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Potential Association between Periodontitis and COVID-19 Infection Outcomes

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Dear Editor,

Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) was declared a pandemic on March 11, 2020, and to date remains a global challenge. Research on infection mechanisms, treatment, and association with other diseases is still ongoing. Therefore, identification of the associated risk factors and co-morbidities is vital to ensure reduction in disease morbidity and mortality. Virus-activated mast cells have a key role in SARS-CoV-2 infection. Mast cells release proteases, histamine, and pro-inflammatory cytokines/chemokines, amplifying asthma and inflammatory reactions in the lungs. Simultaneous activation of T-cells, natural killer cells, macrophages/monocytes, and endothelial cells leads to a cytokine storm. Other immune effects include lymphopenia, eosinopenia, and increased D-dimer, C-reactive protein (CRP) and Th-17 cells. Cytokine storm and lymphopenia are currently used to predict the disease severity and mortality [1].

Periodontitis has not yet been established as a co-morbidity for COVID-19; however, there are related immune mechanisms between the two diseases [2]. Periodontal disease involves a pro-inflammatory host response triggered by a dysbiotic flora leading to the onset of specific immune orchestra that resembles the immune response seen in COVID-19. Periodontal bone destruction is due to a pathogen-associated molecular pattern that induces the production of IL-1β, tumor necrosis factor-α, and prostaglandin E2 which in turn activate osteoclasts. The secretion of matrix metalloproteinases by local polymorphonuclears and fibroblasts also contributes to tissue damage [3].

Parallels drawn between the pathobiology of the two diseases lead to the hypothesis of a potential association between periodontitis and COVID-19 severity and outcome. An elevated Th-17 pathway response has been documented in SARS-CoV and MERS-CoV infections [1]. This pathway mediated by IL-17, has been also implicated in periodontal disease, leading to elevated levels of IL-17 in serum and periodontal tissues [4]. Cheng et al. [4] reported a fall in IL-17 levels in serum and gingival

crevicular fluid after non-surgical periodontal treatment. Since the two disease mechanisms involve overlapping cytokines, drug testing currently targets the same receptors. Anakinra (an IL-1 receptor antagonist) and mast cell stabilizers are potential therapeutic targets for both COVID-19 and periodontitis [1]. CRP, a prognostic marker, was found to be 10-times higher in deceased- compared to recovered-COVID-19 patients [5]. Periodontitis patients have higher serum CRP and fibrinogen than those without periodontitis. Periodontal treatment can decrease the overall systemic inflammatory marker levels [3].

Cardiovascular diseases, hypertension, diabetes, and chronic obstructive pulmonary disease have been associated with periodontitis [2,3]. Poor oral hygiene and periodontal attachment loss are suggested independent risk factors for chronic obstructive pulmonary disease [3]. Individuals with systemic co-morbidities show greater adverse outcomes when infected with COVID-19 [2].

Since the association of systemic diseases with both COVID-19 and periodontitis has been confirmed, the effect of periodontal treatment on SARS-CoV-2 infection susceptibility and outcome requires further investigation. The diagnosis and severity of periodontal infection may directly or indirectly influence COVID-19 severity and outcome.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Kempuraj D, Selvakumar GP, Ahmed ME, Raikwar SP, Thangavel R, Khan A, et al. COVID-19, Mast Cells, Cytokine Storm, Psychological Stress, and Neuroinflammation. Neuroscientist. 2020 Oct-Dec;26(5):402-14.

2. Pitones-Rubio V, Chávez-Cortez EG, Hurtado-Camarena A, González-Rascón A, Serafín-Higuera N. Is periodontal disease a risk factor for severe COVID-19 illness? Med Hypotheses. 2020 Nov;144:109969

3. Newman MG, Takei HH, Klokkevold PR, Carranza FA. Periodontal medicine: impact of periodontal infection on systemic health, in Carranza's Clinical Periodontology. Philadelphia, Saunders Elsevier, 2006:312-27.

4. Cheng WC, Hughes FJ, Taams LS. The presence, function and regulation of IL-17 and Th17 cells in periodontitis. J Clin Periodontol. 2014 Jun;41(6):541-9 .

5. Ali N. Elevated level of C-reactive protein may be an early marker to predict risk for severity of COVID-19. J Med Virol. 2020 Nov;92(11):2409-11.