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Editorial

The Specific Role of Periodontal Disease in Lung Cancer Pathogenesis

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Editorial

It has been proposed that poor oral hygiene, especially in high risk patients could be a predatory factor for respiratory diseases. Oral cavity that coincides with trachea could be a natural gate for colonization by respiratory pathogens. Dental plaque can be colonized by those that have been inhaled from the oro pharynx cavity to the upper airways and then can approach the lower airway and adhere to the bronchial or alveolar epithelium [1,2]. Periodontal Disease (PD) has been associated with chronic lung diseases, such as Chronic Obstructive Pulmonary Disease (COPD) [3,4]. It has also been recorded that pulmonary function in COPD patient's decreases with increasing Clinical Attachment Loss (CAL), and an association between poor oral hygiene and COPD was recorded after controlling for potential confounders, such as gender, age, smoking andsocio-economic level [5]. The mentioned findings were in accordance with those that carried out from previous case-control and prospective studies [6-10].

However, only one similar investigation has not confirmed such observations [11]. Epidemiological researches have investigated the association between COPD and lung cancer (LC) [12-14]. Cigarette smoking is the principle risk factor for developing COPD [15] and LC [12-16]. Smokers with COPD are at a higher risk of developing LC, suggesting that an association exists between the processes that causes both diseases [17,18]. However, the majority of smokers do not develop COPD or LC despite the fact that the accumulated smoking history increases the risk of developing PD and cancer [17-20]. The contribution of smoking to PD development requires poor oral hygiene whereas in cases of established PD, smoking accelerates its progression. Similarly, smoking consists an important risk factor for LC development, however does not cause the disease to all smokers [21].

It is possible that the link between both diseases is mediated by a pre-existing chronic pulmonary disease such as idiopathic pulmonary fibrosis, COPD, and tuberculosis that are associated with an increased risk of LC development. A limited exploration exists regarding the association between COPD and LC risk, especially in non-smokers, as they develop COPD rarely [13]. In a prospective study in health professionals with a PD history a slightly higher cancer incidence compared to those who did not have PD at the time of the study initiation and after controlling for known risk factors, was observed. It was also found that PD was statistically significantly associated with an increased risk for LC. Specifically, regarding LC, a strong association was recorded in individuals with a small number of remaining teeth and it was remained strong among non-smokers although it was weaker and statistically insignificant. However, many limitations were existed especially the self-reported assessment of PD [22]. Hujoel et al. [23] observed an increased risk of death from all types of cancer when the assessment of PD was based on other indicators. Specifically, it was found a statistically increased risk of LC, however it was not confirmed in non-smokers. It was suggested that periodontitis could include an unlimited percentage of smoking history, and therefore its relationship with the LC was invalid. In the same study, gingivitis that reflects the presence of inflammatory load, in individuals with natural teeth showed increased risk levels of death from LC.

Periodontitis could be a substitute indicator for the effects of smoking and therefore provides a link to LC.

The follow-up of NHANES III patients was shown that periodontitis was associated with an increased mortality from respiratory cancers. There was an increased risk tendency in relation to periodontitis severity [24]. Chrysanthakopoulos [25] was recorded that probing pocketdepthasan index for PD severity was statistically significantly associated with the risk of developing LC after controlling for potential confounders. In addition to the suspected effect of COPD and smoking on the interpretation of the association between PD and the risk for developing of LC, the mechanisms that could link the two diseases have not been elucidated. Therefore, future studies are required, and they could be based on the

overall association between chronic inflammation and the risk of cancer development.

The inflammatory response to periodontal inflammation extends beyond the oral cavity and leads to increased levels of circulating inflammatory biomarkers [26,27], a finding that is supported from the highest incidence of cancer cases in individuals with chronic inflammatory conditions [28] and the efficacy of anti-inflammatory drugs in the prevention of certain types of cancer [29], how-ever that finding has not been confirmed in all studies [30,31], nor in LC. A possible mechanism can be supported on the basis of the PD pathogenesis and is characterized by a wide variety of microorganisms, bacteria and their products such as endotoxins, enzymes and metabolic products that are toxic to surrounding cells and can directly induce mutations in tumor suppressor genes and proto-oncogenes or can alter signaling pathways that affect cell proliferation and/or epithelial cell survival [32,33].

Other mechanisms that have been proposed include the immune system deflection and the production of carcinogenic products by periodontal tissues pathogens [34].

In addition, under-lying genetic factors may increase the sensitivity in both situations or may alter the relationship of environmental risk factors such as smoking, with PD and cancer. The role of the common genetic risk factors between the two situations, however, remains unclear. The sensitivity to the disease could be influenced by predetermined factors such as genetic polymorphisms that also need to be investigated [35,36]. Another possible mechanism could be attributed to the large inflammatory load caused by the PD and could be associated with the risk of LC through the mechanisms that are involved and lead to oxidative stress that causes damage to the tissues. The oxidative stress induces mechanisms in the progression of PD and cancer. Various types of PD are characterized by significant inflammation load that could affect the evolution of systematic conditions, including carcinogenesis in the lung [23].

It should be noted that the research regarding the possible association of PD with cancer is limited by the problems of controlling of confounders such as smoking and the socio-economic level. Smoking seems to be the main confounding factor in studies that examine cancer, such as LC. Other possible confounders are gender, age, nationality, diabetes mellitus and genetic factors. The definition of periodontitis in a number of large epidemiological studies was based on substitute indicators [24]. However, despite several limitations, periodontitis has been identified as a potential risk factor for gastrointestinal, respiratory and pancreatic cancer and possibly for cancer in other organs. Therefore, it exists an obvious need for further investigation.

References

1. Didilescu AC, Skaug N, Marica C, Didilescu C (2005) Respiratory pathogens in dental plaque of Hospitalized patients with chronic lung diseases. Clinical Oral Investigations 9(3): 141-147.
2. Scannapieco FA (1999) Role of oral bacteria in respiratory infection. Journal of Periodontology 70(7): 793-802.
3. Deo V, Bhongade ML, Ansari S, Chavan RS (2009) Periodontitis as a potential risk factor for chronic obstructive pulmonary disease: a retrospective study. Indian Journal of Dental Research 20(4): 466-470.
4. Sharma N, Shamsuddin H (2011) Association between respiratory disease in hospitalized patients and periodontal disease: a cross-sectional study. Journal of Periodontology 82(8): 1155-1160.
5. Scannapieco FA, Ho AW (2002) Potential associations between chronic obstructive pulmonary disease and periodontal disease: analysis of National Health and Nutrition Examination Survey III. Journal of Periodontology 72(1): 50-56.
6. Wang Z, Zhou X, Zhang J, Zhang L, Song Y, et al. (2009) Periodontal health, oral health behaviours, and chronic obstructive pulmonary disease. Journal of Clinical Periodontology 36(9): 750-755.
7. Hayes C, Sparrow D, Cohen M, Vokonas PS, Garcia RI (1998) The association between alveolar Bone loss and pulmonary function: the VA Dental Longitudinal Study. Annals of Periodontology 3(1): 257-61.
8. Garcia RI, Nunn ME, Vokonas PS (2001) Epidemiologic associations between periodontal disease and chronic obstructive pulmonary disease. Annals of Periodontology 6(1): 71-77.
9. Hyman JJ, Reid BC (2004) Cigarette smoking, periodontal disease, and chronic obstructive pulmonary disease. Journal of Periodontology 75(1): 9-15.
10. Katancil JA, Kritchevsky S, Weyant RJ, Corby P, BretzW, et al. (2005) Periodontitis and airway obstruction. Journal of Periodontology 76(11): 2161-2167.
11. Azarpazhooh A, Leake JL (2006) Systematic review of the association between respiratory diseases and oral health. Journal of Periodontology 77(9): 1465-1482.
12. Alberg AJ, Samet JM (2003) Epidemiology of Lung Cancer. Chest 123: 21S-49S.
13. Thun MJ, Henley SJ, Burns D, Jemal A, Shanks TG, et al. (2006) Lung cancer death rates in lifelong non-smokers. Journal of the National Cancer Institute 98(10): 691-699.
14. Burcham PC (1998) Genotoxic lipid peroxidation products: Their DNA damaging properties and role in formation of endogenous DNA adducts. Mutagenesis 13(3): 287-305.
15. Waldner MJ, Neurath MF (2009) Colitis-associated cancer: the role of T cells in tumor development. Seminars in Immunopathology 31(2): 249-256.
16. Ezzati M, Lopez AD (2003) Estimates of global mortality attributable to smoking in 2010. Lancet 362(9387): 847-852.
17. Mannino DM, Aguayo SM, Petty TL, Redd SC (2003) Low lung function and incident lung cancer in the United States: data from the First National Health and Nutrition Examination Survey follow-up. Archives of Internal Medicine 163(12): 1475-1480.
18. Lange P, Nyboe J, Appleyard M, Jensen G, Schnohr P (1990) Ventilatory function and chronic mucus hyper-secretion as predictors of death from lung cancer. The American Review of Respiratory Disease 141(3): 613-617.
19. Prendergast GC (2008) Inflammatory mediators in cancer etiology and targets for therapy and prevention. Can Rev Onl 9: 17-18.
20. Taraseviciene Stewart L, Voelkel NF (2008) Molecular pathogenesis of emphysema. The Journal of Clinical Investigation 118(2): 394-402.
21. Tomar SL, Asma S (2000) Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. Journal of Periodontology 71(5): 743-751.

22. Michaud DS, Liu Y, Meyer M, Giovannucci E, Joshipura K (2008) Periodontal disease, tooth loss, and cancer risk in male health professionals: a prospective cohort study. The Lancet. Oncology 9(6): 550-558.

23. Arora M, Weuve J, Fall K, Pedersen NL, Mucci LA (2010) An exploration of shared genetic risk Factors between periodontal disease and cancers: a prospective co-twin study. American Journal of Epidemiology 171(2): 253-259.

24. Chrysanthakopoulos NA (2016) Correlation Between Periodontal Disease Indices and Lung Cancer in Greek Adults: a Case - Control study. Experimental Oncology 38(1): 49-53.

25. Amabile N, Susini G, Pettenati-Soubayroux I (2008) Severity of periodontal disease correlates to inflammatory systemic status and independently predicts the presence and angiographic Extent of stable coronary artery disease. Journal of Internal Medicine 263(6): 644-652.

26. Moutsopoulos NM, Madianos PN (2006) Low grade inflammation in chronic infectious diseases: paradigm of periodontal infections. Annals of the New York Academy of Sciences 1088: 251-264.

27. Erlinger TP, Platz EA, Rifai N, Helzlsouer KJ (2004) C-reactive protein and the risk of incident colorectal cancer. J Am Med Assoc 291(5): 585-590.

28. Tezal M, Sullivan MA, Hyland A, Marshall JR, Stoler D, et al. (2009) Chronic periodontitis and the incidence of head and neck squamous cell carcinoma. Cancer epidemiology, biomarkers and prevention: a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology 18(9): 2406-2412.

29. Fitzpatrick SG, Katz J (2010) The association between periodontal disease and cancer. A review of the literature. Journal of Dentistry 38(2): 83-95.

30. Soder B, Jin LJ, Klinge B, Soder PO (2007) Periodontitis and premature death: a 16-year longi- tudinal study in a Swedish urban population. Journal of Periodontal Research 42(4): 361-366.

31. Abnet CC, Qiao YL, Dawsey SM, Dong ZW, Taylor PR, et al. (2005) Tooth loss is associated with increased risk of total death from upper gastrointestinal cancer, heart disease, and stroke in a Chinese population-based cohort. International Journal of Epidemiology 34(2): 467-474.

32. Zaris S, Bojic B, Jankovic L, Dapcevic B, Popovic B, et al. (2009) Periodontal therapy improves gastric Helicobacter pylori eradication. Journal of Dental Research 88(10): 946-950.

33. Chrysanthakopoulos NA, Vlassi C (2013) Reasons for Extraction of Permanent Teeth and Risk Indicators in a General Dental Practice in Greece. Int J Med Dent 3: 315-321.

34. Coussens LM, Werb Z (2002) Inflammation and cancer. Nature 420(6917): 860-867.

35. Marx J (2004) Cancer research. Inflammation and cancer: the link grows stronger. Science 306(5698): 966-968.

36. Milasin J, Jakoba NN, Stefanovic D, Sopta J, Pucar A, et al. Periodontal Inflammation as Risk Factor for Pancreatic Diseases: 131-156.

DOI: 10.32474/MADOHC.2018.01.000108