

Lyme Disease: A Zoonosis Tick-Borne Borrelia Bacterium [3/4]

Moumaris M*

Institute of Medical Sciences, Research and Development Company, France

***Corresponding author:** Mohamed Moumaris, Research and Development Company, France, Tel: +33762122825; Email: mohamed.moumaris@sciencesettechnologies.com

Keywords: Borrelia Bacterium; Spirochete; Gram Negative Bacterium

Abbreviations: CAA: Chronic Atrophying Acrodermatitis; DBPS: Decorin Binding Proteins; ECG: Electrocardiogram; ECM: Erythema Chronicum Migrans; EM: Erythema Migrans; NMDA Receptor: N-Methyl-D-Aspartate Receptor.

Editorial

Lyme disease, medically known as borreliosis, initiates with a skin manifestation known as erythema chronicum migrans (ECM). ECM is a ring-shaped patch with inflammation that appears around the tick bite and typically develops within a month after being bitten (Figure Editorial Volume 6 Issue 4

Received Date: August 01, 2023 Published Date: August 18, 2023 DOI: 10.23880/izab-16000500

1). If not treated, borreliosis can advance to a later stage, leading to neurological, articular, and cardiac complications. Lyme disease is an intricate illness that manifests in various symptoms affecting multiple bodily systems. The incubation period typically ranges from one to two weeks. Symptoms can present themselves anywhere from a few days to several years later. The primary clue is the development of a circular or oval-shaped rash known as erythema migrans (EM) at the tick bite site. This rash usually appears within one to two weeks but can take up to 32 days. Patients may experience flulike symptoms such as tiredness, migraines, muscle soreness, fever, and shivering sensations. Diagnosing EM relies heavily on clinical manifestations since many patients do not recall being bitten by a tick. However, appropriate antimicrobial treatment generally Leads to favorable outcomes [1].



Lyme disease, transmitted by ticks, manifests with erythema migrans. If untreated, it can result in complications affecting the nervous system, joints, heart, and skin. Neurological complications include meningitis, encephalitis, memory and behavioral disorders, and fatigue. Diagnosis involves serological tests and cerebrospinal fluid analysis when the central nervous system is affected. Proper medical attention is crucial to prevent further harm, as timely treatment can mitigate severe symptoms and complications [2,3]. Lyme disease, caused by Borrelia burgdorferi transmitted through tick bites, presents diverse symptoms and complications. These include migratory polyarthritis and cardiac issues such as atrioventricular blocks, which occur after the initial ECM rash. Chronic rheumatic conditions can affect joints, while dermatological symptoms involve chronic atrophying acrodermatitis (CAA). Lyme arthritis, a late complication, causes swelling, discomfort, and inflammation in large joints like the knee (Figure 2) [4]. Additionally, Lyme disease can trigger Lyme encephalopathy, characterized by cognitive problems, insomnia, and personality changes. It may be mistaken for a central nervous system infection [5]. Lyme borreliosis exhibits three skin manifestations: erythema migrans, borrelial lymphocytoma, and ACA. ACA initially appears as a reddish-blue patch that gradually atrophies over time [6].



Figure 2: Lyme Arthritis from Lyme Disease. This Image is Licensed under Creative Commons Attribution.

Borrelia burgdorferi causes Lyme disease transmitted through tick bites. Tick saliva contains substances that hinder blood clotting, reduce inflammation, and suppress the immune response, aiding bacterial spread. Spirochetes adapt within the host, protected by tick saliva and modified bacterial proteins. They travel through the bloodstream, targeting specific organs like the skin, joints, and neurons.

Borrelia burgdorferi spreads throughout the body, causing various symptoms, including the circular skin lesion called erythema migrans. Despite challenges, the bacteria thrive, leading to Lyme disease [7]. These bacteria modify their surface proteins and produce decorin-binding proteins (Dbps) A and B, which bind to decorin in the host's matrix to evade the immune system [8]. The spirochetes stimulate the release of quinolinic acid, activating the NMDA receptor associated with encephalopathy [9,10]. Exposure to the Borrelia bacterium in Lyme disease is related to chronic arthritis characterized by persistent inflammation. Thus the immune system activates inflammatory mediators that contribute to inflammation in chronic arthritis characterized by joint swelling and inflammation. Lyme carditis can result in an atrioventricular block, disrupting heart conduction. Accurate diagnosis requires symptom evaluation, serological testing, ECG, and echocardiography [11-13].

These editorials highlight the clinical, epidemiology, and diagnosis of tick-borne pathogenic Borrelia. Ticks' role in transmitting Lyme disease is significant, so highlighting the infectious agents tick-borne to humans and animals is primordial [14]. There are two barriers to tick-borne Lyme disease, the host's immunity to tick bites and the tick's immunity to pathogens [15]. In Lyme disease, exposure of the host's immune system to the bacterium Borrelia induces chronic immune disease. Many targeting molecules have a role in modulating the immunity system against Lyme disease [16-25]. Accurate and rapid diagnosis with high sensitivities is one of the challenges in the medical field of infectious diseases for quick treatment in infected patients. The immune system responds to Borrelia burgdorferi infection by producing antibodies, resulting in humoral and cell-mediated immune reactions. Detection of the humoral immune response depends on detecting the antibody response. The diagnosis of Lyme disease is still a significant concern.

Acknowledgments

The author acknowledges Mrs. Norri Zahra and Mr. Regragui Moumaris. The author thinks Nisen Abuaf and Said Youssouf Chanfi (Sorbonne University). The author thinks Jean-Michel Bretagne (AP-HP). The author thinks Marie-Hélène Maës and Monique Abuaf (16th district of Paris).

References

- 1. Nadelman RB (2015) *Erythema Migrans*. Infect Dis Clin North Am 29(2): 211-239.
- 2. Halperin JJ (2015) Nervous System Lyme Disease. Infect Dis Clin North Am 29(2): 241-253.
- Stanek G, Strle F (2018) Lyme Borreliosis-from Tick Bite to Diagnosis and Treatment. FEMS Microbiol Rev 42(3): 233-258.
- 4. Arvikar SL, Steere AC (2022) Lyme Arthritis. Infect Dis Clin North Am 36(3): 563-577.
- 5. Eckman EA, Pacheco Quinto J, Herdt AR, Halperin JJ (2018) Neuroimmunomodulators in Neuroborreliosis and Lyme Encephalopathy. Clin Infect Dis 67(1): 80-88.
- Müllegger RR, Glatz M (2008) Skin Manifestations of Lyme Borreliosis: Diagnosis and Management. Am J Clin Dermatol 9(6): 355-368.
- 7. Martin Y, Zimmerli S (2022) [Lyme Disease Epidemiology and Pathophysiology]. Ther Umsch 79(9): 441-447.
- 8. Liang FT, Brown EL, Wang T, Iozzo RV, Fikrig E (2004) Protective niche for Borrelia burgdorferi to evade humoral immunity. Am J Pathol 165(3): 977-985.
- 9. Ramesh G, Philipp MT (2005) Pathogenesis of Lyme neuroborreliosis: mitogen-activated protein kinases Erk1, Erk2, and p38 in the response of astrocytes to Borrelia burgdorferi lipoproteins. Neurosci Lett 384(1-2): 112-116.
- Lugo-Huitrón R, Muñiz PU, Pineda B, Pedraza-Chaverrí J, Ríos C, et al. (2013) Quinolinic acid: an endogenous neurotoxin with multiple targets. Oxid Med Cell Longev pp: 104024.
- 11. Pancewicz SA, Rutkowski R, Rutkowski K, Zajkowska JM, Kondrusik M, et al. (2007) [Immunopathology of Lyme arthritis]. Pol Merkur Lekarski 23(134): 141-144.
- 12. Radesich C, Mestre ED, Medo K, Vitrella G, Manca P, et al. (2022) Lyme Carditis: From Pathophysiology to Clinical Management. Pathogens 11(5): 582.
- 13. Arvikar SL, Steere AC (2015) Diagnosis and treatment of Lyme arthritis. Infect Dis Clin North Am 29(2): 269-280.
- 14. Ben Said M, Diaz Sanchez S, Bastos A, Silaghi C (2022) Editorial: Current Knowledge on Pathogenic and

Endosymbiotic Tick-Borne Bacteria. Front Vet Sci 9: 900510.

- 15. Tabor AE, de Miranda Santos IKF, Boulanger N (2021) Editorial: Ticks and Host Immunity - New Strategies for Controlling Ticks and Tick-Borne Pathogens. Front Immunol 12: 796558.
- 16. Moumaris M (2022) Lyme Disease: A Zoonosis Tick-Borne Borrelia Bacterium [1/4]. Int J Zoo Animal Biol 5(6): 1-3.
- 17. Moumaris M (2023) Lyme Disease: A Zoonosis Tick-Borne Borrelia Bacterium [2/4]. Int J Zoo Animal Biol 6(2): 1-4.
- 18. Moumaris M, Bretagne JM, Abuaf N (2020) Nanomedical Devices and Cancer Theranostics. The Open Nanomedicine and Nanotechnology Journal 6: 1-11.
- 19. Moumaris M, Bretagne JM, Abuaf N (2019) Biological Membranes and Malaria-Parasites. The Open Parasitology Journal 7: 1-18.
- 20. Moumaris M, Bretagne JM, Abuaf N (2018) Hospital Engineering of Medical Devices in France. The Open Medical Devices Journal 6: 10-20.
- Moumaris M, Rajoely B, Abuaf N (2015) Fluorescein Isothiocyanate-Dextran can track Apoptosis and Necrosis induced by heat shock of Peripheral Blood Mononuclear Cells and HeLa Cells. Open Biological Sciences Journal 4: 7-15.
- 22. Moumaris M, Rajoely B, Abuaf N (2012) The Naïve B Cells are the Lymphocytes with the Highest Anionic Phospholipid Binding Ratios. The Open Immunology Journal 5: 27-35.
- 23. Moumaris M, Abuaf N (2002) Use of labeled dextran for in-vitro assessment of increased cell permeability, cell death and apoptosis. Bulletin officiel de la propriété industrielle (Brevet n°00/09235) 2811682: A3.
- 24. Moumaris M, Benoliel S, Rouquette AM, Rajoely B, Abuaf N, et al. (2000) Phospholipid binding proteins on the plasma membrane of lymphocytes. J Autoimmun 15: A33.
- 25. Moumaris M, Sestier C, Miltgen F, Halbreitch A, Gentilini M, et al. (1995) Effect of Fatty Acid Treatment in Cerebral Malaria-Susceptible and Nonsusceptible Strains of Mice. The Journal of Parasitology 81(6): 997-999.

