ORAL SOFT TISSUE CHANGES IN GERIATRIC PATIENTS*

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As an individual ages, many changes occur in both physical appearance and metabolic processes, and the entire organism has a finite length of life. It is thought that this process reflects the inability of individual cells to replicate themselves beyond a certain point as well as diminished metabolic cellular activity. For instance, in tissue culture, normal cells (as opposed to malignant cells) seem to have a maximum ability to replicate approximately 50 times, depending on the age of the organism from which the cells were taken. The exact cause of this phenomenon is one of the great mysteries of cell biology today. Structural and functional changes specific to age are closely related to impaired cell regeneration and metabolic cellular activity, which affect the homeostatic condition of the whole organism.[1]

Oral tissues react in a fashion similar to other organs with respect to aging, and this paper will review some of these changes with respect to the soft tissues and underlying bone.

SALIVARY GLANDS

The "dry mouth syndrome" is one of the most prevalent complaints after age 65. This is one of the contributing factors to the increase in dental caries, inability to wear dentures, burning sensation in the mouth, and decreased sensitivity of the taste buds of many individuals in this age group. The cause of this problem is very simply diminution of salivary flow caused by acinar destruction and hyalinization, adhesions and obstructions with atrophy of salivary ducts, and infection or disease within the stoma of the glands themselves.[2] Not every individual develops this condition, and many aged patients have adequate salivary flow well into

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the 80s or even beyond. Another cause of decreased flow is Sjogren's syndrome.

Sjogren's syndrome consists of Keratoconjunctivitis sicca, Buccopharyngolarynitis sicca, swelling of various salivary glands, xerostomia and polyarthritis, usually of the rheumatoid type. Middle-aged and older women are affected most frequently by this syndrome. Not every patient with this disease necessarily manifests the complete series of signs and symptoms.

Although swelling of the major salivary glands is often described as a characteristic of Sjogren's syndrome, atrophy and fibrous replacement have greater pathognomonic significance. It is possible that the salivary gland swelling is an early inflammatory phenomenon which eventually leads to atrophy. The etiology is unknown. Some findings suggest antecedent infection, but the predilection of the disease for elderly women favors an endocrine basis. Shafer and Muhler have shown a definite relationship between the endocrine and salivary glands in experimental animals, and this lends interest to this theory.[16]

Chronic sialadenitis can destroy gland acini and decrease saliva production. A variety of organisms have been implicated in this condition.

Tumors of the salivary glands are not infrequent and may cause the "dry mouth syndrome," either due to physical destruction of healthy stroma or removal of the gland itself. In the aged, the problem is one of the consequences of the tumor, rather than the tumor itself, because the incidence of salivary gland tumor mirrors those of the oral cavity in general, with peak occurence between ages 40 and 60 and rapid decrease in incidence per 100,000 thereafter. One should not omit diabetes mellitus as a cause of xerostomia and every effort should be made to rule it out or to control it if present.

Mucous Membranes

Changes in the oral mucosa are most apparent after age 70, but many individuals have some changes earlier in life. The tissue appears satiny and shiny with a stretched appearance. This is usually accompanied by a loss of resilience and elasticity, a friable surface and edema in those areas where tight union to the underlying bone is lost.[3]

Occasionally, extensive keratinization or parakeratosis may occur, but just as frequently thinning epithelium and loss of keratinization are evi-

dent. If the epithelium thins, the tissue is more prone to injury and such individuals tend to avoid hard foods and subsequently all too often have a protein deficiency, allowing still further injury.[4] If one examines the epithelium microscopically, there is thinning of the tunica propria, loss of elastic fibers, and blunting of the rete pegs. A decrease in the number of capillaries contributes to further impairment of the capacity to regenerate. Mucosal, alveolar, and gingival arteries show the changes of arteriosclerosis. Collagenous elements are thicker than usual and arranged in dense parallel bundles similar in architecture to such regular dense connective tissues as tendon. Fibroblasts tend to be rounded, and occasionally are surrounded by a lacunar space. There is a decrease in the ratio of ground substance to collagen, a lower water content, and a decrease in hyaluronic acid. All of these changes cause the loss of resilience and subsequently are subject to the effects of inflammatory, degenerative, and other pathologic processes.[5]

The condition known as "denture sore mouth" is caused by a combination of the mucosal changes mentioned above, as well as the immediate effect of mechanical trauma from ill-fitting dentures and, usually, a superimposed monilial infection. In this condition, the palate appears erythematous, edematous, friable, and spongy. If the dentures are removed, symptoms usually clear on their own after several days. Denture hyperplasia or epulis fissuratum is another mucosal condition caused by ill-fitting dentures. This is simply a heaping up of mucosal tissue, and frequently has extensive ulcerations, but with little malignant potential.

The corners of the mouth are frequently red, ulcerated, and prone to bacterial infection in a condition known as "angular cheilitis," observed most frequently in denture-wearers, but occasionally in individuals who still have their natural teeth. The prime cause of this is saliva pooling at the lip commisures due to a weakness in the musculature in this area. Contributing causes are a loss of vertical height in the face (either with dentures or natural dentition), iron deficiency, and riboflavin deficiency.

A not uncommon mucosal problem in the aged is lichen planus, and most of those with dermal lesions have oral lesions as well. On the oral mucosa, the clinical picture is classically characterized by lesions consisting of a series of radiating white or gray, velvety, threadlike lines crisscrossing in a reticular pattern, most commonly seen bilaterally on the buccal mucosa and known as Wickham's striae. Coalescence of the lines may form white plaques. Sites other than the buccal mucosa (in decreas-

ing order of occurrence) are the tongue, lips, gingivae, and the floor of the mouth. Vesicle or bulla formation may also occur in the erosive variety.

Atropic lichen planus is a not infrequently encountered mucosal lesion among the elderly and shows large shallow erosions covered with yellow fibrin sloughs and surrounded by connecting white striae with a lacy appearance. Occasionally, areas of repair are intermixed with the ulcerations. Many researchers feel that the continuous process of ulceration and repair of an atropic mucosa may be an important cause of precancerous lesions. Lichen planus may be painless, but when complicated by ulcerations may be quite painful.

Benign mucosal membrane pemphigoid is another condition not infrequently seen in the mouth. This is a subepithelial bullous eruption with a predilection for mucosal membranes. Ocular involvement may also occur. The disease runs a chronic benign course, but scarring may occur. All mucous membranes may be involved; the affected mucosa is erythematous with areas of occasional vesicles and bullae, and the bullae may burst and be replaced by necrotic epithelium. The final diagnosis is only accomplished by biopsy.

As shown in studies by Makila,[6] most soft-tissue changes are benign. A partial list in decreasing order of frequency is as follows: fissured tongue, varicosities on ventral surface of tongue, coated tongue, atrophy of buccal mucosa, atropic tongue mucosa, angular cheilitis, hyperkeratotic lesion, ulcerations, benign tumors, and bullous or vesicular lesions.

Most oral carcinomas occur after age 40 and peak in their incidence between ages 60 and 70 with a true decline after age 70. Four percent of all malignant tumors are oral, and 90% of all oral malignancies are squamous cell carcinomas. The sites or oral carcinoma in decreasing frequency are: lower lip, tongue, gingivae, and floor of the mouth.[7] Leukoplakia is defined as a white patch which cannot be scraped off or attributed to any other disease. It is frequently considered a precancerous condition, and use of tobacco and alcohol have been statistically linked to oral neoplastic disease. Their combined use is thought to be synergistic.[8]

TONGUE

The tongue is a frequent site of oral soft tissue changes. Benign changes range from a very slight reddening (which again may be due to a vitamin deficiency, but yet is not corrected by vitamin supplements)[9] to

slight fissuring. The dorsal surface may have changes in texture that begin with minor atrophy of the filiform papillae at the tip, to a completely smooth, lacquered tongue. One may also observe a severely lobulated tongue, again with complete atrophy of the papillae. All of these changes may cause altered taste perception and possibly contribute to the decreased appetite of many elderly individuals.[3]

The next most common tongue change is an increase in lingual varicosities on the ventral surface.[10] It has been shown in Bhashkar's studies[11] to affect well over 50% of the over 65 population.

Glossodynia or burning tongue is a not infrequent complaint of the aged. It may be associated with obvious changes in appearance or it may be present with no apparent cause at all.[12] Geographic tongue or benign migratory glossitis is an interesting lesion of unknown etiology. One sees multiple areas of desquamation of filiform papillae in an irregular pattern. The fungiform papillae persist even in the desquamated area as small, elevated red dots. The areas of desquamation remain for a short time in one location and then heal and appear in another, thereby giving rise to the idea of migration. It is not unusual for the condition to persist for months, to regress spontaneously, and then to recur at a later date.

One further change in the tongue is the increase in size (macroglossia) in many aged individuals. Although this condition sometimes occurs as a congenital problem or is due to tumors, in an elderly population it is more due to a combination of loss of muscle tone and expansion to fill the oral cavity space as teeth are lost.[12]

Bone

No discussion of oral soft tissue change is complete without some mention of periodontal disease and bone changes. The relationship between age and the prevalance of periodontal disease has been reported in numerous epidemiologic studies. In all of these studies, the prevalance and severity of periodontal disease was found to increase throughout life and, by age 45, 97% have some aspect of the problem, with an average of 40% of alveolar bone loss by age 60. Statistically, age and oral hygiene seem to play a supporting role, and recent evidence supports the idea of a genetic component to the problem.

The primary local cause of periodontitis is the accumulation of plaque and subsequent deposition of calculus. If these irritants are not removed,

marginal gingivitis, an irritation, reddening, and swelling of the gingivae, leads to ulceration of the gingival lining, apical migration of the epithelial attachment, and bone loss in the stage known as periodontis. The presence of plaque and calculus initiate and prolong the process by local irritation and by the more serious presence of a bacterial infection. Bacteria within the plaque are responsible for the ultimate prognosis of the disease. As an individual ages, the bacterial composition of the periodontal pocket changes as gram positive facultative cocci increase and gram negative anaerobic rods decrease. The usual mixture of the so-called fusospirochetal complex remains constant.[13]

Aside from the bone loss associated with periodontal disease, there is progressive rapid loss of height of the aveolar ridge once teeth are extracted. Three years after extraction, 50% of alveolar ridge bone is lost and this process continues so that in some individuals absolutely no alveolar bone may be present at all.[14] Therefore, the earlier in life teeth are extracted, the greater will be the bone loss and usually the less comfortable will be the denture. If this problem is considered in relation to the statistics that 80% of all people over the age of 65 are edentulous, it is no wonder that the aged population has such great difficulty with dentures.

One further problem that the aged have with bone is the slowly progressing process of senile atrophy of bone in normal aging.[1] This process of true loss of bone dimension, as well as osteoporosis, is as much a problem in the nonunion of fractures of the mandible of the elderly as it is in the publicized nonunion of hip fractures. In fact, 20% of all fractures of the edentulous mandible result in nonunion.[15]

Summary

Major oral soft tissue changes in aging have been described, with emphasis on salivary gland, mucous membrane, tongue, and underlying bone. Most geriatric oral soft tissue changes are benign, but cause difficulties with the use of dentures or occasionally even with the natural dentition. Infrequently, malignant or painful lesions present greater problems.

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