

Periodontitis - A Part of Aging or a Consequence?

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Abstract:

One of the most common chronic illnesses in the world, periodontitis spreads more readily as people age and is a key factor contributing to tooth loss. The relationship between frailty and periodontal disease has been linked to co-morbid systemic disorders, impaired physical functioning, and restricted capacity for self-care in elderly adults with frailty. Additionally, inflammatory dysregulation along with additional age-associated physiological modifications show substantial connections with both ageing and periodontal disease. The review aims to understand the relationship between geriatric periodontal health, changing periodontium, their interactions, pathology, clinical characteristics, and the specific treatment needed for elderly patients.

Keywords: Aging, Periodontitis, Age Changes, Periodontium, Reduced Periodontium

Introduction

Millions of people worldwide suffer from periodontitis, a serious gum infection that raises an important question: is it a natural aspect of ageing or the result of other factors? Although growing older is a contributing factor to the development of periodontitis, this oversimplification would obscure the complexity of the condition. Instead, it results from a complex interaction between a number of variables, such as lifestyle, genetics, dental hygiene, and overall health. In order to clarify the complex aetiology of periodontitis and its implications for successful management and preventative techniques, this introduction aims to examine the link between the disease and ageing.

The lifespan of man has exponentially increased over time with due credit to modern medicine. However, it has been observed to be accompanied by its own set of 'age-related' problems. This has taken the centre stage in the arena of the developing science. These conditions are treated differently from pathologies, as they are considered to be rather imminent in all individuals. The WHO identifies this population as susceptible and defines effective aging as reduced likelihood of diseases and illness-related disabilities, high intellectual and physical abilities, and proactive involvement with life". In agreement with, dental health considers retention of 20 or more teeth after 80 years of age as a success [1][2]. Periodontitis is said to be one of the main causes of tooth loss. Not only is it a functional and aesthetic concern, but it also has its manifestation in the systemic health of the individuals, making it the foremost concern.

Oral health changes associated with aging are closely related, and among the most common disorders affecting the elderly is periodontal disease. In older populations, periodontitis, a chronic inflammatory disease that affects the gums and underlying bone that support teeth, can be very problematic for dental health and general well-being. Individuals have physiological changes as they age, which might impact the severity and susceptibility to periodontitis. Immune system dysfunction, compromised tissue repair systems, and elevated systemic inflammation are some of these modifications. As a result, older folks are more vulnerable to the advancement of periodontal disease since they frequently have a decreased capacity to fight against periodontal infections and manage inflammatory reactions.

Furthermore, there is a strong correlation between periodontitis and systemic illnesses like diabetes and cardiovascular disease that are more common as people age. For example, diabetes increases periodontal inflammation and impairs immune function in a reciprocal interaction that exacerbates both disorders. Likewise, there is a synergistic effect on overall health outcomes in older persons between periodontitis and cardiovascular disease due to shared risk factors and inflammatory pathways.

In elderly populations, lifelong lifestyle variables also have a role in the onset and progression of periodontitis. Inadequate diet, smoking, and poor dental hygiene all have a substantial negative influence on periodontal health and raise the chance of disease initiation and severity in the elderly.

Moreover, the cumulative consequences of untreated or poorly managed periodontitis from earlier life stages frequently accompany aging. Over time, the degradation of periodontal tissues is caused by chronic exposure to inflammatory mediators and periodontal bacteria. This ultimately results in tooth loss and functional impairment in older adults.

Considering the significant influence which periodontitis has on dental and systemic health outcomes in the elderly population, it is crucial to implement comprehensive methods for prevention, early identification, and treatment. Personalized treatment plans, patient education, and routine dental exams are just a few of the customized oral health treatments that are necessary to address the special requirements and difficulties that come with periodontal disease in the elderly. Senior citizens' quality of life and overall wellbeing can be improved by healthcare providers by addressing modifiable risk factors and maintaining ideal periodontal health.

Globally, oral health has developed in a satisfactory manner, lending to better periodontal health. This has reflected in the decrease in severe forms of periodontitis and thus, tooth loss. Even so, periodontal health in aging individuals remains a primary focus for research. The goal is to ensure entire dentition with minimum loss of attachment and decreased functional limitation [3]. Locker invented concept of 'oral-health-related-quality of life' to measure the impact of poor oral health in overall disability [4]. It is also essential to note that periodontology concerns itself primarily with the interrelation between periodontitis and systemic diseases. This includes Alzheimer's disease, diabetes mellitus and atherosclerosis.

This review aims to further understand the interplay between periodontal health in geriatrics, the aging periodontium, their interplay, the pathology, clinical features and special care required in older patients.

Age related changes in periodontium:

Aging-related alterations to the periodontium, the tissues that support teeth, are significant and intricate. Age-related alterations in the periodontium are influenced by several factors:

Epithelium: Changes in the oral epithelium are associated with thinning of the epithelium and decreased keratinization and flattening of the rete-peg. Epithelial junction formation can be seen in age-related papillae. In addition, an increase in cell density was observed in the oral epithelium. Mitotic activity increases with age [5] [6].

Connective tissue: This demonstrates a steady change with age to denser, coarser tissue in older age groups. The number of cells decreases with age. The rate at which synthesized collagen matures changes with age. The periodontal ligament (PDL) tissue in younger people is well-organized and has a regular structure. As we age, the number of fibres and cells in the ligament reduces and its form becomes increasingly irregular [7] [8].

Cementum: As aging progresses, the cemental width increases, and grows in the region of the tooth apex. With the exception of the apex and furcation zones of teeth with multiple roots, cementum is cellular. With age, the cementum formation becomes completely acellular, cementum remodeling usually does not occur, resorption on the cementum surface locally with subsequent cementum apposition is mostly seen [9] [10].

Bone: As we age, collagen fiber addition to the bone diminishes and the periodontal areas of the alveoli are denticulated. It was discovered that there were more interstitial lamellae [11] [12]. **Wider periodontal ligament:** As people age, their remaining teeth are subjected to increased stresses due to increased tooth loss. This might account for the periodontal ligament's increasing width with ageing. Additionally, it has been shown that as one ages, biting force diminishes [13] [14].

Location of the junctional epithelium: In the intact periodontium, the junctional epithelium's apical end is located at cemento-enamel junction, immediately coronal to the connective tissue fiber attachment. However, in periodontal disease, the dento-gingival fibers break down resulting in apical movement of the junctional epithelium along the surface of the root. As one ages, the gingiva gradually declines physiologically and the epithelium migrates apically at the same time. The teeth's adaptation to the biting and the gum line's stable location leads to gum recession [7] [9].

Gingival Recession: People often experience a progressive receding of their gums as they age, revealing the tooth roots. You can become more susceptible to root caries as a result of this. One of the several reasons of gingival recession, or the exposure of the tooth roots due to gum tissue loss, is age-related changes. Several age-related changes lead to gingival recession, including: the gum tissue becoming thinner, The gums may recede as a result of a person's aging body progressively losing bone mass and density. The cumulative effects of periodontal disease include gum recession and weakening of the tissues supporting the teeth. Chronic periodontal disease can also

deteriorate with time. modified tissue response, which is the ability of our bodies to preserve and mend our tissues including our gums—can change. The gums may become more prone to recession as a result.

Inflammatory Response: The body's natural defence mechanism, inflammation is a natural reaction of bodily tissues to harmful stimuli. It involves immune cells, blood vessels, and chemical mediators. The inflammatory response has been shown to change with age. Immunity is a decline in the immune response associated with the natural aging process. Inflammation is a term used to describe an inflammatory response in the elderly. Altered cytokine production and immunological changes in leukocytes have been associated with increasing age. In geriatrics, periodontitis is one of the factors influencing the increase in chronic inflammatory response [15] [16].

Wound healing: Elderly people experience a delay in the healing of wounds. This is caused by a changed inflammatory response, which delays the infiltration of T cells into the wound site, changes in chemokine synthesis, and a decrease in the phagocytic ability of macrophages. Increased platelet aggregation, elevated inflammatory mediator release, decreased growth factor secretion, impaired macrophage function, delayed reepithelialization, delayed angiogenesis, delayed collagen deposition, and slower collagen turnover are some other variables. Elderly people exhibit a decreased wound strength as a result of all these changes [17] [18].

Risk factors in aging

In dentistry, prevention is crucial, particularly when it comes to patients' long-term treatment. Due to fluctuations in the immune response, regardless of pathogenic bacteria, an individual's vulnerability to periodontitis does not remain constant. There are a number of risk factors that are associated with periodontal disorders. They may be roughly divided into risk categories that are adjustable and non-modifiable. Factors that are effectively reducible or removed make up the first group. Nonmodifiable risk variables, on the contrary, are unchangeable and uncontrollable. Smoking, type 2 diabetes, low socioeconomic status, poor dental hygiene maintenance and, psychological stress, vitamin D insufficiency, and improper oral hygiene practises are modifiable risk factors that are closely linked to periodontitis. Risk factors that are adjustable are those that can be successfully addressed. Smoking is a significant risk factor for periodontal diseases. Diabetes type 2: Linked to delayed wound healing and increased vulnerability to infections.

Low Socioeconomic Status: Dental care and cleanliness may be less accessible.
Inadequate Maintenance of Dental Hygiene: Bad oral hygiene makes periodontitis worse.

Psychological Stress: Extended stress impairs the functioning of the immune system and dental health.
Vitamin D deficiency: Essential for healthy bones and the immune system.

Inadequate dental hygiene practices: Brushing, flossing, and regular exams are crucial.

The process of ageing itself is one of the unchangeable risk factors for periodontitis, along with genetic predisposition, dental anomalies myasthenia gravis, Alzheimer's disease and osteoporosis [19] [20].

Interaction of ageing and periodontal disease in inflammaging:

The physiological mechanisms that result in *functio-laesa* and the ensuing decline of bodily function that leads to mortality are all part of ageing. Genomic instability, telomere attrition, epigenetic changes, loss of proteostasis, deregulated nutrition-sensing, dysfunction of mitochondria, senescence of cells, stem cell fatigue, and influenced intercellular communication are among the nine hallmarks of ageing that result from these molecularly based processes. This process is followed at the systemic level by a persistent, gradual rise in the proinflammatory state, which aids in the development and aggravation of inflammatory diseases, also known as inflammaging. Systemic inflammation has been shown to aggravate vascular pathology, which in turn contributes to cardiovascular illnesses; it also raises cortisol production, which leads to insulin resistance and diabetes mellitus II; furthermore, it stimulates destruction of bones and neurodegenerative disorders. Periodontitis is the finest illustration of how this persistent systemic inflammation simultaneously raises the risk of other inflammatory illnesses and related syndemic functioning [21] [22].

Inflammatory osteoclastogenesis is the primary pathogenic process of periodontitis, and it is controlled via the nuclear factor kappa-B (nf-kB), a factor that regulates transcription which codes for the majority of the proinflammatory substances implicated in this process. Osteoprotegerin (OPG), receptor activator nuclear factor kappa ligand (RANKL), and receptor activator nuclear factor kappa (RANK) form a trio which control the process. In summary, RANKL appears on the outer surfaces of osteoblasts, fibroblasts, stromal cells, B cells, and T cells during inflammation. It also prefers to interact with sequences expressed on the osteoclast membrane and pre-osteoclasts, facilitating the effective maturation of pre-osteoclasts as well as increasing the activity of mature osteoclasts. Apart from bacterial lipopolysaccharide (LPS), proinflammatory cytokines (IL-1, IL-6, IL-8, and IL) and hormones such as parathyroid hormone, glucocorticoids, epinephrine and 17β -estradiol are important mediators that impact the rise in RANKL. (23). Since the most typical hallmarks of

immunosenescence include higher systemic levels of proinflammatory cytokines like TNF- α and PGE₂, phagocytosis, and elevated periodontal quantities of nf-kB itself, this model links immunosenescence to a detrimental effect of ageing. Considering this, it has been established that PGE-2, TNF- α , and basal levels of nf-kB are important regulators of RANKL-mediated osteoclastogenesis triggered by *Porphyromonas gingivalis* (*P. gingivalis*), *Treponema denticola*, and *Treponema socranskii*. This explains in detail where the severity of bone loss in elderly people originates [24]. Even with RANKL independent autoactivation of RANK, microbial challenge must exist.

The interplay between systemic disorders associated with ageing and periodontal inflammation:

Routine clinical practices must be appropriately modified to account for the uniqueness of systemic disorders and their treatment in aged individuals. Regarding the scientific evidence for a pathological link between periodontitis and systemic diseases associated with aging, oral bacterial overgrowth has been found to significantly contribute to atherosclerosis and impaired glycemic control in the context of insulin resistance and diabetes mellitus [25]. The fundamental cause of ischemic stroke and coronary artery disease is atherosclerosis, which is characterised by the buildup of plaque from atherosclerosis on the inner surfaces of arteries. This lowers blood flow and jeopardises the heart's and brain's nutrition [26]. Because hypoxia brought on by the blockage of feeding arteries causes infarction and ensuing destruction in the vascularized living zone of the afflicted arterial branch, thickness of associated with atherosclerosis is therefore a crucial element. The main causes of atherosclerotic plaque thickening include calcification, inflammatory responses in the artery wall, and plaque rupture, which opens up a channel for thrombosis. Given that periopathogens have been found in atherosclerotic plaque, periodontitis has been shown to have a direct impact on atherosclerosis. Although the mechanism is not yet fully defined, these periopathogens clearly contribute to local inflammation, atherosclerotic plaque instability and dysregulation of autophagy [26] [27]. Furthermore, a bidirectional pathological relationship between diabetes (DM) and periodontal disease has been established. In summary, diabetes mellitus (DM) impairs periodontitis by elevating vulnerability to infections and controlling local inflammation; in diabetic patients, DM increases medication responsiveness by elevating systemic inflammation; and in contrast, scaling and root planing enhances sugar balance [28] [29] [30] [31]. DM has been thought to worsen periodontal status by increasing levels of OPG, TNF- α , RANKL, IL-1 β as well as IL-6, while the interplay between advanced glycation end products (AGE) appears to promote extreme destruction and deterioration of tissues. Periodontal treatment of elderly patients aims to restore dental biofilms and related periodontal inflammations, deformations and/or

bone and bone tissues, if appropriate to enable functional tooth support and above all good oral hygiene. Overall, there is a large body of evidence showing that both non-surgical and surgical periodontal therapy is successful regardless of age. However, the scientific evidence on periodontal therapy results in the elderly is not overwhelming, as the effect of age was rarely taken into account in the data analysis [32] [33] [34].

The goal of periodontal treatment

The basic objective of periodontal therapy, regardless of age, is to stop periodontal diseases from getting worse in order to preserve functional dentition. The overall objectives of dental care may also influence the necessity for periodontal therapy.

Access to treatment: Regardless of the age of the patient, the patient must benefit from the treatment and experience no to very minimum damage. Consequently, it is critical that treatment planning be preceded by a thorough examination of all the variables that might impact the course as well as prognosis of the treatment. Reevaluation after periodontal treatment to identify the cause will determine the need for further treatment. Clinical symptoms such as residual periodontal pockets ≥ 6 mm and bleeding from pocket probing are generally used as criteria for further treatment (eg, surgical pocket therapy) [35] [36]. By eliminating plaque and promoting plaque control, surgical periodontal therapy aims to preserve the periodontium's long-term preservation. Restoration of periodontal support could be a further objective. Periodontal surgery is not contraindicated by age, and older persons recuperate from therapy in the same way that younger adults do [37]. Treatment of periodontitis is an important part of the treatment of patients with periodontitis.

Three treatment objectives of supportive periodontal therapy were outlined by Kerry in 1995 [38]:

1. Prevention of the advancement and recurrence of periodontitis in individuals who have had prior treatment;
2. Reduces the incidence of tooth loss;
3. Increases the probability of detecting and treating other oral diseases or conditions.

Conclusion:

People who are older are more likely to develop periodontal disease, which is a chronic inflammatory disorder that affects the tissues around the teeth. Numerous variables that aggravate periodontitis are exacerbated by aging. Firstly, the body's capacity to fight periodontal infections and restore damaged tissues is hampered by physiological changes brought on by aging, such as lowered immune function and compromised tissue healing systems. Furthermore, by further impairing immune function and bone density, age-related systemic diseases such as diabetes and

osteoporosis can aggravate periodontitis. furthermore smoking and other long-term lifestyle choices like poor dental hygiene greatly raise the rate and severity of periodontal disease in older persons. The elderly population is increasing every day while the quality of life is improving. Oral health is one of the biggest age-related problems. There is reasonable evidence to suspect that increasing age may be a potential risk factor for periodontal disease. Treatment of periodontal tissues is an integral part of this. Treatment options should be based on aging, dental and medical conditions, and long-term care of the elderly. Therefore, the treatment of periodontal problems becomes important. Furthermore, older people typically have severe periodontal damage and tooth loss as a result of the cumulative consequences of untreated or poorly managed periodontitis from earlier in life. As a result, older people who have untreated periodontitis are more vulnerable to systemic issues such as respiratory infections and cardiovascular disease. Therefore, minimizing the effects of aging on periodontal health and general well-being requires comprehensive oral health care catered to the specific needs of older persons. This treatment should include routine dental check-ups, careful oral hygiene habits, and management of systemic illnesses.

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