The 2nd International Neuroinflammation Congress and 2nd Student Festival of Neurosience

Shefa Neuroscience Research Center, Tehran, Iran, 17-19 April, 2018

The Neuroscience Journal of Shefaye Khatam

Volume 6, No. 2, Suppl 1

Poster Presentation

The Role of Periodontitis in Alzheimer Pathogenesis

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Published: *17 April, 2018*

Abstract

Alzheimer disease (AD), the most common cause of dementia, is an irreversible progressive neurodegenerative condition. It is characterized by the salient inflammatory features, microglial activation and increased levels of pro-inflammatory cytokines which contribute to the inflammatory states of the CNS. Accumulating data suggest the key role of inflammation in AD pathogenesis. In a prospective longstanding study, increased level of serum Tumor Necrosis factor alpha (TNF-alpha) and C Reactive Protein (CRP) following both acute and chronic systemic inflammation is associated with cognitive decline in AD patients. Periodontitis is the most common oral infection which is initiated by gram-negative bacteria like spirochetes. It's associated with a raised serum pro-inflammatory state with increasing in (CRP), Tumor Necrosis Factor-alpha, interleukin1 and interleukin6 levels. Periodontitis can lead to progression of the AD through two probable mechanisms; Pro-inflammatory cytokines produced by periodontopathic microorganisms and host response to a systemic/peripheral inflammation. Inflammatory molecules can pass the blood-brain barrier to activate microglia cells which results in neural damage. The second mechanism is due to brain invasion by microorganisms present in dental plaque biofilm. They can enter brain either through the bloodstream or via peripheral nerves. Although lacking causal relationship between periodontitis and AD, periodontitis may be accounted as one of the possible risk factors for perpetuating the neurodegenerative process of the AD. Cohort studies profiling the oral clinical pre-sensation with different cognitive functions and during the progression of the AD is needed to clarify this casual association.

Keywords: Periodontitis, Alzheimer, CNS

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