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Persisting atypical and cystic forms of Borrelia burgdorferi and local inflammation in Lyme neuroborreliosis.

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 BACKGROUND: The long latent stage seen in syphilis, followed by chronic central nervous system infection and inflammation, can be explained by the persistence of atypical cystic and granular forms of Treponema pallidum. We investigated whether a similar situation may occur in Lyme neuroborreliosis. METHOD: Atypical forms of Borrelia burgdorferi spirochetes were induced exposing cultures of Borrelia burgdorferi (strains B31 and ADB1) to such unfavorable conditions as osmotic and heat shock, and exposure to the binding

agents Thioflavin S and Congo red. We also analyzed whether these forms may be induced in vitro, following infection of primary chicken and rat neurons, as well as rat and human astrocytes. We further analyzed whether atypical forms similar to those induced in vitro may also occur in vivo, in brains of three patients with Lyme neuroborreliosis. We used immunohistochemical methods to detect evidence of neuroinflammation in the form of reactive microglia and astrocytes. RESULTS: Under these conditions we observed atypical cystic, rolled and granular forms of these spirochetes. We characterized these abnormal forms by histochemical, immunohistochemical, dark field and atomic force microscopy (AFM) methods. The atypical and cystic forms found in the brains of three patients with neuropathologically confirmed Lyme neuroborreliosis were identical to those induced in vitro. We also observed nuclear fragmentation of the infected astrocytes using the TUNEL method. Abundant HLA-DR positive microglia and GFAP positive reactive astrocytes were present in the cerebral cortex. CONCLUSION: The results indicate that atypical extra- and intracellular pleomorphic and cystic forms of Borrelia burgdorferi and local neuroinflammation occur in the brain in chronic Lyme neuroborreliosis. The persistence of these more resistant spirochete forms, and their intracellular location in neurons and glial cells, may explain the long latent stage and persistence of Borrelia infection. The results also suggest that Borrelia burgdorferi may induce cellular dysfunction and apoptosis. The detection and recognition of atypical, cystic and granular forms in infected tissues is essential for the diagnosis and the treatment as they can occur in the absence of the typical spiral Borrelia form.

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