Review Article

Oral health and its association with morbidity and mortality in the elderly.
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Abstract
A strong association has been identified between oral health and morbidity/mortality in the elderly independent of established risk factors for mortality. Studies have also identified dental factors that promote health and consequently increase longevity. This literature review explores this association further.

Keywords
Oral health, Morbidity, Mortality, Elderly

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Review

A strong association has been identified between oral health and morbidity/mortality in the elderly independent of established risk factors for mortality [1]. An association between number of teeth and mortality has been reported in several studies [2-11]. As people age, many lose teeth, which reduces masticatory capacity, influence food selection, nutritional status, and general health. Evidence is also increasing that oral infections play a role in the pathogenesis of some systemic diseases and may be especially debilitating in the old and frail [12, 13].

Good oral hygiene such as tooth brushing at night and flossing and not tooth brushing in the morning and using mouth wash were linked to decreased risk of death [1]. Similarly, previous studies have also identified dental factors that promote health and consequently increase longevity [2]. Hämäläinen et al has shown using the Kaplan–Meier method in conjunction with survival curves and Cox regression analytical models that the more teeth or filled teeth a subject had, the lower was her/his risk for death. The effect of missing teeth was significant after adjusting for the general health variables [2]. Moreover, Shimazaki et al., found that male gender, edentulousness, and older age increased the mortality risk in elderly subjects [3]. This was assumed to be related to deterioration in the systemic health of the elderly, which was confirmed by Österberg et al. [4] of three different cohorts of elderly people. Authors found that the survival rate of men between 70 and 79 years of age was higher among those with a well-preserved dentition at the age of 70 years. Similarly, Appollonio and group showed that dentition, nutritional parameters, use of healthcare services, and smoking were significant predictors of mortality in elderly from Italy [15,16]. Researchers also found that those with an adequate natural dentition (16 teeth or more) had a lower relative risk for mortality compared with denture wearers or subjects with a naturally inadequate dentition.

One of the clearest examples of how poor oral health can aggravate morbidity is illustrated by patients with chronic kidney disease (CKD). Cardiovascular disease (CVD), which is often due to or combined with atherosclerosis and infectious complications, is the main cause of death in patients with CKD [17,18]. A number of traditional and uremia-specific risk factors coexist in CKD and contribute to the increased cardiovascular risk in CKD population [17]. Poor oral health, which is related to advanced age and diabetes mellitus are some of the risk factors for tooth loss. Recent studies have shown periodontitis is associated with coronary heart disease and cerebrovascular disease in the general population [19] as well as in haemodialysis (HD) patients [20]. Periodontitis, in turn, is a risk factor for tooth loss implying that this disease may be linked to tooth loss.

Poor oral health and chronic ailments

As of yet, there are no accepted mechanism that explains poor oral health and mortality. Nevertheless, a plausible mechanism has been proposed in the case of gingivitis and periodontitis and chronic kidney disease [21]. Gingivitis (defined as inflammation of the gingiva) and periodontitis (inflammation of the gingiva plus supporting tissues of the teeth) are common manifestations of poor oral health. Periodontitis lead to formation of periodontal pockets colonized by gram-negative anaerobic bacteria, an inflammatory cell infiltrate exudes into the lesion that secretes proinflammatory mediators [22, 23]. Both gingivitis and periodontitis are seen more frequently in end-stage renal disease (ESRD) patients [24-26]. Gingival hyperplasia is a relatively common periodontal complication in renal transplantation patients, which has been attributed to
cyclosporin and the presence of dental plaques [27].

The causes of periodontitis are not fully elucidated, but it has been proposed that repeated systemic anticoagulation may predispose haemodialysis patients to gingival bleeding promoting bacterial colonization [28]. At the same time, oral barriers might have compromised because of disturbed humoral defence [23]. Commencement of dialysis therapy appears to be accompanied by major changes in the oral condition [23], and therefore periodontal disease has been reported to progress in severity across predialysis, peritoneal dialysis and haemodialysis patients, respectively [28]. However, only few studies have compared the prevalence of these symptoms in haemodialysis patients and peritoneal dialysis patients. One study, however, reported periodontitis to be less severe in peritoneal dialysis patients and moderate in predialysis CKD patients compared to haemodialysis patients [28].

A proposed model for how periodontitis could act as a potential cause of local and systemic inflammation in CKD patients was illustrated by Akar and co-workers [21]. At least two reports support the hypothesis that periodontitis may contribute to the systemic inflammatory burden in the ESRD population [25,29]. Poor oral health status was found in 80% of 253 haemodialysis patients with periodontal disease and was associated with both high C-reactive protein and low serum albumin levels in univariate analysis but not in multivariate [25]. Along with increased risk of systemic inflammation, poor oral health has also been linked to increased susceptibility to infectious diseases, such as HIV and HPV [17].

Periodontitis represents a potential source of episodes of bacteraemia, especially in the immunocompromised patients. This finding may not be surprising considering that the overall size of periodontal lesions may range from 1500 to 2000 mm² [30] and that the number of bacteria can exceed $1 \times 10^8$ in a single periodontal pocket [31]. Bacteraemia can occur after almost any type of dental activities including tooth brushing and flossing. These episodes, however, are usually transient and inconsequential in healthy individuals. In contrast, bacteraemia in patients with dental caries and periodontal disease tends to be more sustained, raising the risk of haematogenous dissemination of the dental infection [32]. Bacteria can adhere to damaged heart valves and cause endocarditis, and for this reason prophylactic antibiotics are recommended in patients with valvular heart disease [32]. It is possible that the immune dysfunction in uraemia [18] may substantially increase the risk for such systemic consequences of periodontitis and other oral pathological conditions because underlying mechanisms are not very well understood. Although links between oral health and infections have not been systematically studied, there are several reports in the literature [32]. Bacteria from oral biofilms may be aspirated into the respiratory tract and may cause the initiation and progression of systemic infections such as pneumonia in immune compromised subjects [33].

The innate immune system provides immediate protection against infection and inflammation by recruiting of immune cells, activation of complement systems, identification and removal of foreign substances, and activation of the adaptive immune system [34]. However, in inflammatory diseases, the responses become chronic, and chronic diseases may develop because of repeated unchecked and maladapted inflammatory responses over the years [35]. The earliest changes in atherosclerosis occur in the endothelium, leading to accumulation of monocytes and T-cells, migration of polymorphonuclear leukocytes into the intima, differentiation and proliferation of the monocytes, and eventually development of fibrous cap. Two case studies, examine the role of chronic
bacterial infections as risk factors for atherosclerotic complications, the association between poor dental health and acute myocardial infarction. The study sample in one of these studies included 100 patients with acute myocardial infarction and 102 controls [36]. Dental health was found to be significantly worse in patients with acute myocardial infarction than in controls and the association remained valid after adjusting for age, social class, smoking, serum lipid concentrations, and the presence of diabetes [36]. Poor oral hygiene, determined by the extent of dental plaque and calculus, was associated with an increased incidence of coronary heart disease, and in men younger than 50 years at baseline, periodontal disease was a risk factor for coronary heart disease [37]. Patients with periodontitis exhibited dyslipidaemia and increased non-fasting serum glucose levels compared with controls, suggesting a possible link between periodontitis, systemic inflammation, and a dysmetabolic state in otherwise healthy individuals [38]. Consequently, in periodontitis, proliferation of gram-negative bacteria may cause endotoxemia and systemic inflammation leading to CVD [39]. Periodontal disease may represent a risk factor for atherosclerosis and thromboembolic events [40]. Periodontal treatment could reduce the risk of coronary heart disease and therefore become one of the preventing strategies [41-42]. One preventative strategy of periodontitis is regular physical exercise. Physical activity has a beneficial effect on person's general health and wellbeing. Research has shown protective effects of regular physical activity on the risk of coronary heart disease, diabetes mellitus, colon cancer, obesity, osteoporosis, arthritis, hypertension, and high cholesterol [43]. In addition, regular physical activity has also been suggested to increase life expectancy and to improve life quality [44]. With respect to periodontitis, an inverse association was found between physical fitness and periodontitis prevalence [45]. Another study, in Finland found that long-term physical inactivity was associated with the need for periodontal treatment [46]. A recent study, conducted in US male health professionals found an inverse relation between physical activity levels and the risk for developing periodontitis [47]. A possible mechanism by which physical activity may protect against periodontitis is by increasing sensitivity to insulin among diabetes and subsequent prevention of progression of the disease. This is a recognized risk factor for periodontitis. Muscle contraction during physical exercise was suggested to have a synergistic effect with insulin in enhancing glucose uptake into the cells [43]. This may be related to both increased blood flow and enhanced glucose transport to the muscle cell [43]. Several studies have showed an association between physical activity and insulin sensitivity [48-51]. Another possible mechanism is the reduction in inflammation, which plays a significant role in the pathogenesis of periodontitis. Two studies, including recent reports using NHANES III data, reported an inverse association between physical activity and the plasma level of inflammatory markers such as C-reactive protein [52, 53]. This reduction was related to the beneficial effect of physical activity on improving both blood flow and efficiency of oxygen exchange [43]. Physical activity may also protect against development of obesity, by increasing the kilocalories used than consumed [43], and obesity has been associated with increased periodontitis prevalence [54, 55]. Finally, physical activity may improve periodontal health by reducing stress, which has been associated with higher periodontitis prevalence [56].

Poor oral health and mental health
Dementia is projected to be an emerging health problem during the next few decades [57]. The contribution of systemic chronic
inflammation in the etiology and pathogenesis of neurodegenerative diseases such as Alzheimer disease or dementia seems to be a focus of current research. It is known that inflammation is a critical component of the pathogenesis of Alzheimer disease [58]. Although inflammation is not an initiator of this disorder, it nonetheless plays a pivotal role as a driving force that can modulate the neuropathology. Evidence from prospective studies is accumulating that elevations of pro-inflammatory serum markers [such as C-reactive protein, interleukin (IL)-1, TNF-α, IL-6 and α1-antichymotrypsin], may precede cognitive impairment [59-61].

One possible pathway between the local oral infection/inflammation and the inflammation in the brain may be that lipopolysaccharides (LPS) affect the passage of other regulatory proteins across the blood-brain barrier. In mice, it was found that microglial activation can exacerbate the neuropathology and that LPS induces CNS inflammation. In human autopsies, antigen of oral treponemes was more often found in subjects with Alzheimer disease (14 of 16) than those without [62]. There are several reasons for the putative association between periodontitis and cognitive impairment may be relevant from a public health perspective: 1) Periodontitis is highly prevalent and leaves a significant proportion (20–30%) of the adult population at a high risk for chronic inflammatory processes. 2) Because more teeth are saved from caries and because life expectancy is extended, more teeth will be at risk for periodontitis [63]. 3) In an ageing population, the prevalence of cognitive impairment increases tremendously. 4) In an ageing population, systemic inflammatory conditions may interact with immune functions in the brain and trigger the progression of Alzheimer’s neuropathology. 5) Periodontitis is a curable disease, and 6) the treatment of periodontitis and retention of teeth could delay or alleviate cognitive impairment.

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