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Letter to the Editor

COVID-19 and risk of inflammatory periodontal disease initiation/progression: a hypothesis for future

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Dear Editor

After reading the interest correspondence of Chaux-Bodard *et al*. "Oral manifestation of Covid-19 as an inaugural symptom?" [1] I thought about possibility of oral tissue manifestations/injuries resulted by this disease.

Coronavirus Disease-2019 (COVID-19) is caused by a novel coronavirus namely severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) which utilizes cell surface angiotensin-converting enzyme 2 (ACE2) as receptor to cell entry [2]. It is suggested that the oral cavity could be considered as a potentially high risk for SARS-CoV-2 infection /transmission [3] and ACE2 expression is demonstrated in gingival and periodontal tissues [3,4].

Gingivitis and periodontitis are two most common forms of inflammatory periodontal diseases (IPDs) and elevated levels of pro-inflammatory cytokines are amongst the most common risk factors for IPDs initiation/progression [5].

If the intracellular events are similar to those reported in airway epithelial cells [2], it may be hypothesized that SARS-CoV-2 infection may be involved in pathogenesis IPDs and facilitate periodontal tissues destruction as mentioned on below:

Virus entry to the cell increases ACE2 expression which affects two main genes of RPS3 and SRC in viral replication and inflammatory responses, respectively. As ACE2 overexpression enhances inflammatory responses through greater pro-inflammatory cytokines production, it leads increase binding of SARS-CoV-2. Consequently, negative regulatory activity of ACE2 on angiotensin II may reduce by SARS-CoV-2 resulting an increase in angiotensin II [2].

In renin-angiotensin system, ACE/Ang II/AT1R axis activates some molecular signaling pathway related to tissue injury including induction inflammatory responses by releasing of cytokines. Oppositely, ACE2/Ang-(1-7)/Mas receptor axis has anti-inflammatory properties. Meanwhile, it seems this axis is an active player in alveolar bone remodeling and its activation can decrease the expression of cytokines directly related to bone resorption [4].

Since ACE2/Ang-(1-7)/Mas receptor axis is in counter-balanced ACE/Ang II/AT1R axis [4] downregulation ACE2 by SARS-CoV-2 which may result in an increase of Ang II [2] enhancing harmful tissue effects of the latter axis.

Although, systemic elevated levels of cytokines can contribute in IPDs onset/exacerbation through enhance the host response to periodontal pathogens [5]. It's pointed out that in COVID-19 patients a higher level of cytokines was observed parallel with the severity of illness [2] which this may increase probability of IPDs development/progression.

Nevertheless, this hypothesis is theoretically possible but future clinical studies are necessary for confirmation of it.

Conflict of interest

The author declares that she has no conflict of interest in relation to the publication of this article.

Ethics committee approval

Not required.

Informed consent

Not required.

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