Editorial
Volume 6:1, 2022

Journal of Microbial Pathogenesis

Open Access

Pathogens associated with Alzheimer's disease

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Editorial

Alzheimer's disease (AD) is a chronic neurodegenerative illness characterized by amyloid-peptide overproduction and buildup in the brain, as well as tau protein hyperphosphorylation. Despite substantial research into the amyloid-based mechanism of AD pathogenesis, the root cause of the disease remains unknown. There are currently no disease-modifying medicines available, and multiple clinical trials have failed to show any benefits. The recent revelation that the amyloid-peptide possesses antimicrobial properties supports the hypothesis of an infectious etiology for AD and shows that infection could cause amyloid plaque formation. Patients with Alzheimer's disease have a compromised blood-brain barrier and immune system, making them more susceptible to microbial infections. Chronic neuroinflammation, development of the antimicrobial amyloid-peptide, and neurodegeneration can all result from such infections. Viruses, bacteria, fungus, and parasites have all been linked to Alzheimer's disease. Individual pathogens have been the focus of most study in this field, with herpesviruses and periodontal bacteria being the most implicated. There are currently no disease-modifying medicines available, and multiple clinical trials have failed to show any benefits. The recent revelation that the amyloid-peptide possesses antimicrobial properties supports the hypothesis of an infectious etiology for AD and shows that infection could cause amyloid plaque formation. Patients with Alzheimer's disease have a weaker blood–brain barrier and immune system, making them more susceptible to microbial infections. Chronic neuroinflammation, development of the antimicrobial amyloid-peptide, and neurodegeneration can all result from such infections. Viruses, bacteria, fungus, and parasites have all been linked to Alzheimer's disease.

The Herpesviridae family consists of eight double-stranded DNA viruses that can infect people and cause neurological illness. Several studies have found links between several herpetic viruses, a loss in cognitive ability, and Alzheimer's disease. HSV-1 and CMV are the most investigated viruses in this context. In a restricted group of samples, a new approach for viral DNA amplification from formalin-fixed AD brain tissue revealed the presence of HSV-1 and CMV but not HSV-2. The discovery of Treponema pallidum in the paretic brains of syphilitic patients was one of the earliest pieces of evidence suggesting bacterial involvement in the development of neurological diseases. Over 70 years later, another bacterium, Borrelia burgdorferi, was discovered in the brains of Alzheimer's sufferers. Both *T. pallidium* and *B. burgdorferi* are members of the phylum Spirochaetes, which, like herpes viruses, has neurotrophic properties and can enter a latent state following infection. *Treponema* species were found in various brain regions of Alzheimer's patients, and in some cases, numerous species were discovered [1-3].

Co-infection with *Helicobacter pylori* and periodontal infections may also affect the beginning of Alzheimer's disease. Another well-designed serological study looked at antibody levels against seven periodontal bacteria and found that in Alzheimer's patients, antibody levels against *Fusobacterium nucleatum* and *Prevotella intermedia* were significantly higher (p = 0.05). Antibodies to different yeast cells, fungal proteins, and (1,3)-glucans were discovered in the blood serum of Alzheimer's patients in early research. A total of eleven AD patients out of a total of 29 showed strong responsiveness to the majority of Candida species examined, while two more patients showed high reactivity to a single Candida species. Furthermore, 6 of 29 patients with AD had very high levels of fungal antigens, 8 had high levels, and 8 patients had high levels of antigens coming from at least one Candida spp. and moderate levels of antigens from at least one other species. Fungitell assays revealed the presence of fungal polysaccharides in the blood serum of 28 of the 29 AD patients, implying that practically all of the patients had a widespread fungal infection.

The multi-microbial infection theory combines two previously established AD hypotheses:

I. Antimicrobial A peptide synthesis as part of an innate immune response, and

II. Neuroinflammation stimulation. New antiviral, antibacterial, anti-inflammatory, anti-fungal, and anti-biofilm therapeutic strategies for Alzheimer's disease can be imagined, based on systematic diagnostic testing for multiple pathogens followed by therapy with antiviral, antibacterial, anti-inflammatory, anti-fungal, and anti-biofilm agents.

Antibiotics are essential medications for treating bacterial and fungal illnesses. Doxycycline and rifampicin are the most commonly studied antibacterial drugs in the setting of Alzheimer's disease (rifampin). There has been no evidence of senile plaques in leprosy patients who have had long-term rifampicin treatment. Changes in brain functionality develop long before the onset of AD-induced cognitive failure, which is important for prevention. Furthermore, some fungi and bacteria have been found in disease-free control subjects, and multiple investigations have linked infectious burden to impaired cognitive performance in adults [4-5].

Conflict of Interest

None.

References

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Received 02-Feb-2022, Manuscript No: jmp-22-58311; **Editor assigned:** 04-Feb-2022, Pre QC-No. P-58311; **Reviewed:** 09-Feb-2022, QC No. Q-58311; **Revised:** 14-Feb-2022, Manuscript No. R-58311; **Published:** 19-Feb-2022. DOI:10.37421/jmp.22.6.109

How to cite this article: Ming, Chien. "Pathogens associated with Alzheimer's disease." J Microb Path 6 (2022): 109.