Aging & Periodontium

Bhadbhade S

Dr. D Y Patil Dental College and Hospital Pimpri Pune 411018, India.

Abstract

Geriatric Dentistry is a branch of dentistry which deals with treating and preventing oral diseases in older patients. Throughout the world, the numbers of older adults are increasing, and these individuals are retaining more teeth. Older people have limited regenerative abilities and are more prone to disease, syndromes, and sickness than other adults. This population has unique changes in their physiology as well as immunity that necessitate alterations in their treatment plans. They use more medications, many of which can cause problems such as xerostomia and decreased saliva. Thus, aging can significantly alter the host immune response and inflammatory reaction to bacterial plaque and in different ways than in younger population. In these older adults, education, nutrition, counseling, regular dental care, and compensatory home and professional care technique may be required. This review focuses on the change in the tissue that occurs with age and immunological alterations with age that contribute to the causation and perpetuation of periodontal disease.

*Corresponding Author:
Smruti bhadbhade,
Dr. D Y Patil Dental College and Hospital Pimpri Pune 411018, India.
Tel: 08390707904
E-mail: smrutibhadbhade@gmail.com

Introduction

Geriatric dentistry or geriodontics is the delivery of dental care to older adults involving the diagnosis, prevention, and treatment of problems associated with normal aging and age-related diseases as part of an interdisciplinary team with other health care professionals.

Last century has witnessed a number of remarkable demographic changes related to health, diseases, longevity and mortality of the population all over the world. By now one third of the world's elderly population is living in the developing countries and one out of twelve persons in the developing countries is over sixty five.

The rise in life expectancy is attributed primarily to the substantial reduction in mortality at different stages of life, which has been brought about by improved health care facilities, sanitation, environmental and public health reforms coupled with better hygiene and living conditions. As a result of the increasing life expectancy, the proportion of the elderly in the total population is projected to be around 20% in India and 32% in the developed nations by 2050.

The mouth is referred to as a mirror of overall health, reinforcing that oral health is an integral part of general health. In the elderly population poor oral health has been considered a risk factor for general health problems. On the other hand, older adults are more susceptible to oral conditions or diseases due to an increase in chronic conditions and physical/mental disabilities. Thus, old people form a distinct group in terms of provision of care. The physiology in general and oral tissues in particular change with age. This review aims to bring to light the various physiological changes that can be seen in the ageing population with special emphasis on the periodontium as well as the changes in immunity.

Changes in the Tissues of the Periodontium with Age

Gingival Epithelium

Thinning and decreased keratinization of the gingival epithelium have been reported with age [1]. The significance of these findings could mean an increase in epithelial permeability to bacterial antigens, a decreased resistance to functional trauma, or both, which might influence long-term periodontal outcomes. However, other studies have found no age-related differences in the gingival epithelium of humans or dogs [2]. Other reported changes with aging include the flattening of rete pegs and altered cell density.

The effect of aging on the location of the junctional epithelium has been the subject of much speculation. Some reports show migration of the junctional epithelium from its position in healthy individuals (i.e., on enamel) to a more apical position on the root surface with accompanying gingival recession [2]. However, in other animal studies, no apical migration has been noted [3]. With continuing gingival recession, the width of the attached gingiva

Bhadbhade S (2015) Aging & Periodontium. *Int J Dentistry Oral Sci*. 2(6), 79-83.

79

would be expected to decrease with age, but the opposite appears to be true [4]. Alternatively, the migration of the junctional epithelium to the root surface could be caused by the tooth erupting through the gingiva in an attempt to maintain occlusal contact with its opposing tooth (passive eruption) as a result of tooth surface loss from attrition. The consensus is that gingival recession is not an inevitable physiologic process of aging but is explained by cumulative effects of inflammation or trauma on the periodontium [2].

Gingival Connective Tissue

Increasing age results in coarser and denser gingival connective tissues [5]. Qualitative and quantitative changes to collagen have been reported. These include an increased rate of conversion of soluble to insoluble collagen, increased mechanical strength, and increased denaturing temperature. These results indicate increased collagen stabilization caused by changes in the macromolecular conformation. Not surprisingly, greater collagen content has been found in the gingiva of older animals despite a lower rate of collagen synthesis decreasing with age [2, 6].

Periodontal Ligament

Changes in the periodontal ligament that have been reported with aging include decreased numbers of fibroblasts and a more irregular structure, paralleling the changes in the gingival connective tissues [2, 7]. Other findings include decreased organic matrix production and epithelial cell rests and increased amounts of elastic fiber. Conflicting results have been reported for changes in the width of periodontal ligament in human and animal models. Although true variation might exist, this finding probably reflects the functional status of the teeth in the studies because the width of the space will decrease if the tooth is unopposed (hypofunction) or will increase with excessive occlusal loading [7]. Both scenarios might be anticipated as a result of tooth loss in this population. These effects also might explain the variability in studies reporting qualitative changes within the periodontal ligament.

Cementum

Some consensus regarding aging effects on cementum exists. An increase in cemental width is a common finding; this increase may be 5 to 10 times with increasing age. This finding is not surprising because deposition continues after tooth eruption. The increase in width is greater apically and lingually [8]. There is increase in the fluoride content in the cementum with age and decrease in its permeability. Although cementum has limited capacity for remodeling, an accumulation of resorption bays explains the finding of increasing surface irregularity.

Alveolar Bone

Reports of morphologic changes in alveolar bone mirror age-related changes in other bony sites. Specific to the periodontium are findings of a more irregular periodontal surface of bone and less regular insertion of collagen fibers. Although age is a risk factor for the bone mass reductions in osteoporosis, it is not causative and therefore should be distinguished from physiologic aging processes. Overriding the diverse observations of bony changes with age is the important finding that the healing rate of bone

in extraction sockets appears to be unaffected by increasing age [1]. Indeed, the success of osseointegrated dental implants, which relies on intact bone healing responses, does not appear to be age related. However, balancing this view is the recent observation that bone graft preparations (decalcified freeze-dried bone) from donors more than 50 years old possessed significantly less osteogenic potential than graft material from younger donors. The possible significance of this phenomenon on normal healing responses needs to be investigated.

Changes in Periodontium with Age

Intrinsic changes

In epithelium, a progenitor population of cells (stem cells), situated in the basal layer, provides new cells of the oral epithelium. These cells of the basal layer are least differentiated cells of oral epithelium [8].

"By definition, this differentiated cell, or epithelial cell, can no longer divide. On the other hand, the basal cell remains as a part of the progenitor population of the cells, ready to return to the mitotic cycle and again produce both types of cells. Thus there is a constant source of renewal". In the aging process, cell renewal takes place at a slower rate with fewer cells, so the effect is to slow down the regenerative processes. As the progenitor cells wear out and die, there are fewer and fewer of these cells to renew the dead ones. This effect is characteristic of the age related changes and biologic changes that occur with aging.

By the actions of gerontogenes or replicative senescence (Hayflick's limit and telomere shortening), the number of progenitor cells decreases. Hayflick, an American microbiologist, observed that fetal cells (i.e. Fibroblasts) displayed a consistently greater growth potential (Approximately 50 cumulative population doublings) than those derived from adult tissues (20-30 cumulative population doublings) [9]. As a result, the decreased cellular component has a concomitant effect to decrease cellular reserve and protein synthesis. This affects the oral epithelium in that the tissue becomes thin, with reduced keratinization.

Stochastic Changes

Stochastic changes occurring in the cells also affect tissue; for example, glycosylation and cross-linking produce morphologic changes and physiologic changes. Structures become stiffer, with loss of elasticity and increased mineralization. With a loss of regenerative power, structures become less soluble and more thermally stable. Somatic mutations lead to decreased protein synthesis and structurally altered proteins. Free radicals contribute to the accumulation of waste in the cell [8].

All these changes produce a decline in the physiologic processes of the tissues. Most changes are primarily a result of aging, although some are secondary to physiologic deterioration. For example loss of elasticity and increased resistance of the tissue may lead to decreased permeability, decreased nutrient flow, and the accumulation of waste in the cell. Thus, vascular peripheral resistance (decreased blood supply) may secondarily decrease cellular function.

Physiologic Changes

Bhadbhade S (2015) Aging & Periodontium. *Int J Dentistry Oral Sci*. 2(6), 79-83.

80

In the periodontal ligament, a decrease in the number of collagen fibers leads to a reduction or loss in the tissue elasticity. A decrease in vascularity results in decreased production of mucopolysacchrides [8].

All these types of changes are seen in the alveolar bone. With aging, the alveolar bone shows a decrease in bone density and an increase in bone resorption, a decrease in vascularity also occurs. In contrast, however, cementum shows cemental thickness.

Functional Changes

With aging, the cells of the oral epithelium and periodontal ligament have reduced mitotic activity, and all cells experience a reduction in metabolic rate. These changes also affect the immune system and affect healing in the periodontium. There is a reduction in healing capacity and rate. Inflammation, when present, develops more rapidly and more severely. Individuals are highly susceptible to viral infection and fungal infection because of abnormalities in T-cell function [8].

Clinical Changes

Compensatory changes occur as a result of aging or disease. These changes affect the tooth or periodontium that presents the clinical condition. Attrition is a compensatory change that acts as a stabilizer between loss of bony support and excessive leveraging from occlusal forces imposed on the teeth. In addition, a reduction in the overjet of the teeth is seen, manifesting as an edge-to-edge contact of the anterior teeth. Typically this is related to the approximal wear of the posterior teeth. An increase is seen in the food table area, with loss of "sluiceways" and in mesial migration [8].

Functional changes are associated with reduced efficiency of mastication. Although effectiveness of mastication may remain, efficiency is reduced because of missing teeth, loose teeth, poorly fitting prostheses, or non-compliance of the patient, who may refuse to wear prostheses appliance.

Aging and Periodontal Microbiota

Susceptibility to many diseases of microbial origin depends on the subject's age. Some diseases strike early in childhood, whereas others are seen mainly in adults and elderly people. Some diseases, mumps or rubella for instance, affect distinct organs at different periods of life and strike with variable intensity. *Candida* infection is mainly associated with the very young, the very old or very sick. Periodontitis is said to be a disease of the adult and epidemiologically correlated with age. Different forms of periodontitis are distinguished based on the patient's age at onset. However, there is an important difference between periodontitis and the other infectious diseases mentioned. While mumps and rubella heal spontaneously, periodontal disease may at best stop progressing further [10].

Although in many elderly patients periodontal disease may appear more severe than in the young, quite the opposite may be true in terms of disease progression. Although the amount of periodontal tissue destruction, measured as clinical attachment loss

may be greater in an 80- year-old individual than in an 18-year-old, the young patient may suffer from more severe disease and may lose more attachment in a short period. If left untreated, such an individual will lose the most heavily affected teeth early in life, whereas the more resistant ones will be maintained for a longer period. Thus, even if the involvement of residual teeth appears to be severe in an older subject, these teeth can be considered quite resilient and resistant: they have a history of withstanding periodontal disease for a prolonged period.

Since periodontal tissue damage accumulates over time, it should be possible to demonstrate shifts in the composition of the subgingival microbiota with increasing age [10]. Nevertheless, as these differences can be attributed largely to differences in ecological conditions prevailing in shallow or deep pockets, the specific role of particular microorganisms at different periods of life cannot be established using such a simple approach. Association studies must compare study populations matched for attachment level or pocket depth.

Ultimately it would be helpful to have information showing that - all other conditions being equal.

(i) Age modifies the composition of the subgingival microbiota.
(ii) Age modifies the risk of any given microbial composition for disease progression.

In recent years, much emphasis has been given to the possibility that periodontal disease may not be a continuous process but may be characterized by episodes of activity, followed by periods of relative quiescence of the disease. Consequently, epidemiological studies have focused on the identification of microorganisms associated with episodes of activity. Nevertheless, the true impact of short bursts of activity on the accumulated loss of periodontal tissues in elderly people remains to be determined and may be greatly overestimated. Goodson et al. [11] who introduced the concept of bursts of activity, not only documented phases of rapid loss of clinical attachment but also witnessed phases of remission with apparent regain of attachment. Bursts of activity did not inevitably lead to irreversible attachment loss. Slow continuous attachment loss, on the other hand, may have considerable consequences over the long run, although it is undetectable in studies limited to a few months' duration. Given the inaccuracy of periodontal probing, the detection of a continuous disease process leading to 6 mm of attachment loss over 60 years would require a minimal study period of 20 years (subsequent measurements must yield a difference of at least 2 mm in order to distinguish tissue destruction from measurement error with sufficient confidence). None of the longitudinal studies conducted with the purpose of risk assessment have covered such a long period. Thus, in addition to microorganisms recognized as important periodontal pathogens because of their association with active phases of disease, bacteria with low pathogenic potential may also contribute significantly to periodontal tissue destruction if present over protracted periods of apparent "health'. The impact of these organisms would probably increase with age and should be taken into consideration in any discussion of periodontal disease among older adults.

Periodontal Diseases in Older Adults

Etiology

Periodontal disease is age associated and not a consequence of aging. Periodontal disease in older adults is commonly referred to as chronic periodontitis [12]. Because periodontitis is a chronic disease, much of the ravages of the disease detected in older adults results from an accumulation of the disease over time. Research has shown that the advanced stages of periodontitis are less prevalent than the moderate stages in the older-adult population. One theory is that many sites of advanced periodontal disease have resulted in tooth loss earlier in life, suggesting that older age is not a risk factor for periodontal disease. Little evidence is available as to whether the risk factors for periodontal disease differ with age. General health status, immune status, diabetes, nutrition, smoking, genetics, medications, mental health status, salivary flow, functional deficits, or finances may possibly modify the relationship between periodontal disease and age [13]. Some medications that are frequently prescribed to older adults can alter the gingival tissues. Steroid-induced gingivitis has been associated with post-menopausal women on steroid therapy. Gingival overgrowth can be induced by certain medications such as cyclosporines, calcium channel blockers, and anticonvulsants (e.g., nifedipine or phenytoin) in the presence of poor oral hygiene. This gingival overgrowth further decreases a person's ability to maintain good oral hygiene."

Interactions between Periodontal Disease, Medical Diseases and Immunity in the Older Individual

Elderly people lose manual dexterity as a result of arthritis, injury, stroke, aging itself, etc., and accordingly, are likely to have poor oral hygiene [14]. Elderly people are unlikely to seek dental treatment but do receive medical treatments that result in the utilization of approximately 25% of the national total of prescription drugs. Medications such as antidepressants, antihistamines, antihypertensives and diuretics, which are often prescribed on a continuing basis, cause a reduction in salivary flow as a side effect [15]. Thus, many elderly people may be deficient in salivary flow (an intrinsic cleansing mechanism), and this combined with diminished oral hygiene practices (extrinsic cleansing mechanism), could lead to heavy plaque accumulation on their tooth and denture surfaces [16]. These are conditions that predispose to both dental caries and periodontal disease, and accordingly, one would anticipate an increased need for dental treatment among elderly people.

Dental disease in elderly people may also have medical consequences. Both dental caries and periodontal disease present a microbial challenge to the host that involves an immune response. However, these host-microbial outcomes may differ significantly from what is seen in younger individuals, because of a concomitant senescence of immunity that occurs with the aging process. This senescence may partially explain the role for poor dental health in the occurrence of aspiration pneumonia. For example, there is evidence that poor oral hygiene can lead to the emergence of periodontopathic anaerobes from within the plaque flora and to the selection and/or colonization of the gram-negative enteric bacilli in the oral flora, such as *Escherichia coli*, *Pseudomonas* species, *Proteus* species, *Klebsiella* species [17]. If these gram-negative enteric bacilli and periodontal anaerobes are present in any saliva (oropharyngeal secretions) that happens to be aspirated into the lung, a possible age-related sluggish immune response to this mixed inoculum could give rise to pneumonia [18]. The relationship between dental procedures and transient bacteremia are well documented [19] and must be considered whenever there is endocarditis or infections of prosthetic implants. There are now several reports that dental disease, especially periodontal disease, may contribute to cardiovascular disease [20]. The increase in the numbers of periodontal bacteria on the dentogingival surfaces could result in the penetration of bacteria and their byproducts into the gingival tissue, provoking an inflammatory response with production of inflammatory mediators, multiple antibody responses and a white blood cell response, among other reactions. As periodontopathic species are mostly gram-negative anaerobes [21], their penetration could expose the host to endotoxin, thereby initiating a variety of potentially harmful events that could predispose to cardiovascular disease. For example, endotoxin affects endothelial integrity, metabolism of plasma lipoprotein, blood coagulation and the function of platelets. The relationship between dental disease and cardiovascular disease could involve *Streptococcus sanguis,* an organism that has been extensively studied for its role in endocarditis, with one virulence factor being identified as a platelet aggregation-associated protein. If strains positive for platelet aggregation-associated protein were to gain increased access to the bloodstream, because of poor oral hygiene, then there exists the possibility that they could cause platelet aggregation and subsequent vascular pathoses [22].

If any of these associations with poor dental health can be shown to be causal, it may be possible to reduce the incidence of aspiration pneumonia and/or heart disease by simply promoting and establishing dental health. The implications of this in terms of quality of life, longevity and reduced medical costs are stunning. The treatment of these individuals will require a major paradigm shift for the dental profession, as they will need to change from a youth and caries-oriented treatment strategy to one based on the treatment of older individuals with a focus on periodontal disease.

Immunity in elderly people

A key consideration in the evaluation of the interplay between dental and medical disease is the role of host immunity in the moderation of the outcomes of these diseases. Quite different outcomes may be realized with the same dental conditions for the young versus elderly individual. Immune competence, which is necessary for successful resistance to the host of microbial insult, is compromised in elderly people. It has been suggested that immune senescence is a primary predisposing factor responsible for the increase in respiratory infection in elderly people. These compromises have been demonstrated in both systemic and mucosal immunity and appear to affect both cell-mediated and humoral immune functions [23]. Various changes in immunity associated with aging are as follows: Senescence of the immune system. The aging process includes the various compartments of the immune system and their interactions. For cell-mediated immunity, functional cell-mediated immunity rapidly diminishes with advanced age. Functional immunity must be distinguished from absolute cell numbers, since most studies do not reveal age-related changes in the absolute numbers of leukocytes (lymphocytes, monocytes, natural killer cells or granulocytes). T-lymphocyte functions, as measured by mitogen responsiveness, elaboration of cytokines (IL-21, delayed-type hypersensitivity and natural killer cell function have been shown to be reduced in elderly people (over 60 years of age) [24]. An interesting study by Marrie et al. demon-

Bhadbhade S (2015) Aging & Periodontium. *Int J Dentistry Oral Sci.* 2(6), 79-83.

82

strated a relationship between delayed-type hypersensitivity and functional independence of the subjects [23]. A consistent observation is also that the ratio of memory T cells to unstimulated T cells increases with increasing age. This suggests that immune memory is preserved but that the ability to respond to new antigenic challenge is reduced with increasing age.

A similar pattern emerges in the humoral immune response. Although the absolute numbers of B lymphocytes and total serum levels of IgG, IgM and IgA do not appear to change, there are significant changes in immune function, so that the ability to respond with antibody following antigenic challenge is reduced. In addition, the kinetics are slower, the peak response is lower and persistence of the antibodies in the serum is shorter in elderly people. These changes have been attributed to senescence of the T-helper function as well as more rapid turnover of B cells. Sims et al. have calculated an increase in susceptibility to pneumococcal infection of 1.33-fold for each decade after 50 years. This is associated with a significant reduction in the efficacy of immunization at each decade.

Few studies have examined the granulocytic arm of host defense. However, it has been reported that clonal proliferation of stem cells is reduced with age, placing considerable demands on the cellular reserves. The neutrophils of elderly people have impaired chemotactic reactivity, whereas phagocytosis and cytotoxicity appear to be relatively conserved.

Age-related changes in mucosal immunity have not been well studied. The existing data are somewhat confusing, since there is considerable contradictory information when animal and human studies are examined. Most of the human studies have focused on gastrointestinal mucosal immunity, and the majority agrees that it is compromised in advanced age.

Loesche et al. suggest that the major issues remaining to be resolved include [23]:

1. How aging impairs the secretory immune response.
2. Whether or not immunosenescence predisposes elderly people to infectious diseases; and
3. Whether immunodeficiency associated with aging might be reversible.

References

[1]. Fedele DJ, Niessen LC (2003) Carranza's Clinical Periodontology ,Periodontal Treatment of Older Adults. (IXth edn) ,WB Saunders. 551-557.
[2]. Berglundh T, Lindhe J, Sterrett JD (1991) Clinical and structural characteristics of periodontal tissues in young and old dogs. J Clin Periodontol 18(8): 616-623.
[3]. Lindhe J, Hamp SE, Loe H (1975) Plaque induced periodontal disease in beagle dogs. A 4-year clinical, roentgenographical and histometrical study. J Perio Res 10: 243.
[4]. Ainamo A, Ainamo J, Poikkeus R. (1981) Continuous widening of the band of attached gingiva from 23 to 65 years of age. J Periodontal Res 16(6): 595-599.
[5]. Wentz FM, Maier AW, Orban B (1952) Age changes and sex differences in the clinically "normal" gingiva. J Periodontol 23: 13.
[6]. Richard P Ellen (1998) Periodontal disease among older adults: what is the issue? Periodontol 2000 16(1): 7-8.
[7]. Michael G Newman, Henry H Takei, Fermin A Carranza (2006) Carranza's Clinical Periodontology, Periodontal Treatment of Older Adults. (Xth edn), Saunder's Publications. 675-691.
[8]. Ronald Klatz ,Robert Goldman (1997) Theories of Aging. Keats Publishing, New Canaan,CT ©.
[9]. Van der Velden U (1984) Effect of age on the periodontium. J Clin Periodontol 11(5): 281-294.
[10]. Mombelli A (1998) Aging and the periodontal and peri-implant microbiota. Periodontol 2000 16: 44-52.
[11]. Goodson JM, Tanner AC, Haffajee AD, Sornberger GC, Socransky SS (1982) Patterns of progression and regression of advanced destructive periodontal disease. J Clin Periodontol 9(6): 472-481.
[12]. Brown LJ, Brunelle JA, Kingman A (1996) Periodontal status in the United States, 1988-1991: Prevalence, extent, and demographic variation. J Dent Res 75: 672-683.
[13]. Beck JD (1996) Periodontal implications: Older adults. Ann Periodontol 1(1): 322-357.
[14]. Kiyak HA (1986) Explaining patterns of dental service utilization among the elderly. J Dent Educ 50: 679-687.
[15]. Loesche WJ, Lopatin DE (1998) Interaction between periodontal disease, medical disease and immunity in the older individual. Periodontol 2000 16: 80-105.
[16]. Sreebny LM, Schwartz SS (1986) A reference guide to drugs and dry mouth. Gerodontology 5(2): 75-99.
[17]. Gibbons RJ, Etherden I (1986) Fibronectin-degrading enzymes in saliva and their relationship to oral cleanliness J Periodont Res 21: 386-395.
[18]. Scannapieco FA, Mylotte JM (1996) Relationships between periodontal disease and bacterial pneumonia. J Periodontol 67: 1114-1122.
[19]. Silver JG, Martin AW, McBride BC (1977) Experimental transient bacteremias in human subjects with varying degrees of plaque accumulation and gingival inflammation. J Clin Periodontol 4(2): 92-99.
[20]. Beck JD, Garcia R, Heiss G, Vokonas PS, Offenbacher S (1996) Periodontal disease and cardiovascular disease. J Periodontol 67: 1123-1137.
[21]. Haffajee AD, Socransky SS (1994) Microbial etiologic agents of destructive periodontal disease. Periodonto1 2000 5: 78-111.
[22]. Herzberg MC, Meyer MW (1996) Effects of oral flora on platelets: possible consequences in cardiovascular disease. J Periodontol 67(10): 1138-1142.
[23]. Loesche WJ, Lopatin DE. (1998) Interactions between periodontal disease, medical diseases and immunity in the older individual. Periodontology 2000 16(1): 80-105.
[24]. Page RC (1991) The role of inflammatory mediators in the pathogenesis of periodontal disease. J Periodont Res 26(3): 230-242.

Bhadbhade S (2015) Aging & Periodontium. *Int J Dentistry Oral Sci.* 2(6), 79-83.

83