Geriatric Dentistry-An overview

Syeda Arshiya Ara[1,*], Humera Ayesha[2], Uzma Roohi[3], Shilpa Shastri[4], Priyanka A[5]

[1]Professor, [2,3,4,5]PG Student, Dept. of Oral Medicine & Radiology, Al-Badar Rural Dental College & Hospital, Gulbarga

***Corresponding Author:**
Email: drarshiyazaka@gmail.com

Abstract

Geriatric dentistry deals with the delivery of oral health care to elderly subjects which includes diagnosis, prevention and treatment of problems associated with physiologic and pathological age-related diseases.as a part of an interdisciplinary team with other oral health care professionals.

Keywords: Geriatric dentistry, Oral health care, Oral mucosal diseases.

Introduction

The term aging is defined in chronologic, physical, and social terms, indicating changes in an individual's participation within their sociocultural context or physical or functional capabilities.(1) The aging population can be categorized into three broad functional groups: (1) functionally independent older adults or those who are physically well despite advanced age, (2) frail older adults or those at high risk for major adverse outcomes, and (3) functionally dependent older adults or those who have experienced deterioration of physical capacities and must rely on assistance from others.(2)

Following termonologies was given by Douglass(1)

Old age: is the terms which reflect social constructs of chronologic age indicating changes in an individual's participation in their sociocultural context or physical or functional capabilities.

Frail elderly: the term refers to those older individuals with physical and functional impairments influencing daily living activities.

Vulnerable older adults: Older adults at risk of morbidity, frailty, and mortality within 2 years, identified by risk factors such as age, and physical and functional status and self-reported health status.

Oldest old: refers to the upper age groups in a given population. The age limit that identifies this group ranges from 75 to 90 years.

Successful aging: refers to decline in mental and physical function with chronologic age.

Geriatric dentistry programs had been well adopted in the USA since the early 1970s.(3) There is a better development of geriatric oral health care in the developed nations like USA, Australia, Canada, UK, and the European nations compared to developing countries.(4) The geriatric dentistry has not received attention by dental professionals. In developing countries, elderly population accounts for about 1/6th of the total world population including those of Southeast Asia.(5)

In India, with the population of over one billion people, elderly groups of more than 60 years constitute 7.6% of the total population, which amounts to 76 million. Prevalence of oral cancer is highest in the elderly subjects among Indians.(6) There had been a rapidly increased "65 plus" population in India in the last decade.(7)

Pharmacokinetics: The pharmacokinetics of medications—the absorption, distribution, metabolism, and elimination—can be altered in an older patient due to the aging process. The absorption of medications is least affected by the aging process. Most drugs are absorbed passively, simply by being in the stomach or intestine. If absorption is affected in older adults, it is usually decreased either due to increased amount of acid in the stomach or due to decreased movement of the muscles of the digestive system or even decreased surface area for absorption.(8)

Pharmacodynamics: Medications that are lipophilic may have prolonged effects due to increased amount of body fat. Drugs that are hydrophilic may have a more rapid increase in concentrations in the blood because of less water. These changes often necessitate the lower dosing of medication.(9)

Systemic changes associated with oral health

Cardiovascular: There are both structural and functional cardiovascular changes with age. There will be an overall decreased cardiovascular reserve due to hypertrophy of and loss of myocytes. Around 90% of pacemaker cells in the sinus node are lost by the age of 75 years,(10) resulting in maximum heart rates and slower resting. As described by Cefalu,(10) normal aging increases the stiffness of the left ventricle and decrease in left ventricular compliance. Arterial stiffness as a result of age-related calcification with collagen deposition in place of elastin. The vasodilatory effects of decreased nitrous oxide lead to an increase in systolic vascular resistance impedes blood flow. This increases myocardial oxygen demand and cardiac work.

In addition to the normative changes in left ventricular function the aging heart experiences lower ability to raise the heart rate which results in muted responses to cholinergic and sympathomimetic stimulation. Thus there will be an increased risk of congestive heart failure and heart block in the presence of chronic diseases such as diabetes, hypertension, and coronary heart disease. The cardiovascular system compensates to maintain function, but finds it difficult to adapt to stress or a medical intervention including dental appointments.[11]

Pulmonary: The respiratory system shows structural changes with aging which includes: rib cage stiffening; reduced intercostal muscle strength, early fatigue of diaphragm; decreased chemoreceptive response, alterations in connective tissue; narrow airways and shallower alveolar cells and sacs; and reduced vital capacity and forced expiratory volume, with rise in functional residual capacity and residual volume.[10] All these physiologic changes lower the threshold for adaptive ability, increases the risk of disease. Decreased cough reflex and deficient mucus clearance directly affect oral health with increased risk of aspiration during treatment and through plaque accumulation on teeth and dentures.[11] Also, decreased genioglossal reflex and an elongated soft palate in geriatrics causes an increased risk for obstructive sleep apnea (OSA) and hypertension.

Musculoskeletal: There is an altered architectural strength of the bone matrices with decreased bone mineralization with the normal aging process of the musculoskeletal system. There is a decline in water content in the ligaments, tendons, cartilage, and synovial compartment resulting in joints stiffen and microfractures.[10] There is an overall fat deposition with aging and conversely decreased total water content and muscle mass. This process accelerates the distribution of water-soluble drug like acetaminophen, resulting in precipitation of the drug. The distribution of lipid-soluble drugs decreases, and drugs such as lidocaine and diazepam have a longer half-life owing to their distribution throughout adipose tissue.[12]

Age-related functional changes include a decrease in hand-grip strength in the lower extremities.[10] In frailer adults, this affects tooth brushing efficiency.

Liver: It is reported that with the normal aging there is an overall reduction in the size of the liver, blood flow to the liver and hepatic metabolic activity resulting in reduced hepatic metabolic activity reducing liver function with age.[13] These changes alter drug metabolism in hepatic circulation and should be taken strictly under consideration when prescribing medications.

Kidney: The normal aging kidney shows physiologic as well structural. This may include a decrease in glomerular filtration rate and renal blood flow. The GFR declines by the age of 40 years. In older adults the renal excretion of medications takes longer.[10]

Thus the drugs prescribed by dentists for which changes in dosage are important are penicillins (seizures, cognitive dysfunction), fluoroquinolones (phototoxicity, hallucinations, delusions, seizures, cognitive dysfunction), fluconazole, and aminoglycoside antibiotics.[10]

Due to a diminished physiologic reserve of liver and kidneys associated with the aging process and comorbidities, the body's ability to respond to external stress decreases. To reduce potential external stress and thus consideration in appointment time, duration, when treating elderly subjects.[14]

Age related oral changes

Dentition: Occlusion attrition, pulpal recession, fibrosis, and decreased cellularity are some of the more common changes seen— all of which may lead to diminished tooth sensitivity and reduced perception of painful stimuli. In addition, staining, chipping and cracking, and increased susceptibility to tooth fracture is common in older patients.[15]

Enamel: Physical appearance and molecular composition are altered in healthy aging adults. A study in 2008 found that there was an increase in elastic modulus and hardness of the outer layer of enamel,[16] resulting in more brittle and less permeable resulting in cracks and microfractures in tooth enamel in aging teeth.[17]

Dentin: There is a formation of both physiologic, reparative dentin and sclerosis of dentinal tubules with age resulting in decreased sensation to cold, hot, sweet, and pain. Unill the age of 80 years, there is a possibility of most of the dentinal tubules to be fully occluded.[17] resulting in reduced fracture toughness.[18]

Pulp: The is an increases in pulp response time with age due to more number of collagen fibers and calcification reduced blood supply and degenerating nerve fibers.[19] The size of root canal decreases with age, narrowing of the pulpal chamber due to secondary and tertiary dentin formation, increased connective tissue and decreased innervation and vascularization.[20]

Cementum: The cementum increases in thickness 3-fold till the age of 75 years, with the thickest layer at apex and varying degrees of thickness along the root depending on recession and wear of the root surface.[17]

There is decreased sensitivity to thermal conduction. Fewer older adults report tooth pain attributable to molecular changes in enamel, dentin, pulpal tissue, and cementum.[21]

Periodontal Apparatus: It is thought that minimal loss of alveolar bone and the periodontal attachment apparatus is common in older adults, not as a consequence of aging but of the disease process.

Epidemiologic studies have shown a higher prevalence of periodontal disease in older adults. This is attributed to cumulative tissue destruction.[21]

Oral Mucosa: The oral mucosa shows a diminished keratinisation[22] and thinning of the epithelial structure with aging,[23] however, there is no evidence of change in the morphology of the human oral epithelial cells as a result of aging.[24]

Age-related changes of the salivary glands: The decrease in salivary flow has been associated with aging.[25] Studies have reported the loss of approximately 40% of acinar cells with aging.[26,27] Similar morphologic changes have been reported in the parotid gland[28] and labial salivary glands.[29] Clinically, a reduction in saliva resulting in xerostomia with difficulty in chewing, should not be considered a consequence of aging.[30] Few medications may directly affect salivary gland output.[31] Radiation and chemotherapy may also result in salivary impairment. The acinar cells of the salivary glands are destroyed by ionizing radiation used in the management of head and neck cancers.[30] Autoimmune disease also impairs salivary function for example Sjogren's syndrome either affects the salivary and lacrimal glands alone or may be associated with a rheumatoid disease.[32]

Root caries: Root caries occur as a result of increased gingival recession, salivary gland dysfunction, less effective oral hygiene, and diminished oral motor function. These changes reduce the ability to withstand insults from caries.[15] Management includes 10% carbamide peroxide gel delivered in custom-fitted trays for caries prevention in patients with compromised oral hygiene. Plaque is suppressed, and therefore, caries is controlled due to increase in salivary and plaque pH.[15]

Periodontal disease in the elderly population: The individuals with the periodontal disease increase in the elderly population.[33] This is also associated with an increase in loss of attachment and gingival recession[34] due to reduced oral cleanliness with aging.

Other conditions

Oral candidiasis: The most common forms found in the elderly subjects include acute pseudomembranous and chronic atrophic. The pseudomembranous form appears as a curdlike raised lesion associated with burning mouth. Tongue and pharynx are the commonest sites although it may occur at any site. The lesions are scrapable and a definitive diagnosis made microscopically by the presence of yeast and hyphae.

Angular cheilitis a form of chronic atrophic candidiasis occurs on the commissures of the lips. This occurs due to pooling of saliva around the corners of the mouth and appears to be an erythematous lesion.

MacDonald DE et al[35] suggests the following line of treatment for oral candidiasis consisting of using oral antifungal agents. Nystatin oral suspension (100,000 U/mL) to be rinsed four times daily after meals and at bedtime. Nystatin in powder form (1 billion units) can be used by mixing a 1/8th teaspoon into half a cup of water and allow to swish for 1 minute. For denture stomatitis and angular cheilitis, application of a nystatin ointment or cream (100,000 U/g), applied to the inner denture or the corner of the mouth four times daily for a 2-week period. For a systemic approach, ketoconazole, 200 mg, can be given once a day over a 2-week period. Fluconazole, 100 mg, is a good alternative because of the one dosage per day regiment, namely two tablets on the first day followed by one tablet for the remaining 13 days. It is recommended that the patient's denture be soaked in a solution of half a cup of 0.12% chlorhexidine gluconate to water or 1 teaspoon sodium hypochlorite in one cup of water during the treatment phase.[35]

Xerostomia: Saliva helps the digestive process by through lubrication during the masticatory process. It maintains oral pH, calcium and phosphate levels. Reduction in salivary flow referred to as "xerostomia", which occurs in various conditions such as certain medications which are the most common cause, autoimmune disorders, glandular blockage, alcoholism, Parkinson's disease and radiotherapy of the salivary glands.[36,37] The drug-induced xerostomia is reversible whereas xerostomia secondary to radiotherapy is irreversible.[35]

Oral Lichen Planus: Lichen planus an autoimmune condition characterized by ulcerative region circumscribed by a lacelike white pattern. Its primary site is tongue, gingiva, and cheek mucosa and may be initiated by stress, drug allergy, infection, or genetic predisposition.[38] It is painful and episodic. MacDonald DE et al[35] suggest the following line of treatment which includes clobetasol propionate ointment 0.05%, fluocinonide gel 0.05%, triamcinolone acetonide in gel base 0.1% topical medications.[39] These are applied to the lesions four times daily. The lesions not responding to oral applications, systemic steroids are administered.[40] Oral cancers incidence are more with erosive lichen planus.[41]

Hyperkeratotic lesions: Hyperkeratotic lesions as a result of chronic trauma commonly on the lateral border of the tongue referred to as "leukoplakia". The lesion should be biopsied to rule out the dysplastic changes. A simple screening method uses are computer-assisted brush biopsy technique (OralCDx, CDx Laboratories, Suffern, New York) that is very effective in screening unexplained red, white, mixed, ulcerative, or hyperkeratotic lesions for the possibility of cancer.[42] It also serves as an adjunct to the oral examination and to rule out the need for biopsy.

Lesions of dental prosthetic origin: Episodic mechanical trauma due to ill-lfitting dentures results in an overgrowth of gingival connective tissue evident along the borders of the prosthesis. Treatment includes adjusting the prosthesis or replace it with a new one. For more severe overgrowth it is recommended to surgically excise.[35]

Painful, burning mouth: The painful burning sensation of the oral cavity and tongue occurs most commonly as a result of infection from the candidal organisms.[43] It may also occur due to metabolic disease like diabetes mellitus, nutritional deficiencies including vitamin B and iron deficiencies and other causes include xerostomia, non-functional oral habits, allergy and menopause.[44] MacDonald DE et al[35] suggest that the treatment depends on the cause treatments include antifungal medication for Candida infection. Drug-induced xerostomia can be treated by methods of stimulating to increase the salivary flow and use of salivary substitute would be of help. Also, vitamin or iron supplementation may be indicated.[35]

Conclusion

The present article aimed to review the age related systemic changes and age related oral changes and also the common oral mucosal lesions seen in geriatric subjects. In a vast developing country like India, there is a need to establish and expand geriatric oral health care. It should be considered as a separate branch of dentistry.

References

1. Douglas CW, Jiméne MC. Our Current Geriatric Population Demographic and Oral Health Care Utilization.
2. Gallagher JC. Vitamin D and aging. *Endocrinol Metab Clin North Am*. 2013;42(2):319–332.
3. Preshaw PM, Mohammad AR. Geriatric dentistry education in European dental Schools. Eur J Dent Educ 2005;9:73–77.
4. Shetty S. The Need for Geriatric Dental Education. J Indian Prosthodont Soc. 2014;14(1):1–2.
5. World Health Organization Working Panel on Professional Training (1972). The sociology of professional training and health. manpower: summary report. World Health Organization, Geneva.
6. Panchbhai AS. Oral health care needs in the dependant elderly in India. Indian J Palliat Care 2012;18(1):19-26.
7. Vissink A, Spijkervet FK, Amerongen VA. Aging and saliva: A review of the literature. Spec Care Dentist.1996;16(3):95-103.
8. Stein PS, Aalboe JA, Savage MW, Scott AM. Strategies for communicating with older dental patients. *J Am Dent Assoc*. 2014;145(2):159–164.
9. Roett MA, Coleman MT. Practice improvement, part II: health literacy. *FP Essent*. 2013;414:19–24.
10. Cefalu C. Theories and mechanisms of aging. Clin Geriatr Med.2011;27:491–506.
11. Bergman SA, Coletti D. Perioperative management of the geriatric patient. Part II: cardiovascular system. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102:e7–12.
12. Dental considerations for geriatric patients. Available at: http://www.netce.com/ 839/Course_3956.pdf.
13. Zeeh J, Platt D. The aging liver: structural and functional changes and their consequences for drug treatment in old age. Gerontology.2002;48:121–7.
14. Abrams AP, Thompson LA. Physiology of Aging of Older Adults. Systemic and Oral Health Considerations. Dent Clin N Am. 2014. Available at: http://dx.doi.org/10.1016/j.cden.2014.06.002.
15. Greenberg, Glick M, Ship. Burket's Oral Medicine. 12th ed. Peoples medical publishing house USA;2015.p.653-667.
16. Park S, Wang DH, Zhang D, et al. Mechanical properties of human enamel as a function of age and location in the tooth. J Mater Sci Mater Med.2008;19:2317–24.
17. An G. Normal aging of teeth. Geriatr Aging.2009;12(10):513–7.
18. Lund AE. Dentin grows more brittle with age. J Am Dent Assoc 2009;140(12):1472.
19. Farac RV, Morgental RD, de Pontes Lima RK, et al. Pulp sensibility test in elderly patients. Gerodontology 2012;29:135–9.
20. Goodis HE, Rossall JC, Kahn AJ. Endodontic status in older U.S. adults. J Am Dent Assoc 2001;132:1525–30.
21. Timmerman MF, van der Weijden GA. Risk factors for periodontitis. Int J Dent Hyg 2006;4(1):2–7.
22. Shklar G. The effects of aging upon oral mucosa. J Invest Dermatol 1966;47:115– 20.
23. Lfe H, Karring T. The three dimensional morphology of the epithelium connective tissue interface of the gingiva as related to age and sex. Scand J Dent Res.1971;79:315–26.
24. Plewig G. Regional differences of cell sizes in the human stratum cornium II. Effects of sex and age. J Invest Dermatol.1970;54:19–23.
25. Langer A. Oral signs of aging and their clinical significance. Geriatrics.1976;31:63–9.
26. Waterhouse JP, Chisholm DM, Winter RB, et al. Replacement of functional parenchymal cells by fat and connective tissue in human salivary glands: an age-related change. J Oral Pathol 1973;2:16–27.
27. Scott J. Degenerative changes in the histology of the human submandibular gland occurring with age. J Biol Buccale 1977;5:311–9.
28. Scott J, Flower EA, Burns J. A quantitative study of histological changes in the human parotid gland occurring with adult age. J Oral Pathol 1987;16:505–10.
29. Drummond JR, Chisholm DM. A qualitative and quantitative study of the aging human labial salivary glands. Arch Oral Biol 1984;29:151–5.
30. Wu AJ, Ship JA. A characterization of major salivary gland flow rates in the presence of medications and systemic diseases. Oral Surg Oral Med Oral Pathol 1993;76: 301-6.
31. Silverman Jr S. Precancerous lesions and oral cancer in the elderly. Clin Geriatr Med 1992;8:529– 41.
32. Talal N. Overview of Sjogren's syndrome. J Dent Res 1987;66:672– 4.
33. Albandar JM. Epidemiology and risk factors of periodontal diseases. Dent Clin North Am 2005;49:517–32.
34. Johnson ES, Kelly JE, Van Kirk LE. Selected dental findings in adults by age, race, and sex, United States, 1960–1962. Vital Health Stat 11 1965;11:1–35.
35. MacDonald DE. Principles of Geriatric Dentistry and Their Application to the Older Adult with a Physical Disability Clin Geriatr Med.2006;22:413–434.
36. Navazesh M, Brightman VJ, Pogoda JM. Relationship of medical status, medications, and salivary flow rates in adults of different ages. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:172–6.
37. Bergdahl M, Bergdahl J. Low unstimulated salivary flow and subjective oral dryness: association with medication, anxiety, depression, and stress. J Dent Res 2000;79:1652–8.
38. Regezi JA, Sciubba JJ. Oral pathology: Clinical Pathologic Correlations. Philadelphia: Saunders; 1999.

39. Gibson G, Niessen LC. Aging and the oral cavity. In: Cassel CK, Leipzig RM, Cohen HJ, et al, editors. Geriatric medicine, an evidence-based approach. New York7 Springer; 2003.p. 901–17.
40. Chainani-Wu N, Silverman Jr S, Lozada-Nur F, et al. Oral lichen planus: patient profile, disease progression and treatment responses. J Am Dent Assoc 2001;132:901–9.
41. Eisen D. The clinical features, malignant potential, and systemic associations of oral lichen planus: a study of 723 patients. J Am Acad Dermatol 2002;46:207–14.
42. Sciubba JJ. Improving detection of precancerous and cancerous oral lesions. J Am Dent Assoc.1999;130:1445–57.
43. Terai H, Shimahara M. Atropic tongue associated with Candida. J Oral Pathol Med 2005;34:397–400.
44. Pinto A, Stoopler ET, DeRossi SS, et al. Burning mouth syndrome: a guide for the general practioner. Gen Dent 2003;51:458–61.
45. Scala A, Checci L, Montevecchi M, et al. Update on burning mouthy syndrome: overview and patient management. Crit Rev Oral Biol Med 2003;14:275–91.