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Lyme disease: an important vector-borne bacterial zoonotic disease with emerging concern

Abstract

Vector-borne diseases are a significant cause of morbidity and mortality in humans and animals worldwide. Lyme disease, or *Lyme borreliosis*, is an important emerging vector-borne anthropozoonosis caused by *Borrelia burgdorferi*. It belongs to the genus *Borrelia*, phylum *Spirochetae*. *B. burgdorferi* is a gram-negative, flagellated spirochete having loosely coiled spiral morphology. Lyme disease is distributed in many parts of Asia, Europe, and the United States. It is the most common arthropod-borne disease in the United States. Due to its dramatically increased prevalence, the disease has become a significant public health problem in many parts of the United States. Hunters, hikers, campers, and travelers involved in outdoor activities in forested areas are at risk of getting the infection. Lyme disease is characterized by a fever that may continue for five days. It is transmitted by the bite of the *Ixodid* tick (also known as the black-legged tick), commonly found on deer, rodents, and other small mammals. If diagnosed early, the disease can be managed with antibiotics and supportive treatment. Severe complications may occur in immune-compromised, viral-infected, and malnourished individuals. Due to poor diagnosis and the unavailability of vaccines, prevention from tick bites becomes the key strategy to fight the disease.

Keywords: *Borrelia burgdorferi, ixodes* tick, lyme disease, spirochetes, public health, vector-borne zoonoses

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Introduction

A vector is an arthropod or an invertebrate host that transmits the infection by inoculation into the skin or mucous membrane by biting or depositing infective material on the skin, food, or other objects.[1] There are many vector-borne zoonoses, such as babesiosis, dengue fever, eastern equine encephalitis, ehrlichiosis, epidemic typhus, Japanese encephalitis, Kysanur forest disease, leishmaniasis, Lyme disease, murine typhus, plague, rickettsial pox, scrub typhus, trypanosomiasis, West Nile fever, yellow fever, and others that are reported from developing as well as developed nations of the world.[1–6] Among these, *Lyme borreliosis* (LB), or Lyme disease, is an important zoonotic, vector-borne disease caused by spirochete *Borrelia burgdorferi*. Besides humans, the natural infection has been reported in several species of animals, including deer, dogs, horses, mice, raccoons, squirrels, and opossums.[1] Among vector-borne diseases, LB is the most frequently reported disease in the United States.[7] The skin rash was earlier called Erythema *Chronicum Migrans*, which later became popular as Erythema Migrans (EM). It is one of the earliest and specific symptoms of LB. The pathogenic spirochete *Borrelia burgdorferi* of the sensu lato complex is the causative agent of the disease and is mainly harbored by ticks. Bite of the infected tick, especially of the *Ixodes* genus, is the primary route of disease transmission to humans.[7,8] There are three stages of pathogenesis: early stage or the localized disease, second stage or early dissemination, and third stage or late dissemination. Non-specific flu-like symptoms such as fever, headache, and muscle pain could be the primary symptoms of the disease. Erythema migrans is an early indication of LB. In the absence of proper treatment, erosive arthritis, a condition similar to rheumatoid arthritis, may develop and, ultimately, lead to chronic progressive encephalitis and encephalomyelitis. Cardiac abnormalities and unilateral facial paralysis are also associated with LB.[7,9] Enzyme-linked immunosorbent assay (ELISA) is the most common diagnostic test for LB, but it may give false positive results. Immunoblot (western blot) generally confirms or validates the ELISA.[10] Therefore, two-tier testing is recommended by CDC to confirm the diagnosis of Lyme disease. ELISA test, along with western blot, offers 99.9% precision. There is no current vaccine for LB, which makes the disease important from a public health point of view. Thus, this review aims to provide a general overview of Lyme disease, including pathogen characteristics, diagnosis, treatment, epidemiology, prevention, and control measures.

Historical background

In 1975, Lyme disease was first identified in the rural part of Connecticut, where mysterious outbreaks of rheumatoid arthritis-like symptoms were reported in the affected people.[11] During the outbreak, an unusual cluster of children (51 cases) who have rheumatoid arthritis in a small coastal town in Connecticut called Lyme was reported. The team headed by Allan C. Steere, MD, and Stephen E. Malawista, MD, of Yale School of Medicine, investigated the outbreak and soon suggested the association of the disease occurrence with the bites of infected ticks, specifically of the family *Ixodidae*.[12] In 1977, Yale scientists identified and named the cluster "Lyme arthritis." However, the name was changed to "Lyme disease" in 1979 when newly reported symptoms were associated with the disease.[13] Later, in 1982, Dr. Willy Burgdorfer discovered the bacterium *Borrelia burgdorferi*, the causative agent of the disease, and named after him.[14] The typical skin lesion of LB, i.e., erythema chronicum migrans (now, *Erythema migrans*), is the term initially used by Swedish physician Arvid Afzelius in 1909.[8] Erythema migrans looks like bull's eye rash, occurring 3-30 days after an infected tick bites the person. The rash may appear like a ringworm infection but differs due to the warmness of the lesion without any itching. Later studies documented that the onset of arthritis was a late symptom of a multi-system Lyme disease.

The involvement of vectors in disease transmission may vary according to geography. For example, *Ixodes scapularis* (the deer tick) transmits disease in the United States, whereas other species of *Ixodes* ticks have been reported from Europe and Asia.[12] Infected ticks constitute the main risk of contracting Lyme disease; however, mosquitoes can also play a role in disease transmission.[15]

Since the late 1980s, the incidence of Lyme disease has dramatically increased, especially in the United States. It has become a significant public health problem in many areas of the United States, with the reporting of ~30,000 cases per year.[16] However, increasing evidence from recent studies as well as the CDC estimated that the actual number of diseased people is approximately ten times more than the yearly reported number.[14,17,18]

Epidemiology

Etiology

Borrelia burgdorferi is a tick-borne obligate parasite whose life cycle alternates between arthropod vectors and mammalian hosts. It belongs to the genus *Borrelia*, phylum Spirochetae, which includes a number of *borreliae*. *B. burgdorferi* is a gram-negative, flagellated spirochete having loosely coiled spiral morphology and measure typically from 5-50 µm in length and up to 0.5 µm in width.[19] The cell wall of *Borrelia* consists of a plasma membrane, a peptidoglycan layer, and an outer membrane. *B. burgdorferi* differs from other spirochetes because it has linear chromosomes and circular plasmids. A microaerophilic atmosphere with 30^0C–34^0C temperature is the best for the growth of the organism. Under optimum circumstances, it multiplies every 8–12 h throughout the log phase of growth. Even though *Borrelia* is gram-negative, it's quite different from typical gram-negative bacteria. *B. burgdorferi* possesses many immunoreactive glycolipids on its surface but lacks LPS, which usually presents in the outer membrane of gram-negative bacteria.[20] The Osps (outer surface proteins) in the outer membrane play a vital role during disease transmission from the vector to the susceptible host. *B. burgdorferi* has periplasmic flagella located in the periplasmic space, whereas other bacteria usually have them outside of the cell. Flagellin is the main structural component of flagella. It provides unique rotational motility and confines the overall shape of the *Borrelia*.

Geographical distribution

LB has been described in many countries of the world, including India.[1,21–23] The causative agent belongs to the *Borrelia burgdorferi* sensu lato (sl) complex (s.l.- meaning "in the broad sense). The complex consists of worldwide distributed ~20 genospecies with a complex genome.[24] Although there are around 18 *Borrelia* species known that are transmitted by *Ixodes* ticks,[11] only a few have been considered critical human pathogens, such as *B. burgdorferi* sensu stricto, *B. garinii,* and *B. afzelii,* depending upon the location.[25]

Borrelia burgdorferi sensu stricto (s.s.- "in the strict sense") is the single species in that complex that is responsible for nearly all cases of Lyme disease in North America.[26,27] In the US, LB is primarily caused by *B. burgdorferi* and, to a minor extent, by *B. mayonii*, recently reported from the upper midwestern states.[16] In Europe and Asia, *B. afzelii* and *B. garinii,* respectively, are the most common species causing LB.[24] Another pathogenic *borrelia* species, i.e., *Borrelia miyamotoi,* was identified from Japan.[28] *B. burgdorferi* sensu lato species have been isolated from various animals and ticks, but *B. burgdorferi* sensu stricto is zoonotic in nature and has been isolated only from humans.[27]

The temperate climate of the northern hemisphere is favorable for the survival of the *Ixodes* ticks.[9] In North America, *Ixodes scapularis* is the primary species that transmits LB.[29] *Ixodes ricinus* in Europe, whereas *Ixodes persulcatus* is the predominant tick species responsible for disease transmission in most parts of Russia and Asia.[24] *Borrelia* spp. is not a recently emerged bacterium. The history of bacterial evaluation indicates that diverse lineages of *B. burgdorferi* existed for a very long time in the Northern part of America before the onset of the human outbreak. The existing LB epidemic is the consequence of ecological alterations and human encroachment that have pushed deer, ticks, and, finally, bacteria to invade human dwellings.[13,14]

Transmission

Generally, the *Ixodes* tick has a 2-year life cycle consisting of three stages of life: larva, nymph, and adult, and mostly found in deciduous and mixed forests with mild winters and warm, humid summers.[30] *Borrelia* organisms cannot transmit vertically in arthropods (i.e., through egg infection) and, thus, adopted a strategy for spreading the infection to and from the hosts while the arthropod feeds upon the host. Each molting into the next stage of *Borrelia* occurs after a blood meal. There is host specificity depending upon the stage of the life cycle. It was found that larvae and nymphs feed mainly on the white-footed mouse, whereas fully-grown females feed mainly on white-tailed deer or any infected animal reservoir.[14] The tick inserts its hypostome into the skin of the host and, first, injects the complex mixture of bioactive chemicals into the host. Later while sucking blood, *Borrelia* can be ingested, which travels with the hemolymph to the midgut. Once it reaches the midgut, *B. burgdorferi* remains dormant until the tick molts and attains the next developmental stage. In order to transmit the infection to the host, it is a must for *B. burgdorferi* to remain attached to the host for at least 36 to 48 hours.[31]

The choice of host plays a vital role in disease transmission. Although both nymph and adult ticks can transmit LB to humans,[32] nymphal ticks are accountable for most human infections since they are tinier than adults and difficult to detect. Small mammals, such as white-footed mice, shrews, voles, chipmunks, squirrels, skunks, etc., act as the primary hosts, as well as reservoirs of larvae and nymphs of *Ixodes* tick harboring LB in endemic areas. Whereas large mammals, especially deer, are the preferred host of adult ticks and are essential for efficient egg production by female ticks.[11,14] Until now, ≈237 animal species have been found as hosts for maintaining the population of *I. ricinus*. They, therefore, are available to act as competent reservoir hosts for multiple tick-borne diseases, including LB.[33]

Clinical spectrum

Lyme disease is a multisystemic infection affecting several organs, such as the heart, joints, brain, and central nervous system, and can result in many diverse symptoms. Clinical presentation of the disease is often divided into early and late stages. Early LB includes characteristic symptoms of skin rash (erythema migrans), fatigue, chills and fever, muscle pain, headache, pain in joints, and swollen lymph nodes. Reddish or purple-colored skin rash normally appears 3 to 14 days after the bite of an infected tick. Common sites to find ticks are the thighs, groin, trunk, and armpits of a host. The rashes are not painful but usually warm to the touch. Sometimes, patients with early LB do not develop or notice any rash and instead have "flu-like" symptoms of headache, fever, and muscle pain. It is not necessary that all rashes that appear at the site of a tick bite are due to Lyme disease. It could be an allergic reaction to tick saliva after a tick bite and may cause confusion with an erythema migrans. Therefore, a differential diagnosis is needed. Allergic reactions due to tick saliva may result in a rash, usually within a few hours after the tick bite, which commonly does not expand and disappears within a few days of the bite.[31]

Symptoms of late-stage LB: Some signs and symptoms of Lyme disease appear weeks or months after a tick bite and are considered late stages. Arthritis, with brief attacks of pain and swelling, usually occurs in one or more large joints, especially the knees. Numbness,

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pain, nerve paralysis (often one side of the facial muscles), and meningitis with fever, stiff neck, and headache are the symptoms associated with the nervous system. On rare occasions, irregular heart rhythms may occur in patients. Other disease complications may include memory or concentration issues, headache, fatigue, and sleep disturbances that sometimes resume even after treatment.[9]

Diagnosis

The clinical history plays a vital role in the diagnosis of LB. The appearance of the characteristic rash, i.e., erythema migrans, with a history of potential tick exposure in an endemic area is very suggestive of LB. Early diagnosis and treatment are essential for a good prognosis of the disease. In the absence of treatment, the disease progresses to affect the vital parts of the body like the heart, nervous system, and major joints. The human body releases immunoglobin M (IgM) and immunoglobin G (IgG) in response to *B. burgdorferi* infection, which has diagnostic importance. These *B. burgdorferi*-specific immunoglobulins, IgM and IgG, will appear in serum 2-4 and 4-6 weeks after infection, respectively, and both will peak approximately 6-8 weeks after infection. CDC recommends a two-tiered serological test for the diagnosis of LB. The first step consists of an enzyme immunoassay or immunofluorescent assay using a *B. burgdorferi* sensu stricto lysate or whole cells, respectively. In the second step, an immunoblot for *B. burgdorferi* sensu stricto specific-IgM or IgG using a whole cell lysate is recommended. The two-tier routine is to be followed if the patient presents with relevant symptoms and history. Both tests must show positive results to consider the diagnosis as true positive.[34] The absence of erythema migrans in some patients may pose an additional diagnostic complication. It is advised that patients with a history of tick bites and the presence of skin lesions should be thoroughly investigated for Lyme disease.

Treatment

Doxycycline is the first choice for the treatment of erythema migrans. However, treatment of early localized/disseminated erythema migrans by doxycycline, amoxicillin, and cephalosporins (oral) was found equally effective, with more than 90% response.[35] According to randomized trials, doxycycline for 10 days is as effective as 15 or 21 days.[34] An open-label alternate treatment trial among 200 patients reported that in cases of multiple erythema migrans, oral doxycycline treatment for 14 days is as effective as intravenous ceftriaxone.[9] Intravenous ceftriaxone treatment for at least 14 days usually treats Lyme neuroborreliosis. The neurologic sequelae, impaired cognition, and other long-term complications of the diseases were found to be similar regardless of the treatment regime.[36] For the treatment of Lyme arthritis, oral doxycycline is recommended for 30 days. If the patient still shows symptoms of arthritis, re-treatment is done by giving either doxycycline or intravenous ceftriaxone. Ten to twenty percent of patients may develop a condition called "antibiotic refractory" Lyme arthritis that is unresponsive to antimicrobial therapy. It requires injections of corticosteroids and non-steroidal anti-inflammatory drugs directly administered into the joint (intra-articular injection).[12,37] A systematic review on the efficacy and safety of pharmacological treatments for acute Lyme neuroborreliosis is presented by Dersch and co-workers.[38]

Prevention and control

Avoidance of tick-infested areas is the best approach to prevent incidences of LB. However, occupational exposure, recreational activities, and people living in proximity to woods are more susceptible to disease occurrence. In such circumstances, applying insect repellents [diethyl-meta-toluamide (DEET)] and full body coverage may effectively prevent tick bites. A head-to-toe bath within 2 hours after exposure may also be an effective way to avoid infection. Usually, ticks take more than 2 hours to fully attach to the host body, so an immediate bath is an effective way to wash off any ticks from the body. Regular checking of the clothes, entire body surface, and especially body folds for the presence of ticks. No vaccine is currently available to prevent human LB.[39,40]

Conclusion and recommendations

The dedicated research in B. *burgdorferi* ecology revealed many *Borrelia* spp. isolates recovered from many vertebrates, including humans. However, only three spp. *B. burgdorferi* s. s. (in North America and Europe), *B. afzelii* and *B. garinii* (in Europe and Asia) were found to be pathogenic to humans for a long time. The Lyme disease pathogen, *Borrelia burgdorferi,* is a gram negative, motile bacterium. Rodents act as the reservoir, and deer are the primary hosts of *Ixodes* ticks, an arthropod vector responsible for disease transmission. Since reporting the first outbreak in the US, it has become the most commonly reported tick-borne illness in the US and is also prevalent in central Europe. Moreover, the worldwide burden of the disease has amplified, and now the LB has extended into the locations where the disease was not reported earlier. After two days of exposure to a tick bite, the infection may appear as an unexpected fever or rash. The clinical signs of the diseases may vary from flu-like symptoms to severe arthritis and neurological conditions. The disease can be treated with antibiotics esp., doxycycline, in order to relieve the disease. Diagnosis of the disease is still in the nascent stage. Even though diseases is associated with a considerable loss in terms of health, economy, and social aspect, there is still a need for in-depth knowledge of the ecology of *Borrelia* species involved in human LB worldwide. In this scenario, the best approach is the avoidance to tick encounters. Therefore, the following conclusion and recommendations are forwarded from the precautionary point of view:

a) Animals should be kept away from tick-infested areas.

b) Animals should be de-wormed whenever the season is changed with appropriate acaricides.

c) It is advised to create tick-safe zones in and around your garden/yard.

d) People living in endemic areas should perform daily tick checks on their bodies and be alert for unexpected fever or rash.

e) Emphasis is given to early diagnosis and prompt therapy to prevent the complications.

Author's contribution

All the authors contributed equally. They read the final version and approved it for publication.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

References

1. Pal M. *Zoonoses*. 2nd Ed. Satyam Publishers, Jaipur, India, 2007.
2. Pal M. Importance of zoonoses in public health. *Indian J Anim Sci.* 2005;75(5):586–591.
3. Pal M. Scrub typhus: An emerging and re-emerging metazoonosis of global public health significance. *Madridge J Immunol.* 2017;1(1):16–17.
4. Pal M. Dengue fever: An emerging and re-emerging viral disease of major public health importance. *Madridge J Microbiol.* 2018;2(1):51–52.
5. Pal M, Dave P. Epidemic typhus: a re-emerging rickettsial zoonosis. *Acta Sci Microbiol.* 2019;2(9):104–107.
6. Pal M, Gutama KP, Steinmetz CHD, et al. Leishmaniasis: an emerging and re-emerging disease of global public health concern. *Am J Infect Dis Microbiol*. 2022;10(1):22–25.
7. Pal M, Gutama KP, Shiramo MY, et al. Plague: a re-emerging life-threatening bacterial zoonosis of public health concern. *Acta Sci Microbiol.* 2021;4(11):21–24.
8. Cervantes J. Lyme disease in Perú. A clinical and epidemiological review. *Rev Peru Med Exp Salud Publica*. 2018;35(2):292–296.
9. Elelu N. Tick-borne relapsing fever as a potential veterinary medical problem. *Vet Med Sci.* 2018;4(4):271–279.
10. Trevisan G, Bonin S, Ruscio M. A practical approach to the diagnosis of *Lyme borreliosis*: from clinical heterogeneity to laboratory methods. *Front Med (Lausanne)*. 2020;7,265.
11. Cook MJ. *Lyme borreliosis*: a review of data on transmission time after tick attachment. *Int J Gen Med*. 2015;8:1–8.
12. Antony S. Mosquito and tick-borne illnesses in the United States. Guidelines for the recognition and empiric treatment of zoonotic diseases in the wilderness. *Infect Disord Drug Targets*. 2019;19(3):238–257.
13. Garfinkle SE. Bicentennial: close to home: a history of Yale and Lyme Disease. *Yale J Biol Med*. 2011;84(2):103–108.
14. Stone BL, Tourand Y, Brissette CA. Brave new worlds: the expanding universe of Lyme disease. *Vector Borne Zoonotic Dis*. 2017;17(9):619–629.
15. Bogacka DIK, Grygiel WK, Jaborowska M. Ticks and mosquitoes as vectors of *Borrelia burgdorferi* s. l. in the forested areas of Szczecin. *Folia Biol (Krakow)*. 2007;55(3–4):143–146.
16. New Lyme- disease–causing bacteria species discovered. CDC. 2016.
17. Nelson CA, Saha S, Kugeler KJ, et al. Incidence of clinician-diagnosed Lyme disease, United States, 2005–2010. *Emerg Infect Dis.* 2015;21(9):1625–1631
18. Schiffman EK, McLaughlin C, Ray JA, et al. Underreporting of Lyme and other tick-borne diseases in residents of a high-incidence county, Minnesota, 2009. *Zoonoses Public Health.* 2018;65(2):230–237.
19. Petri WA. Relapsing fever and other *borrelia* infections. Goldman's Cecil Medicine (24th Ed), 2012;1935–1937.
20. He H, Pramanik AS, Swanson SK, et al. A *Borrelia burgdorferi* LptD homolog is required for flipping of surface lipoproteins through the spirochetal outer membrane. *Mol Microbiol*. 2023;119(6):752–767.
21. Joppe WR, Hovius, et al. Coinfection with *Borrelia burgdorferi* sensu stricto and *Borrelia garinii* alters the course of murine *Lyme borreliosis*. *FEMS Immunol Med Microbiol.* 2007;49(2):224–234.
22. Jairath V, Sehrawat M, Jindal N, et al. Lyme disease I Haryana, India. *Indian J Dermatol Venereol Leprol.* 2014;80(4):320–323.
23. Pal M. Lyme disease- emerging metazoonosis of public health concern. *J Clin Immunol Microbiol.* 2021;2(1):1–4.
24. Steere A, Strle F, Wormser G, et al. Lyme borreliosis. *Nat Rev Dis Primers.* 2016;2:16090.
25. Rizzoli A, Hauffe H, Carpi G, et al. Lyme borreliosis in Europe. *Euro Surveill*. 2011;16(27):19906.
26. Rudenko N, Golovchenko M, Grubhoffer L, et al. Updates on *Borrelia burgdorferi* sensu lato complex with respect to public health. *Ticks Tick Borne Dis*. 2011;2(3):123–128.
27. Pritt B, Mead P, Johnson D. Identification of a novel pathogenic *Borrelia* species causing *Lyme borreliosis* with unusually high spirochaetaemia: a descriptive study. *Lancet Infect Dis.* 2016;16(5):556–564.
28. Fukunaga M, Takahashi Y, Tsuruta Y, et al. Genetic and phenotypic analysis of *Borrelia miyamotoi* sp. nov., isolated from the *Ixodid* tick *Ixodes persulcatus*, the vector for Lyme disease in Japan. *Int J Syst Bacteriol*. 1995;45(4):804–810.
29. Xu G, Pearson P, Dykstra E, et al. Human–biting *Ixodes* ticks and pathogen prevalence from California, Oregon, and Washington. *Vector Borne Zoonotic Dis*. 2019;19(2):106–114.
30. Ostfeld RS, Brunner JL. Climate change and Ixodes tick–borne diseases of humans. *Philos Trans R Soc Lond B Biol Sci.* 2015;370(1665):20140051.
31. McKenzie I, Tsarfati E. Lyme radiculopathy in a septuagenarian. *BMJ Case Rep*. 2023;16(6):e251982.
32. Norris S. *Lyme disease*. Encyclopedia of microbiology (3rd Ed). 2009;199–207.
33. Gern L. *Borrelia burgdorferi* sensu lato, the agent of *Lyme borreliosis*: life in the wilds. *Parasite*. 2008;15(3):244–247.
34. Yeung C, Baranchuk A. Systematic approach to the diagnosis and treatment of Lyme carditis and high–degree atrioventricular block. *Healthcare (Basel)*. 2018;6(4):119.
35. Bransfield RC. Neuropsychiatric Lyme borreliosis: an overview with a focus on a speciality psychiatrist's clinical practice. *Healthcare (Basel)*. 2018;6(3):104.
36. Arnez M, Sabljic ER. Azithromycin is equally effective as amoxicillin in children with solitary erythema migrans. *Pediatr Infect Dis J*. 2015;34(10):1045–1048.
37. Torbahn G, Hofmann H, Rucker G. Efficacy and safety of antibiotic therapy in early cutaneous Lyme borreliosis: a network meta-analysis. *JAMA Dermatol*. 2018;154(11):1292–1303.
38. Dersch R, Freitag M, Schmidt S. Efficacy and safety of pharmacological treatments for acute Lyme neuroborreliosis- a systematic review. *Eur J Neurol.* 2015;22(9):1249–1259.
39. Jacquet C, Goehringer F, Baux E, et al. Multidisciplinary management of patients presenting with Lyme disease suspicion. *Med Mal Infect*. 2019;49(2):112–120.
40. Pal M. Ehrlichiosis: an emerging tick-borne zoonotic disease. *Acta Sci Microbiol.* 2020;2:3–5.

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