Leptospirosis : A Disease of Public Health Importance

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ABSTRACT: Leptospirosis has been recognized as an important emerging global public health problem because of its epidemic proportions and increasing incidence in both developing and developed countries. The disease is endemic in Kerala, Tamil Nadu, Gujarat, Andaman, Karnataka, Maharashtra. It has also been reported from Andhra Pradesh, Orissa, West Bengal, Uttar Pradesh, Delhi and Puducherry. It is an infectious disease caused by pathogenic bacteria called leptospires which has over 20 serogroups and more than 200 serovars. Man is infected either directly through contact with an infected animal or indirectly by water or soil contaminated with the urine of an infected animal. Human-to-human transmission occurs only very rarely. Leptospirosis can manifest in many ways. Clinical diagnosis is difficult because of the varied and non-specific presentation. In this situation, ELISA tests is particularly useful in making early diagnosis. Other serological tests available are indirect flouroscent antibody test (IFAT), indirect hemagglutination tests (IHAT), latex agglutination tests (LAT), micro capsule agglutination tests (MicAT), counter immune-electrophoresis (CIE) and CFT etc. Antibiotic treatment is effective within 7 to 10 days of infection and it should be given immediately on diagnosis or suspicion. In severe cases, admission to a hospital is necessary. Identification of the potential risk factors would help understand the transmission dynamics of the disease and formulate public health interventions.

it was common among coal miners (Faine, 1982) and soon after in Germany (*Spirochaeta icterogenes*) by Uhlenhuth and Formme (Uhlenhuth and Formme, 1916). Inada and Ido (1915) successfully demonstrated the transmission of infection to guinea pigs from the blood of the infected animals which produced the responsible organism (Vijayachari et al., 2008). Noguchi proposed the name ‘Leptospira’ (thin spirals) in 1918, following detailed microscopical and cultural observations (Noguchi, 1928).

 Soon after the breakthrough that Weil’s disease was caused by leptospires several other disease entities were being recognized to have leptospiral aetiology. These include ‘nanukayami’ or the Japanese seven-day fever, ‘akiyami’ the harvest fever and more recently Andaman fever or Andaman haemorrhagic fever (AHF). Much of the basic current knowledge about leptospires and leptospirosis was understood within a decade following the discovery of leptospires and several types of them were recognized during this period (Vijayachari et al., 2008 and Kmety and Dicken, 1988 and 1993).

### Outbreaks in India:

India is a developing country, about 72-74 per cent of the people live in rural areas. The main source of income is agriculture; several millions of them are either marginal farmers or work on hired labour and struggle for bare necessities of life. It is said that nearly 11 per cent of the total wild animal population is in India. It is usually observed that, animals are house under the same roof as human being. Therefore, all possibilities exist for the various diseases to be transmitted by the animals to man (Ghulani, 2008). Since 1980’s the disease has been reported from various states during monsoon months in mini epidemic proportions. The disease is endemic in Kerala, Tamil Nadu, Gujarat, Andamans, Karnataka, Maharashtra. It has also been reported from Andhra Pradesh, Orissa, West Bengal, Uttar Pradesh, Delhi and Puducherry (WHO, 2006 and Kamath and Joshi, 2003). Leptospirosis has been under reported and under diagnosed from India due to a lack of awareness of the disease and lack of appropriate laboratory diagnostic facilities in most parts of the country (Shivakumar, 2008).

In 1931, an extensive survey of the disease outbreak in the Andaman Islands was made and researcher’s isolated *L. andamans* and *L. grippotyphosa* (Taylor and Goyle, 1931). Several others have confirmed the prevalence of leptospirosis in India by isolating leptospires from human material (Das Gupta and Chopra, 1937; Das Gupta, 1938 and Lahiri, 1941). Andaman and Nicobar Islands are endemic for leptospirosis since early part of the 20th century (Shivakumar, 2008). In 2005, 58 cases of confirmed leptospirosis were admitted and 14 patients died [Case Fatality Rate (CFR) - 24.1%] (WHO, 2006). The disease is endemic in south Gujarat since 1994 (WHO, 2006; Patel et al., 2006 and Clerke et al., 2002). In the year 2005, 392 cases and 81 deaths due to leptospirosis were reported from various districts of south Gujarat. There were 310 males and 82 females, mostly in the age group of 26 - 45 year (Clerke et al., 2002). Leptospirosis has been reported regularly in Maharashtra since 1998 (WHO, 2006; Karande et al., 2003 and 2002). 2355 cases and 167 deaths were reported in 2005, mainly due to large outbreak during the post-monsoon floods. The number of districts in Maharashtra reporting leptospirosis has expanded from two in 1998 to ten districts in 2005. Kolenchery is in the midlands of Kerala. In this area leptospirosis was rarely diagnosed before 1987. Since then a yearly increase in incidence was observed. In a study of 976 cases of leptospirosis confirmed by culture and / or serological tests, a mortality rate of 5.32 per cent was observed. Autumnalis, Australis and Icterohaemorrhagiae were the common serogroups identified (Kuriakose et al., 1997). It has been reported from Chennai since 1980’s (Ratnam et al., 1983). There has been a dramatic increase in the number of leptospirosis cases and during 2006, 2765 cases were reported. Leptospirosis outbreaks have been reported from 15 districts of Karnataka. During the year 2004, 152 cases and 11 deaths were reported and during 2005, 224 cases and 19 deaths were reported (WHO, 2006). After the cyclone during the October-November 1999, leptospirosis was confirmed in Orissa (Sehgal et al., 2000). 143 people were evaluated by the Orissa Multi-disease Surveillance System (OMDSS) during the period June - July 2002 (Jena et al., 2004). Evaluation of acute febrile patients in Uttar Pradesh revealed that 7 per cent had leptospirosis (25/346), 17 of the 25 patients had jaundice (Manocha et al., 2004).

Recent outbreaks were reported in Gujrat (750 cases, 13 fatal) in 2011 and 61 cases in 2013. In 2011, 256 cases were also reported in Kerala in which 43 were fatal and 120 cases were found in Andhra Pradesh during the year 2013. An outbreak (200 cases) of dengue,
chikungunia and leptospirosis was reported from Tamil Nadu in 2014 (Berger, 2015) and at present in the 1st week of July 2015, leptospirosis has struck in a big way and claimed 12 lives and affected 21 others in various municipal hospitals of Mumbai. The Brihanmumbai Municipal Corporation (BMC) officials pegged this 57 per cent death rate to the delay among the patients in seeking treatment. Most of the victims, including a 12-year-old girl, lived between Goregaon and Dahisar and were under 30 years of age. It was the largest monsoon killer in 2005 - the year of the 26/7 deluge - as well as in 2007 when thousands were affected and 66 and 79 died, respectively (Times of India, 2015).

What is leptospirosis?

Leptospirosis is an infectious disease caused by pathogenic bacteria called leptospires (WHO, 2003), which has over 20 serogroups and more than 200 serovars. Rodents, domestic and wild animals form the reservoir of infection where domestic animals such as cattle, dogs and pigs 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. Leptospires are excreted in the urine of the animals and they affect human when they come into contact with urine of infected animals, directly or indirectly, when they are exposed to an environment contaminated by the urine of the infected animals such as soil and surface water following monsoon rains (Fig. 1). Therefore, this illness commonly occurs during the monsoon months. The infection is probably transmitted when they wade through stagnant rainwater contaminated by infected urine of animals. These organisms can survive for 6 hours in dry soil and for 6 months in flooded condition. They enter the host through the abrasions of the skin of the feet or intact mucous membranes of eye, throat and gut (Faine, 1982). It is, therefore, a zoonosis. Human-to-human transmission occurs only very rarely (WHO, 2003). The alternative names of Leptospirosis are weil disease, 7-day fever, harvest fever, ictehemorrhagic fever, swine herd’s disease, rice field fever, cane-cutter fever, swamp fever, mud fever, heamorrhagic jaundice, stuttgart disease, canicola fever and, rat fever and farmer’s disease in India (Goldman and Ausiello, 2007). It has been historically known as “black jaundice” (Clapham, 2004).

Persons of all ages and races are susceptible. Adult men however, are more frequently infected because they tend to work in high-risk jobs. The number of cases in a region often fluctuates from year to year due to various factors such as rainfall, flooding and animal infections. Leptospirosis tends to occur as individual/small cluster of cases or large outbreaks/epidemics. In India, urban leptospirosis has been reported from Chennai and Mumbai while rural leptospirosis has been reported from Gujarat, Kerala and Andamans. Non-reporting of leptospirosis from other states of India does not mean that it is absent in those parts (Shivakumar, 2008).

It is estimated that seven to ten million people are infected by leptospirosis in a year. The number of deaths causes are not clear (NHS, 2012). Leptospirosis occurs worldwide but is most common in tropical and subtropical areas with high rainfall wherever humans come into contact with the urine of infected animals or a urine-polluted environment (WHO, 2003). Therefore, it may occur in anywhere.

Presently two different classification systems- one based on phenotypic characters and other on the genetic homology are being used. In the phenotypic classification there are two species namely, the L. Interrogans (pathogenic) and the L. biflexa (non-pathogenic). Both the species have several serovars and serovar is the basic taxon, which is defined in the basis of surface antigenic makeup. Closely related serovars are arranged in serogroups. Based on genetic homology in DNA hybridization experiments, 15 genomic species (L. interrogans, L. kirschneri, L. borgpetersenii, L. santarosai, L. noguchii, L.welii, L.inadai, L.bifl exa, L.meyeri, L.wolbachii, Genomo species 1, Genomo
species 3, Genomo species 4 and Genomo species 5) have been described in the genus Leptopsira whereas Leptonema and Turneria have one species each (L. illini and T. parva) (Zaki and Sheih, 1995 and Vijayachari et al., 2008).

### Clinical features:

Leptospirosis can manifest in many ways (Terepstra et al., 2003). The various syndromes of presentation are as follows:

- Acute febrile illness
- Weil’s syndrome characterized by jaundice, renal failure and Myocarditis with cardiac arrhythmias
- Pulmonary Haemorrhage with respiratory failure
- Meningitis / Meningo encephalitis.

Some other common symptoms of leptospirosis include fever, chills, headache, muscle aches, vomiting, diarrhoea, abdominal pain, skin rash, red eyes.

Clinical diagnosis is difficult because of the varied and non-specific presentation. Confusion with other diseases, e.g. dengue and other haemorrhagic fevers, is particularly common in the tropics. Presentations may overlap as infection progresses (WHO, 2003).

The incubation period is 7-14 days, but ranges from 2-21 days. The incidence rate ranges from 0.1-1/100,000 per year in temperate climates to 10-100/100,000 in tropical countries. During outbreak the incidence may reach over 100/100,000. Hospital based data on clinical manifestations confirmed by laboratory tests (Rapid tests / MAT) are usually needed to obtain the incidence rates. Mild cases may not be admitted to hospitals and hence, these data may result in a bias towards severity in assessing the public health importance of leptospirosis (Terepstra et al., 2003). The prevalence rates are obtained from asymptomatic individuals of selected high risk groups. Sero surveillance provides data on infection rather than as a disease.

MAT (Microscopic Agglutination Test) is required for sero surveys. It is the gold standard for diagnosis but it is complicated and less sensitive compared to newer tests like ELISA IgM and slide agglutination tests (SAT). ELISA IgM and slide agglutination tests are simple, sensitive tests and can be used to diagnose current leptospirosis. ELISA tests is particularly useful in making early diagnosis, as it is positive as early as 2 days into illness, a time when the clinical manifestation may be nonspecific. It was found to be 100 per cent sensitive and 93 per cent specific in one study. Dot-ELISA and Dip-stick methods for detecting IgM antibodies are newer screening methods. Other serological tests available are indirect flouroscent antibody test (IFAT), indirect hemagglutination tests (IHAT), latex agglutination tests (LAT), micro capsule agglutination tests (MicAT), counter immune-electrophoresis (CIE) and CFT etc (Dutta et al., 2012).

### Pathogenic mechanisms:

Leptospirosis is a primary bactraemic infection. Localisation of leptospires at the site of entry does not occur in natural conditions. Leptospires are not pyogenic bacteria; they do not cause inflammatory reactions except through secondary tissue damage. The central lesion, characteristic of all forms of leptospirosis, is damage to the walls of small blood vessels, leading to leakage and extravasation of cells, including haemorrhages. Other lesions follow as secondary effects (Faine et al., 1999). Paradoxically, the most obvious adhesion of leptospires to cell surfaces, in the renal tubules, does not appear to damage the cells, nor lead to inflammation around the affected tubules in the absence of repair or scarring from the acute infection, in most animals (Andersson, 2009).

The primary lesion in all forms of leptospirosis in all animals, including humans, is damage to the membranes of the endothelial cells of the small blood vessels, caused by leptospiral toxin. The immediate effect is to loosen the junction between cells, allowing fluid and leptospires to migrate into extravascular spaces, followed by
erythrocytes wherever the damage is severe and prolonged. The secondary effects of ischemic changes, anoxia and increased pressure in the tissues reinforce damage resulting in cellular functional disintegration and death (Faine et al., 1999).

**Treatment:**

Antibiotic treatment is effective within 7 to 10 days of infection and it should be given immediately on diagnosis or suspicion. The antibiotic of choice is benzyl penicillin by injection in doses of 5 million units per day for five days. Patients who are hypersensitive to penicillin may be given erythromycin 250 mg 4 times daily for 5 days. Doxycycline 100 mg twice daily for 10 days is also recommended. Tetracyclines are also effective but contraindicated in patients with renal insufficiency, in children and in pregnant women (Vijayachari et al., 2008). In more severe cases cefotaxime or ceftriaxone should be preferred. Injection of Hydrocortisone 100mg every 8 hourly is also given in severe cases.

Doxycycline has been used as a chemo prophylactic agent for short time exposure, but it cannot be recommended for routine continuous use or for a long-term occupational exposure (Sehgal et al., 2000). In severe cases, admission to a hospital is necessary. Aggressive supportive care with strict attention to fluid and electrolyte balance is essential (WHO, 2003). Therefore, glucose and salt solution infusions may be administered. Elevations of serum potassium are common and if the potassium level gets too high special measures must be taken. Serum phosphorus levels may likewise increase to unacceptable levels due to renal failure. Peritoneal or haemodialysis is indicated in renal failure. Excellent supportive care and dialysis have reduced the mortality of this illness in recent years (WHO, 2003).

**Prevention and control:**

Prevention of leptospirosis is essentially by identifying the source and interrupting the transmission (Faine, 1994). Control can be achieved by controlling the reservoir or reducing infection in animal reservoir populations such as dogs or livestock. Control of wild animals may be difficult. Preventive measures must be based on a knowledge of the groups at particular risk of infection and the local epidemiological factors (WHO, 2003). Human vaccines are available only in a few countries, such as Cuba and China (McBride et al., 2005). Vaccines have been reported to give some degree of protection and this is particularly important in areas where more serious forms of leptospirosis occur and where access to medical services is limited or delay in receiving treatment is likely. However, protection is of relatively short duration and boosting at regular intervals is necessary to maintain protective titre of antibodies. Vaccines may also produce side effects, such as pain at the injection site, and fever (WHO, 2003).

According to Leptospirosis Burden Epidemiology Reference Group (LERG), leptospirosis is a preventable disease when risk factors are appropriately identified and managed and interventions are targeted towards risks at individual and community levels. If all relevant sectors collaborate and coordinate prevention and control measures, it can be controlled. For this, awareness of the significance of the problem and a willingness to act are very necessary.

![Different control strategies for leptospirosis (Vijayachari et al., 2008)](image_url)

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