Aspiration pneumonia is one of the major causes of death for elderly persons. In nursing home-associated pneumonia (NHCAP), micro-aspiration, higher mortality, and antibiotic-resistant bacterial infections are well-known problems. In NHCAP cases, several factors on the host side are related to high mortality. The importance of daily oral care in elderly patients with dysphagia is also well recognized. Silent aspiration of bacteria-containing saliva and salivary pro-inflammatory cytokines produced by interdental plaque and periodontitis might stimulate lower respiratory inflammation and lead to aspiration pneumonia. However, the relationship between micro-aspiration and inflammatory cytokines produced in the lower respiratory tract remains uncertain. In this context, oral care, dental care, and the inhibitory effects of dental care on recurrent aspiration pneumonia in elderly persons will be discussed.

**Key words:** Oral care; Pneumonia; Inflammatory cytokines; Micro-aspiration; Geriatric

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**ABSTRACT**

Aspiration pneumonia is one of the major causes of death for elderly persons. In nursing home-associated pneumonia (NHCAP), micro-aspiration, higher mortality, and antibiotic-resistant bacterial infections are well-known problems. In NHCAP cases, several factors on the host side are related to high mortality. The importance of daily oral care in elderly patients with dysphagia is also well recognized. Silent aspiration of bacteria-containing saliva and salivary pro-inflammatory cytokines produced by interdental plaque and periodontitis might stimulate lower respiratory inflammation and lead to aspiration pneumonia. However, the relationship between micro-aspiration and inflammatory cytokines produced in the lower respiratory tract remains uncertain. In this context, oral care, dental care, and the inhibitory effects of dental care on recurrent aspiration pneumonia in elderly persons will be discussed.

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**List of abbreviations**

NHCAP: nursing home associated pneumonia; HCAP: healthcare-associated pneumonia; POHC: professional oral health care; CVD: cerebral vascular disease; IL: interleukin; COPD: chronic obstructive lung disease

**PNEUMONIA, MICRO-ASPIRATION, AND ORAL CARE IN ELDERLY PERSONS**

Aspiration pneumonia is well known as a cause of pneumonia in elderly persons. We previously reported the involvement of interleukin (IL)-17A in extravasation of neutrophils in cases of aspiration pneumonia[1]. In elderly patients with aspiration pneumonia, cerebral vascular disease (CVD) and diabetes mellitus were found to be related to a poor prognosis[2]. Improvement of mortality and inhibiting recurrent pneumonia are major problems in elderly patients with pneumonia. The importance of oral care in elderly patients with swallowing disorders is also well recognized, and many papers have already been published[3-5]. Periodontitis is a polymicrobial infection and multifactorial disease, and it is characterized by chronic inflammation of the periodontium[6]. Oral health is closely related to systemic diseases, including CVD, and pneumonia. Inadequate oral care might result in accumulation of bacterial plaque biofilm[7,8].

Generally, oral care is divided into two categories. One is oral
health self-care (basic daily oral care), and the other is professional oral health care (dentist care or dental hygienist care). Basic daily oral care includes brushing teeth and dentures and swabbing the buccal mucosa and tongue. On the other hand, professional oral health care includes brushing teeth, swabbing the buccal mucosa and tongue, cleaning dentures, mouth-rinse, and dental visit. So far, differences between and the efficiency of the two categories have not been well examined. Furthermore, the relationship between micro-aspiration of bacteria-containing plaque and cytokine stimulation in the lower respiratory tract remains uncertain. In this manuscript, professional oral health care and its inhibitory effect on recurrent aspiration pneumonia in elderly persons are discussed. In addition, oral care and lower respiratory inflammation are also discussed from the viewpoint of cytokines.

**BASIC ORAL HEALTH CARE**

The purpose of basic daily self-oral care is cleaning the oral cavity, improving eating function training, and prevention of dry mouth. After daily oral hygiene care, including oral moisture retention and cleaning of the tongue and palate, pathogen colonization could be minimized[43]. Mechanical stimulation of the oral mucosa leads to brain stimulation and improvements in dysphagia, latency time of the swallowing response, and the cough reflex. Oral health care can help prevent aspiration pneumonia associated with dysphagia. Improving eating function reduces the occurrence rate of massive aspiration. One merit of conventional oral care is that nursing staff and non-specialists, including the patient’s family, can be involved in the patient’s care. Recently, Takahashi et al reported[16] that aspiration of periodontopathic bacteria due to poor oral hygiene potentially contributed to the aggravation of COVID-19 causing by the severe acute respiratory syndrome coronavirus 2.

**PROFESSIONAL ORAL HEALTH CARE (POHC) AND DENTAL CARE**

POHC is expected to prevent recurrence of aspiration pneumonia in elderly persons. However, there are only a few lines of evidence showing that POHC intervention after illness improves severe aspiration pneumonia[13]. Since POHC requires seeing a dentist or dental hygienist, not all patients can receive such care. One major merit of POHC is plaque removal and treatment of periodontitis[13], which might lead to mechanical removal of oral pathogens. One systematic review by Liu et al reported[10] that it was not possible to establish the effects of POHC on the incidence rate of healthcare-associated pneumonia (HCAP) compared with usual oral care over an 18-month period of observation. Furthermore, they reported that POHC reduced pneumonia-associated death by 60% in comparison with usual oral care over a 24-month follow-up period. In addition, another paper reported[11] that POHC suppresses mortality due to pneumonia and prevents morbidity with influenza.

**INDUCED-SPUTUM AND SALIVARY CYTOKINES AS BIOMARKERS**

In an animal model experiment[17], heat-inactivated periodontopathic bacteria induced the production of inflammatory cytokines including IL-6 and IL-8 by pharyngeal, bronchial, and alveolar epithelial cells, where these bacteria induced cytokine production more than *S. pneumoniae*. When the periodontopathic bacterium *F. nucleatum* is injected into the murine trachea, protein levels of IL-6 and keratinocyte-derived chemokine were markedly increased in the lower respiratory tissues and serum, indicating that the bacterium could induce pro-inflammatory cytokine production in vivo. This micro-aspiration stimulates inflammation of the lower respiratory tract and produces pro-inflammatory cytokines[18]. Salivary cytokines stimulated by periodontitis and interdental plaque might release lower respiratory tract cytokines. So far, several other reports[14-16] about periodontitis and salivary inflammatory cytokines have been published. Escalona et al reported[17] that the gingival fluid levels of IL-1α, IL-1β, tumor necrosis factor-α, IL-6, and IL8 in chronic periodontitis cases were higher and associated with the severity of periodontitis. Members of the IL-1 family are thought to act as the key mediators of the host response to microbial invasion, inflammation, and tissue injury[14-18]. We also reported the involvement of IL-36γ one of the IL-1 family members, in acute lung injury[19]. Do induced-sputum inflammatory cytokines act as biomarkers of micro-aspiration? Do salivary cytokines act as biomarkers of pneumonia risk? As far as we could determine, there have been no reports of the inhibitory effects of salivary and or induced-sputum inflammatory cytokine changes after POHC. However, in chronic obstructive lung disease (COPD), there have been many reports about induced-sputum cytokine levels and sputum microbiota. For example, Tangedal et al reported[20] the changes in microbial composition between stable COPD and exacerbations. Pro-inflammatory cytokine levels were significantly different between the groups, and changes in the sputum microbiota with COPD states were accompanied by changes in cytokines. Other research also reported[17] that IL-1β was associated with acute exacerbation of COPD and reaffirmed the association between IL-1β pathway activation and airway bacterial infection in COPD. Therefore, induced-sputum cytokines and salivary cytokines could become non-invasive biomarkers of aspiration pneumonia. Comparing with induced sputum, saliva can be easily, repeatedly, and noninvasively obtained and stored without the use of specialized equipment. From the point of view of POHC, removal of interdental plaque and treatment of periodontitis would suppress salivary cytokine production. Furthermore, aspiration of salivary cytokines stimulates inflammation of the lower respiratory tract and increases sputum-induced cytokines[17,21].

**FUTURE DIRECTION**

Taken together, POHC is recommended for elderly persons with swallowing disorders and those with aspiration pneumonia. POHC might have the possibility of reducing pneumonia-associated death compared with daily self-oral care. Salivary cytokines and/or sputum-induced cytokines might act as biomarkers for the risk of aspiration pneumonia. However, more scientific evidence from the laboratory is needed before this knowledge can be translated into practice. Finally, we should pay attention to the future of the field of oral care-related research.

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