Alzheimer’s disease - Aspirochetosis

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The fact that pathogens may suppress, subvert or evade host defenses and establish chronic or latent infection has received little attention in the past. Increasing number of recent observations show the involvement of pathogens in various chronic inflammatory disorders, including stomach ulcer, atherosclerosis, cardio- and cerebrovascular disorders, diabetes, neurodegenerative disorders, and Alzheimer’s disease. More than a century ago, in 1907, Fischer suggested the possibility that senile plaques may correspond to colonies of micro-organisms. It has also been known from more than a century that chronic bacterial infection, namely spirochetal infection can cause dementia. Treponema pallidum, the causative agent of syphilitic dementia, can reproduce the hallmarks of AD, including beta amyloid deposition. Recent observations indeed show that various types of spirochetes, including six periodontal pathogen Treponema spirochetes and Borrelia burgdorferiare associated with AD-type lesions, indicating that in an analogous way to Treponema pallidum they can persist in the brain, establish chronic infection and cause dementia and beta amyloid deposition. Association of co-infecting pathogensin biofilm formation may further worsen the degenerative process and the outcome of dementia. A recent analysis of the substantial amount of data available in the literature indicates a statistically significant association between spirochetes detected in the brain and Alzheimer’s disease with a high risk factor. Analysis of causality following Koch’s and Hill’s criteria is in favor of a causal relationship. Following Hill if a probable causal relationship is established prompt action is needed. Further studies are necessary to detect and characterize all types of spirochetes and co-infecting microorganismsinvolved in Alzheimer’s disease. This emerging field of research needs attention, as similarly to syphilitic dementia, Alzheimer’s dementia might be prevented.

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