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Can gingivitis cause Alzheimer's ?

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Abstract:

Recent research shows that one of the organisms that we find in periodontal disease, the ***Porphyromonas gingivalis***, are being found in the brains of Alzheimer's patients at a much higher degree than those without Alzheimer's Disease.

Introduction:

Alzheimer's disease is an irreversible, progressive brain disorder that slowly destroys memory and thinking skills and, eventually, the ability to carry out the simplest tasks. As a person's condition declines, they often withdraw from family and society. Gradually, bodily functions are lost, ultimately leading to death.

The cause of Alzheimer's disease is not clear yet. About 70% of the risk is believed to be inherited with many genes usually involved. Other risk factors include a history of head injuries, depression, and hypertension.

The disease process is associated with abnormal clumps in the Alzheimer brains which they are called ***Amyloid plaques***, and tangled bundles of fibers called ***neurofibrillary, or tau, tangle.***

There is no treatments stop or reverse its progression, but there are drug treatments that can temporarily ease symptoms, or slow down their progression.(2)

Gingivitis is a normal and mild type of gum disease (periodontal disease) that causes the gingiva, the part of the gum around the base of the teeth, to become irritated, swollen, and swelling (inflammation).

It occurs when plaque, (a naturally occurring sticky film that contains bacteria), builds up on the teeth and causes inflammation of the gum tissue around it. Plaque is producing toxins which irritate the gums and triggers an immune response, this can cause the gums to become inflamed, leaving them red or puffy, or bleeding.

When gingivitis is left untreated, it can advance to periodontal disease, and the ***Porphyromonas gingivalis*** bacteria will be found at the gingival sites in periodontal disease.

Porphyromonas gingivalis Found almost exclusively at subgingival sites, particularly in advanced periodontal disease: considered a parodontal pathogen by consensus. Tongue and tonsils also recovered.

It's a Non-motile, asaccharolytic, short, pleomorphic, Gram-negative coccobacilli. And it Produces a range of virulence factors including collagenase, endotoxin, fibrinolysin, phospholipase A, many proteases that destroy immunoglobulins, gingipain, a fibroblast-inhibitory factor, complement and haem-sequestering proteins and a haemolysin. [1]

Materials and Methods :

the data was collected from , which used the following : the gum of a healthy mice, were swapped with ***P. gingivalis*** to spread the infection in their gum.

Later on the bacteria was detected in their brain (which caused "higher level" of dying neurons and ***β-amyloid*** protein.

Discussion :

P.gingivalis can be found in the brains of deceased people with Alzheimer's, and they detected the microbe's DNA in living patients' spinal fluid. In more than 90% of more than 50 Alzheimer's brain samples, they also spotted toxic enzymes produced by the ***P. gingivalis***, called ***Gingipains***, it interacts with Alzheimer's proteins which they are "***amyloid-β and Tau***".

The researchers found Gingipains causes amyloid-β to accumulate and it reacts with it to form *amyloid plaques.* This insoluble plaque creates inflammation and destroys brain cells, and gingipains makes *Tau* to behave abnormally, both are signs of Alzheimer's disease. This shows that P. gingivalis and gingipains in the brain play a central role in the development of Alzheimer's disease.

The reason why the majority of (AD) patients have a higher risk of development of gingivitis and periodontal disease is that their oral hygiene is very compromised .[4]

Thu resulted halitosis and discomfort can also hinder the carers of these patients and taking care of their oral hygiene ,thus negative cycle leads to higher rate of the development in periodontal destruction leading to sever periodontal disease.

(mainly attributed to poor plaque control), resulting in a long term inflammatory load or systemic circulation which eventually leads to neural degeneration.

The exact role of ***P. gingivalis*** in the etiology of (AD) is still in conclusive.

Even though it is somehow the central player in its development these hypothesis is supported by a wide variety of mice base experiment which eventually display neuropathology that are similar to the ones found in Alzheimer patients thus giving a link between the accumulation of this protein and development of (AD).[4]

Conclusion:

Inflammation plays a significant role in increasing the brain's inflammatory state which make contribute to the progression of A.D.

The hypothesis is that P.D (periodontal disease) gingivitis may enhance the inflammation in the brain by the two previously mentioned mechanisms in the discussion, the interaction between the periodontopathic bacteria and host response results in locally increased production of inflammatory molecules.

Understanding the factors and the mechanisms involved in the initiation and progression of A.D is very important, although no direct evidence is associating the two diseases indirect findings suggest a great possibility and if this hypothesis is true then there are several implications such as the presence of P.D may alert the patient to the risk of acquiring A.D later in life, and the second important aspect, is that the early diagnosis and treatment of P.D may limit the severity and progression of A.D.[3]

References:

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