Alzheimer’s, cognitive decline linked to periodontal disease

Treating periodontitis could reduce, delay risk

By David L. Hoexter, DMD, FACD, FICD, Editor in Chief

Recent studies provide increasing evidence that untreated periodontal inflammation is linked to cognitive decline and Alzheimer’s disease, and that treating periodontitis may reduce or delay risk of this disease.

About one out of every nine Americans have some form of Alzheimer’s disease, according to the Alzheimer’s Society. It is the worst type of dementia, involving the most cognitive decline and memory loss. According to Alzheimers.org, the magnitude and severity of this fatal disease mandates that research continues to explore every avenue that might lead to a cure.

Researchers have already found Porphyromonas gingivalis (P. gingivalis) in brains of Alzheimer’s patients. P. gingivalis is a bacterium associated with chronic periodontitis. Researchers propose that when these bacteria reach the brain, an immune response is stimulated in the brain to release proteins that kill the bacteria, but also cause broader destruction.

Periodontal disease bacteria enter bloodstream, brain

A study by professor Stjoh Cread and Dr. Sim Singhrao at the University of Central Lancashire (UClan) School of Medicine and Dentistry in England involved the examination of brain tissue samples of 10 deceased people with Alzheimer’s and 10 people without it.

It was found that bacteria found in chronic periodontal disease were present in the brains of four of the 10 people with Alzheimer’s but in none of the 10 without it. Researchers theorized that chronic periodontal disease bacteria, P. gingivalis, enter the bloodstream and brain, prompting an immune system response, which over time is thought to contribute to cognitive decline and Alzheimer’s disease.

Amyloid proteins trap microbes

Recently, there was a study done at Massachusetts General Hospital, funded by the National Institutes of Health. Dr. R. Tanzi’s research with live mice showed that beta amyloid proteins in live mice trapped microbes — but the microbes remained free in live mice without the amyloid proteins.

Results indicated that with no amyloid to trap the infections, infection spread rapidly, but with the amyloid, infection was stopped. However, the study revealed that too much beta amyloid protein can over clump down on itself, without trapping microbes — indicating that efficacy requires a delicate balance.

Tanzi further theorized that these play a large role, specifically ApoE2 and ApoE4. The gene ApoE2 enabled the brain to clean up the beta amyloid after it killed the microbes, leading to minimal cognitive decline in old age. The gene ApoE4, however, was inefficient at enabling such a “cleanup,” and the risk of developing Alzheimer’s disease was much greater.

All blood vessels are anastomosed in the human body. Therefore, all have connecting relationships and can transmit inflammatory by-product easily. As mentioned, numerous articles allude to this connection with regard to periodontal tissue circulating to the heart and brain.

At last year’s induction of fellowships for the International Academy for Dental Facial Esthetics in New York, Dr. Ray Williams, former dean of Stony Brook Dental School and a highly regarded researcher, presented a detailed overview of the latest findings on periodontal vascularity.

Williams listed numerous publications showing a direct vascularity relationship between the periodontia and the heart. However, Williams also listed an equal amount of publications that infer no direct connection between periodontia and the heart.

Ability to self-maintain oral health can diminish with age

A key area that needs to be addressed is the challenge of self-maintenance of periodontal disease as people’s physical abilities diminish with age.

When I had the privilege to be involved with several senior-care facilities, it was obvious that a large number of residents were unable to maintain their oral health at a desired level. Some residents with dentures would misplace or lose a prosthesis or even borrow one from another resident.

Earlier this year, researchers at Harvard University conducted a study hypothesizing that infections such as chronic periodontitis produce reactions that leave debris in the brain that later contribute to Alzheimer’s disease.

The study found that in the subjects involved, bacteria passed through the blood-brain barrier, prompting the brain’s defense mechanism to release the “sticky” beta amyloid protein. This protein entrapped and killed the bacteria, but what remained was plaque, termed “the hallmark of Alzheimer’s.” This plaque signaled another brain defense mechanism — a release of a tangle of tau proteins that not only killed the plaque, but also the brain’s nerve cells, leading to more and more brain inflammation — and resulting in continued cognitive decline.

Vicious, repetitive cycle

The vicious, repetitive cycle leads to further and further destruction. However, the brain was adversely affected because of the periodontal bacteria. Researchers concluded that genetically, some brains (ApoE2) can clean out the beta amyloid, get rid of the plaque and lower the risk for Alzheimer’s — but other brains (ApoE4) can’t, correlating to a higher risk of getting the disease.

Alzheimer’s disease, with its cognitive decline and memory loss, is destructive not only to the patient, but to all of the patient’s loved ones. Understanding and addressing periodontal vascularity may well be a major pathway toward better and longer functioning of the brain.

Perhaps going to the periodontist is the most expedient, economical and easiest pathway to slowing cognitive decline and onset of Alzheimer’s disease.

Researchers theorize that the chronic periodontal disease bacteria, P. gingivalis, enter the bloodstream and brain, which over time is thought to prompt an immune system response that contributes to cognitive decline and Alzheimer’s disease.

Left: APP to beta-amyloid to plaque. Below, left: Healthy, and diseased neurons and formation of tau tangles. Images Provided by National Institute on Aging/ National Institutes of Health.

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