and frequently misdiagnosed as other diseases such as influenza, viral hepatitis, encephalitis, pneumonitis and acute renal failure.[21] Weil`s disease is only one of the many manifestations of leptospiral infection in man.[4] In most cases leptospirosis presents as a mild flu like illness. [22] Leptospirosis occurs as two clinically recognizable syndromes. [13] Anicteric leptospirosis is the most common syndrome, a self-limited illness seen in almost 90% of the total cases. [13] Icteric leptospirosis or Weil's syndrome is a more serious, potentially fatal syndrome and occurs in 5% to 10% of the cases. [13] Even though subclinical infection is not common, serological testing has shown that it could occur in people following occupationally exposure to leptospires.[13] After the incubation period, an acute leptospiraemic phase is followed by an immune phase. [18] The distinction between the first and second phase is not always clear and milder cases do not always include the second phase. [18]

Anicteric leptospirosis

The incubation period for leptospirosis is usually 7 to 12 days, but it can range from 2 to 20 days. [13] The onset of anicteric leptospirosis is abrupt and is characterized by fever, headache, severe myalgia, chills with rigors, prostration and sometimes, circulatory collapse. [13] The septicemic (or first) phase lasts 3 to 7 days. [13] Fever is high and remitting. Headache is intense, unremitting and possibly throbbing. [13] Anorexia, nausea, vomiting and abdominal pain occur in most patients. [13] The most common physical finding is conjunctival suffusion in the absence of purulent discharge. [13] Other signs include maculopapular skin rash, pharyngeal injection, lymphadenopathy, splenomegaly, hepatomegaly, and muscle tenderness.[13] Cervical, axillary and mediastinal lymph nodes may be enlarged.[8] The symptoms are prominent for 4 to 7 days during the septicemic stage. [13] Leptospires can be isolated from the blood and the CSF during this phase. [13] The immune stage of anicteric leptospirosis is preceded by a one to three-day asymptomatic period. [13] The onset of the immune stage coincides with the appearance of IgM antibodies. [13] The duration of the immune stage ranges from 4 to 30 days, and the leptospires are cleared from the blood and the CSF after this stage. [13] Leptospiruria develops and persists for 1 to 3 weeks. [13] Aseptic meningitis is the hallmark of the immune stage.[13] Leptospiral meningitis accounts for 5 - 40% of all cases of aseptic meningitis. [23] Uveitis, iritis, iridocyclitis and chorioretinitis may also appear during the immune stage. [13]

Icteric leptospirosis

Icteric leptospirosis or Weil's syndrome is a form of disease characterized by symptoms of hepatic, renal and vascular dysfunction. [13] Jaundice remains the hallmark of Weil syndrome, the intensity of jaundice varies. [12] Jaundice may appear as early as the third day of illness or may not appear until the second week.[12] The clinical manifestations vary in terms of severity and symptomatology. [13] Supportive therapy has reduced the mortality to between 5% and 10%. [13] During the leptospiraemic phase of icteric leptospirosis, the symptoms do not suggest leptospirosis until the third to seventh day of illness, when jaundice and azotaemia develops. [13] The biphasic course of the disease is obscured by severe and

persistent fever, jaundice and azotaemia. [13] Jaundice appears, but there is no evidence of hepatocellular destruction. [13] Hepatic dysfunction occurs, but it resolves and it is rarely the cause of death. [13] Azotaemia, oliguria and anuria commonly occur during the second week of illness. [13]

Epidemics of leptospirosis in Korea, Brazil and Nicaragua were characterised by massive pulmonary haemorrhages, including fatal sudden haemoptysis. [10] Haemorrhages of varying severity frequently occur in any organ or tissue. [10] They may bleed into the lumina of the respiratory, gastrointestinal, renal and genital tracts, subarachnoid space and adrenals, causing appropriate symptoms and occasional fatal results. [10] Thrombocytopenia occurs in severe cases with renal failure. [10]

Complications

Serous meningitis is the most common form of neurological complication, in the second phase. [10] Occasionally encephalitis may occur, in which the patients may lose memory and hallucinate, be delirious, confused, disorientated, or semicomatose, or develop extra pyramidal symptoms. [10]

Renal failure is an important cause of death in patients with leptospirosis.[12] Cardiac dysfunction may also lead to hypo perfusion in severe leptospirosis.[12] Focal haemorrhagic myocarditis, pericarditis and cardiac arrhythmias have also been well documented. [12] Acute hemorrhagic lobar pneumonia and massive haemoptysis have been observed in fatal cases. [12] Myocarditis is a cause of death in those whom renal failure can be managed successfully. [10] Inflammation of the uveal tract, presenting as iritis, iridocyclitis or occasionally chorioretinitis are important but less frequent complications. [10] Acute infection in pregnancy has been reported to cause abortion and foetal death.[2] Rare complications include cerebrovascular accidents, rhabdomyolysis, thrombotic thrombocytopenic purpura, acute acalculous cholecystitis, erythema nodosum, aortic stenosis, Kawasaki syndrome, reactive arthritis, epididymitis, nerve palsy, male hypogonadism and Guillain Barre syndrome.[2]

Epidemiology

Leptospirosis is recognized as the most common zoonotic infection in the world.[1] Leptospirosis has been known as Weil`s disease, mud fever, trench fever, rice field fever, cane cutters fever, swamp fever, flood fever, autumnal fever, seven days fever of Japan, Swine herds disease ,pea picker's fever, spirochaetal jaundice, canicola fever etc. etc.[19]

The source of infection in humans is usually either direct or indirect contact with the urine of an infected animal. [2] The incidence is significantly higher in warm climate countries than in temperate regions, this is due mainly to longer survival of leptospires in the environment in warm, humid conditions. [2] The disease is seasonal with peak incidence occurring in summer or fall in temperate regions, where temperature is the limiting factor in survival of leptospires and during rainy seasons in warm climate regions, where rapid desiccation would otherwise prevent survival .[2] The core determinants of transmission of Leptospiral infection are the presence of carrier animals, suitability of the environment for the survival of leptospires and interaction between man, animals and environment.[24] South east Asia is an endemic area for leptospirosis and infection in humans has been reported throughout the region.[25] The spirochete requires a warm, moist, climate of 25^0 C and water and soil PH level of 7.0-8.0 for optimal survival outside the host.[26]

Human infections may be acquired through occupational, recreational or avocational exposures. [2] Direct contact with infected animals accounts for most infections in farmers, veterinarians, abattoir workers, meat inspectors, rodent control workers and other occupations which require contact with animals. [2] Indirect contact is important for sewer workers, miners, soldiers, septic tank cleaners, fish farmers, game keepers, canal workers, rice field workers, taro farmers, banana farmers, and sugar cane cutters. [2] The major occupational risk today is among farm workers.[27] There is a significant risk associated with recreational exposures occurring in water sports including swimming, canoeing, white water rafting, fresh water fishing and other sports. [2]

Animals including humans can be divided into maintenance hosts and accidental hosts. [2] Maintenance population is defined as "a population of a species of animal which acts as a continuous reservoir of a serotype in a particular ecosystem". [28] Accidental hosts are characterized by low susceptibility to infection, if the infection is established the pathogenic effect may be severe, with a short renal phase and inefficient intraspecies transmission. [28] A human case of Weil`s disease demonstrates most of the features of an accidental host. [28]

The disease is maintained in nature by chronic infection of the renal tubules of maintenance hosts. [2] Other animals (such as humans) may become infected by indirect contact with the maintenance host.[2] Humans are dead end hosts and do not provide an infection reservoir.[29] The natural reservoir for pathogenic leptospires is wild animals ,particularly the rodent family.[27]Rodents are prolific shedders of leptospires voiding them in urine continuously.[1]

The most important sources for infection of humans are the various forms of rodents with which humans live in all parts of the world in domestic, agricultural or occupational association, and the domesticated large animals used for work or for food. [10] Rodents closely associated with human habitation, such as the black and brown rats (*Rattus rattus* and *Rattus norvegicus*) and the common domestic mouse (*Mus*

musculus) can act as sources of leptospirosis for humans, dogs and farm animals. [10] Pets and laboratory animals are also potential carriers and excretors. [10]

Severe leptospirosis seems to be the tip of the iceberg of leptospiral infection. [30] The incidence of leptospirosis is remarkably under estimated in estimates from endemic regions. [8] According to the recently available reports, incidence ranges from approximately 0.1-1 per 100,000 per year in temperate climates to 10-100 per 100,000 per year in humid tropic climates.[15] Leptospirosis is estimated to affect tens of millions of humans annually with case fatality rates ranging from 5 to 25%. [8] Leptospirosis is under reported due to lack of clinical suspicion and barriers to diagnostic capacity.[8] Occupational exposure probably accounts for 30-50% of human cases.[8] Men suffer more frequently from leptospirosis than women because of greater occupational exposure to infected animals and contaminated environment. [19] Leptospiral infections occur more frequently in persons 20-30 years of age. [19] The rural epidemiologic pattern, which occurs often in agrarian communities in the developing world, is usually associated with cultivation cycles which in turn depend on meteorological phenomena such as monsoons. [24] The urban epidemiological form is often seen in overcrowded cities and towns of developing countries where the environmental sanitation and the personal hygiene of the people are poor. [24] Two other epidemiological forms are the recreational leptospirosis and leptospirosis associated with natural disasters.[24] The disease typically occurs as an epidemic lasting a few weeks during the monsoon season. [31] Extensive flooding and seasonal rainfall are significant risk factors for exposure to water contaminated with leptospires. [26]

Certain serovars of *Leptospira* are of greater epidemiological significance. [19] These include L.pomona, L.grippotyphosa, L.hebdomadis, L.canicola, L.icterohaemorrhagiae etc. [19] The majority of infections in humans and farm animals are caused by these serovars. [19]

Leptospirosis in animals

Leptospirosis is widespread in domestic animals. [4] Bovine leptospirosis is common throughout the world. [4] Infections are often inapparent. [4] Leptospires isolated from cattle include those belonging to serogroups Australis, Autumnalis, Ballum, Bataviae, Canicola, Grippotyphosa,Hebdomadis,Javanica,Sejroe,Mini,Icterohaemorrhagiae,Pomona,Tarassovi, Panama and Pyrogenes. [4] Hardjo is the commonest serotype in cattle throughout the world.[4] Rats are generally maintenance hosts for serovars of the serogroups Icterohaemorrhagiae and Ballum, dairy cattle may harbour serovars hardjo, pomona and grippotyphosa, pigs may harbour pomona, tarrasovi or bratislava, sheep may harbor hardjo and pomona & dogs may harbor canicola. [2] Dogs are a significant reservoir for human infection in many tropical countries and may be an important source of outbreaks. [2]

Treatment

There is still some dispute about the value of antimicrobial therapy for leptospirosis. [32] It is generally believed that antimicrobial agents are effective only if given as early as possible.[32] Antimicrobial treatment benefits leptospirosis patients, whether children or adults, decreasing the duration of the illness, reducing the accompanying thrombocytopenia and limiting the severity of the renal failure.[33] In the mild forms of leptospirosis management is symptomatic, as indicated by the nature and severity of the manifestations.[10] Treatment should be initiated as early as possible. [18] The severe form of leptospirosis requires intensive-care support and urgent symptomatic treatment. [10] In severe cases of renal failure, intensive renal management, including peritoneal dialysis, may be necessary. [10] Dialysis support should continue until natural renal function recovers, which it usually will unless the patient succumbs to other lesions.[10] Those with Weil 's syndrome may need transfusions of whole blood and\ or platelets. [18]

Penicillin G Sodium is the generally recommended treatment for leptospirosis.[34] For severe cases of leptospirosis, intravenous administration of Penicillin G, Amoxicillin, Ampicillin or Erythromycin is recommended.[18]

Prevention and Control

Prevention of leptospirosis in all situations is not possible, because it is widespread in so many animals and places all over the world. [13] The best that can be done is to limit the effects of leptospirosis on humans and the animals they depend on. [13] This involves identification of sources, containing them and eliminating them or their effects. [13] The best way to avoid leptospirosis is to keep away from animals and areas that may be contaminated by their urine. [13] People whose occupation, travel or hobbies involve risks should know of the disease and how to avoid it.[13] Occupational hygiene is relevant for prevention of human leptospirosis, wherever the disease is known to occur predominantly in certain occupational groups.[10] People should be aware of the dangers and be dissuaded from swimming in rivers or pools suspected to be contaminated. [13] Rat control in and around food storage and preparation areas, crop storage areas, stables, milking sheds, intensive animal production installations and dwellings is difficult but will remove a major source of leptospirosis for humans and domesticated animals. [13] All the people involved in high-risk activities should wear protective clothing and need to adopt a reasonable standard of hygiene. [13] Long term control strategies of the disease include adoption of hygienic measures, rodent control and vaccinations.[35]

Vaccines for animals

They are made by combining suspensions of different serovars, chosen according to local needs.[10] Dogs are immunized to protect them and human companions. [13] Effective vaccines containing suspensions of killed *L.borgpetersenii* serovar *hardjo* and *L. interrogans* serovar *pomona* are widely available commercially. [13] The use of locally prevalent strains is recommended.[13]

Vaccines for humans

Vaccination of humans is justified where they cannot be separated from the animal sources of leptospirosis, or where the animals cannot be immunized successfully. [10] Examples are where people live and work in proximity to rodents in wet, tropical conditions, in wet rice planting and harvesting, in military operations, or working in sewers.[10] Vaccines composed of killed cultures of leptospires protect people against leptospirosis. [13] These vaccines may cause side effects, ranging from local soreness to fever and incapacity for a few days. [13] Two doses are given subcutaneously, 3 to 4 weeks apart, followed by annual boosters. [13] Multivalent combinations effective against several serovars are compounded, as required by local needs. [13] They are made available to selected high-risk groups, wherever the side effects are preferable to severe leptospirosis.[13] In France, a monovalent vaccine, containing only serovar icterohaemorrhagiae is licensed for human use. [2] A vaccine containing serovars Canicola, icterohaemorrhagiae and Pomona has been developed recently in Cuba.[2] Vaccines for use in humans were used in Vietnam, China and Japan. [10] The Chinese and Japanese vaccines are not available or licensed for use outside those countries at present. [10] The vaccines are given annually in two doses, and boosters are required annually. [10] In addition to their use to protect occupationally or environmentally exposed people, they are used to immunize large numbers of people exposed to natural emergencies, especially floods, and laboratory workers prophylactically or after exposure to risk in laboratory accidents.[10] The use of a DNA construct encoding leptospiral proteins is a promising new approach for vaccination against leptospirosis. [35] Constraints in leptospirosis vaccine development and use- since the vaccines largely confer serovar specific immunity, continuous epidemiological monitoring of the prevalence of *Leptospira* serovars in a zone or region is desired to select the correct serovars for incorporating into the vaccine.[35]

DISCUSSION

Leptospirosis is an acute febrile disease. It is recognized as being emerging and re-emerging infection. The disease ranges from an inapparent infection to a fatal fulminant disease. The actual number of leptospirosis cases could be several folds the number of patients clinically diagnosed as having leptospirosis as it is under reported due to lack of clinical suspicion and barriers to diagnostic capacity. Early suspicion and confirmation is crucial to reduce the morbidity and the case fatality rate. Vaccines are available only for animals. No approved vaccine is available for humans.

CONCLUSION

Leptospirosis is frequently underdiagnosed because of the nonspecific symptoms in the initial stage of the disease. Early diagnosis is essential as untreated the illness can progress rapidly and mortality rates are high in severe cases. Hence leptospirosis should be suspected in all fever cases, especially in the males of the occupationally active group with history of animal contact.

ACKNOWLEDGEMENTS

Authors acknowledge the immense help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

Source of funding

None

Conflict of interest

None

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