Leptospirosis in Humans and Dogs

Carlos Victor Hernández Ramírez*
Health Services of Sinaloa, Department of Prevention and Control of Vectors and Zoonosis, Mexico

Submission: January 15, 2019; Published: January 24, 2019
*Corresponding author: Carlos Victor Hernández Ramírez, Health Services of Sinaloa, Department of Prevention and Control of Vectors and Zoonosis, Calle Mariano Escobedo 1 026. Colonia Las Vegas Culiacán, Sinaloa Mexico.

Perspective

Leptospirosis is the most widespread zoonotic disease in the world, with great economic and health importance, is an infectious disease caused by serovars of Leptospira interrogans. It is a worldwide public health problem that affects both industrialized and developing countries; the number of cases is difficult to establish, but approximately 1.03 million cases are estimated to occur each year throughout the world, with 59,900 deaths [1]. The World Health Organization (WHO) and the Pan American Health Organization (PAHO) classify Leptospirosis icterohaemorrhagiae with the key A 27.0 [2], is caused by a spirochete belonging to the pathogenic strains of the genus Leptospira, which affects wild animals, domestic animals and humans [3,4]. This genus is traditionally classified based on the phenotypic properties, serological reactions and pathogenesis of each strain. Saprophytic species of Leptospira are grouped mainly into L. biflexa, (after L. biflexa sensu lato) and L. interrogans (after L. interrogans sensu lato); these include most of the pathogens [5]. Currently, the classification of the genus Leptospira is based on DNA homology and is divided into 17 species [6,7].

Leptospires are strictly aerobic microorganisms; morphologically they are spirchoetes of about 0.1μ wide and 6-15μ long, with flexion, translation, and propulsion movements, as well as active undulation. They are Gram-negative and divide by binary fission, this microorganism is sensitive to drying, heat, excessive cold and pH variations; they do not tolerate the acid medium due to the loss of their motility in approximately 15 min, the optimum pH for their multiplication is from 7.2 to 7.4, they do not survive in salt water, but they can remain up to 180 days in fresh water; three weeks in stagnant waters and up to about a year in viscous solutions, such as sludge with low content of organic matter [8], moist soil they survive for a long time, while in dry soil the survival is short [9]. The natural regions of leptospirosis are generally found in humid and rain areas in conditions with high temperatures and humidity with a higher proportion of surface fresh water such as lakes, rivers, dams, channel systems among others [10-12].

In the tropical climatic zone, environmental conditions are more favorable for the survival of leptospires and the highest morbidity is observed, extreme weather phenomena such as cyclones and floods can lead to an increase in the incidence of the disease, as well as the magnitude of leptospirosis outbreaks [13,14]. Socio-economic factors such as migration give rise to the transmission of acquired infections in tropical countries, people with lower income or poor communities, whose consequence is less hygienic conditions [15]. Infection is typically transmitted through direct contact of oral or nasal mucosa, with contaminated urine or water, and dogs are at risk of infection from drinking contaminated water [16,17]. Dogs play an important role as potential indicators of areas with high endemicity for leptospirosis. Thus, recognizing and preventing canine leptospirosis has implications for human health as well as dogs [18,19].

Leptospires infect an organism penetrating through mucous membranes, skin lacerations or skin softened by moisture, and intake of contaminated food and water. They then migrate through the blood, tending to locate and grow in parenchymal organs such as liver, kidney, spleen and, occasionally, the meninges. They remain in sites such as renal tubules, ocular humors and the uterus, where antibody activity is minimal; they cause vascular damage to the endothelium, producing bleeding [20-22]. The serovars icterohaemorrhagiae and pomona produce hemolysins, which are responsible for hemoglobinuria [23]. In the case of the serovar icterohaemorrhagiae, it causes severe jaundice in dogs, very similar to the infection caused in humans. It presents more frequently in dogs younger than two years old; there is a transient increase in body temperature that usually goes unnoticed, sudden or progressive onset of jaundice, going from a pale yellow to an orange yellow color in skin and mucous membranes, yellowish brownish
urine, weakness, chills, depression, anorexia, emesis, polydipsia, emaciation, dehydration, petechial bleeding, ecchymosis of conjunctiva and oral cavity, and halitosis [24-26].

In humans the clinical manifestations and severity of leptospirosis vary from a flu-like illness to severe renal and hepatic failure, myocarditis, hemorrhage and death, depending on the concentration of the inoculum, the virulence of the infecting serovar, the susceptibility of the host, and the affected organ or systems [27]. Two cases of the disease have been recorded in patients transplanted with kidneys infected with Leptospira, one of them fatal [28,29]. It is characterized by two phases, the first bacillary or leptospirémic, has an abrupt onset of duration seven to ten days, the signs and symptoms are not pathognomonic, they can easily be confused with other infectious processes of bacterial type or viral, such as dengue, zika, chincungunya, malaria, brucellosis, rickettsiosis. The second phase presents the characteristics of the immune phase and correlates with the appearance of circulating antibodies of the IgM class and the invasion of vital organs [30-32]. Two clinical types are generally distinguished: icteric and anicteric. The icteric or hepatonephritic type (Weil’s disease) is found in approximately 10% of the cases, while numerous infections occur in anicteric form [31,33].

The invasive power of leptospires is related to their mobility and their chemical and antigenic structure; they cause cellular damage, not only by mechanical strain, but by the production of cytotoxic substances, which damage the capillary endothelium. This cytotoxic protein has been found in the serovars Pomona and Copenhageni [34,35]. The diagnosis of leptospirosis is performed by various methods, including direct immunofluorescence, silver staining of fixed tissues, polymerase chain reaction (PCR), culture isolation, evidence of serum antibodies (ELISA), fast plate agglutination and latex agglutination, as well as rapid card tests (Leptospira dipstick); however, plate microagglutination (MAT) is regarded as the gold standard. The reactions determine the presence of agglutinating antibodies against the tested serovars [36,37]. Animals may remain serologically positive for the disease for years. In dogs, leptospirosis is mainly caused by L. canicola or L. icterohaemorragiae; these serotypes are internationally considered as the most important. The main source of infection for animals, especially dogs, is the urine of asymptomatic carrier animals (dog to dog), as well as vectors, rodents being a natural reservoir [38,39]. Leptospirosis continues being serious public health problem, for humans and animals’ diagnostic and control are complicated by the great adaptability and pathogenic characteristics, treatment not complicated by diagnosis is early, new researches provide more information to propose better public health policies for control and diagnosis of disease.

References

1. Costa F, Hagan JR, Cacagnio J, Kane M, Torgerson P, et al. (2015) Global Morbidity and Mortality of Leptospirosis: A Systematic Review. PLoS Negl Trop Dis 9(9): e0003898.

2. Organización Mundial de la Salud (1992) Organización Panamericana de la Salud. Clasificación Estadística Internacional de Enfermedades y Problemas Relacionados con la Salud CIE-10.

3. Organización Mundial de la Salud (OMS) Leptospirosis humana: guía para el diagnóstico, vigilancia y control / Organización Mundial de la Salud (2008) Traducción del Centro Panamericano de Fiebre Aftosa. Rio de Janeiro: Centro Panamericano de Fiebre Aftosa VP/GPS/OMS.

4. Dírcio MS, González FE, Verdalhe GM, Soler HE, Rivas SB, et al. (2012) Leptospirosis prevalence in patients with initial diagnosis of dengue. J Trop Med pp. 519701.

5. Céspedes M (2005) Leptospirosis: enfermedad zoonótica reemergente. Rev Peru Med Exp Salud Pública 22: 4.

6. Faine S, Adler B, Bolin C, Perolat P (1999) Leptospira and leptospirosis. (2nd edn), Melbourne, Australia.

7. Brenner DJ, Kaufmann AF, Sulzer KR, Steigerwalt AG, Rogers PC, et al. (1999) Further determination of DNA relatedness between serogroups and serovars in the family Leptospiraceae with a proposal for Leptospira alexanderi sp. nov. and four new leptospira genospecies. Int J Syst Bacteriol 49(2): 839-858.

8. Trueba G, Zapata S, Madrid K, Cukken O, Haake D (2004) Cell aggregation a mechanism of pathogenic Leptospira to survive in fresh water. Int Microbiol 7: 35-40.

9. Levet P (2001) Leptospirosis. Clin Microbiol Rev 14(2): 296-326.

10. Bielskansky A, Surujballi O (1996) Association of Leptospira burgdorferi with a pathogenic isolation and serovars of L. icterohaemorragiae. Int J Syst Bacteriol 49(2): 839-858.

11. Torres, CM, Hernández BS, Agudelo FP, Arroyave SE, Zavala CJ, et al. (2016) Revisión actual de la epidemiología de la leptospirosis. Rev Med Inst Mex Seguro Soc 54(5): 620-625.

12. Luna, AMA, Moles CLP, Gavaldón RD, Nava VC, Salazar GF (2005) Estudio retrospectivo de seroprevalencia de leptospirosis bovina en México, considerando las regiones ecológicas. Rev Cubana Med Tropical 51: 1.

13. Vijayachari P, Sugunan AP, Shriram AN (2008) Leptospirosis: an emerging global public health problem. J Biosci 33: 557-569.

14. Lau CL, Lee D, Smythe LD, Scott B, Cnaig SB, et al. (2010) Climate change, flooding, urbanisation and leptospirosis: fuelling the fire? Royal Society of Tropical Medicine and Hygiene, 104(10): 631-638.

15. Vinez, JM, Glass GE, Flecne CE, Mueller P, Kaslow DC (1996) Sporadic urban leptospirosis. Ann Intern Med 125(10): 794-798.

16. Nelson RW, Couto CG (2003) Small Animal Medicine, (3rd edn), Mosby, St. Louis, USA.

17. Heymann D (2008) Control of Communicable Diseases Manual, (19th edn), American Public Health Association, Washington DC, USA.

18. White AM, Zambarna-Torrealo C, Allen T, Rostal MK, Wright AK, et al. (2017) Hotspots of canine leptospirosis in the United States of America. Veterinary Journal 222: 29-33.

19. Hernández Ramírez CV, Gaxiolla Camacho SM, Osuna Ramírez I, Enríquez Verdugo I, Castro del Campo N, et al. (2017) Prevalence and risk factors associated with serovars of Leptospira in dogs from Cuilacan, Sinaloa. Veterinaria México OA 4: 2.

20. Ortega PA, Colín FRF, Gutiérrez BE, Jiménez CM (2008) Frequency and type of renal lesions in dogs naturally infected with Leptospira species. Ann NY Acad Sci 1149: 270-274.

21. Greenlee JJ, Alt DP, Bolin CA, Zuerren RL, Andreassen CB (2005) Experimental canine leptospirosis caused by Leptospira interrogans serovars pomona and broadi. Am J Vet Res 66(10): 1816-1822.

22. Van de Maele I, Olaus A, Haesebruck F, Daminet S (2008) Leptospirosis in dogs; a review with emphasis on clinical aspects. Vet Rec 163(14): 409-413.
23. Goldstein RE, Lin RC, Langston CE, Scrivani PV, Erb HN, et al. (2006) Influence of infecting serogroup on clinical features of leptospirosis in dogs. J Vet Intern Med 20(3): 489-494.
24. Low DG (1981) Leptospirosis canina en: Terapéutica veterinaria. (3rd edn), DF (MX): Continental, México.
25. Solano-Chinchilla A, Boza-Cordero R, Saenz-Bolaños E (1996) Leptospirosis en humanos. Rev Cost de Ciencias Médicas. 17(2): 41-60.
26. Ellis WA (1994) Leptospirosis as a cause of reproductive failure. Vet Clin North Am Food Anim Pract 10(3): 463-478.
27. Min Ja Kim (2013) Leptospirosis in the Republic of Korea: Historical Perspectives, Current Status and Future Challenges. Infect Chemother 45(2): 137-144.
28. Khosravi M, Bastani B (2007) Acute renal failure due to leptospirosis in a renal transplant patient: a brief review of the literature. Transplantation proceedings 39(4): 1263-1266.
29. Gerasyuchuk L, Swami A, Carpenter CE, Samarapungavan D, Batke M, et al. (2009) Case of fulminant leptospirosis in a renal transplant patient. Transpl Infec Dis an Official Journal of the Transplantation Society, 11(5): 454-457.
30. Adler B, de la Peña A (2010) Leptospira and leptospirosis. Veterinary Microbiology 140: 287-296.
31. Barrido EM, Alexanderson E, Halabe CA (1989) Enfermedad de Weil, cinco casos en el Valle de México. Rev Invest Clín, México, 41: 253-257.
32. Castillo M, Araiza A, Caballero A (1994) Leptospirosis. Informe de 61 casos en el Valle de México. Rev Med IMSS. 32: 571-577.
33. García GR, Reyes TA, Basilio HD, Ramírez PM, Rivas SB (2013) Leptospirosis: un problema de salud pública. Rev Latinoamer Patol Clín 60(1): 57-70.
34. García PR (2009) Leptospirosis humana. La Habana: Editorial Científico Técnica.
35. Zunino E, Pizarro R (2007) Leptospirosis. Puesta al día. Rev Chil Infectol 24: 3.
36. Luna AMA (1977) Aspectos clínicos reportados en leptospirosis canina. Primer Seminario Taller Nacional sobre el diagnóstico y control de la leptospirosis, México.
37. Brod CS, Aleixo JA, Jouglard SD, Fernandes CP, Teixeira JL, et al. (2005) Evidence of dog as a reservoir for human leptospirosis: a serovar isolation, molecular characterization and its use in a serological survey canine leptospirosis. Rev Soc Bras Med Trop. 38(4): 294-300.
38. Luna AM, Moles CL, Gavaldón RD, Nava VC, Salazar GF (2005) Estudio retrospectivo de seroprevalencia de leptospirosis bovina en México, considerando las regiones ecológicas. Rev Cubana Med Trop 51(1): 28-39.
39. Secretaría de Salud, Sagarpa, IMSS, UNAM, UAM, UADY. NOM-029-SSA2-1999, para la vigilancia epidemiológica, prevención y control de la leptospirosis en el humano. DF (MX): Diario Oficial de la Federación, México.