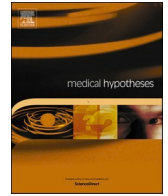




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Letter to Editors

**Periodontal pocket and COVID-19: Could there be a possible link?**

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ABSTRACT

COVID-19 is the emerging health emergency ruining the well being of individuals and devastating the global economies. Sustained research focusing on the virus has been on throughout the world. However, no definitive remedies have yet been derived in the containment of the virus. Steady knowledge on the pathogenesis of the virus has revealed certain consistent features specific to the disease which includes massive destruction of the lung due to the presence of excessive angiotensin-converting enzyme receptors (ACE2) which are essential for the viral entry inside the host. Once, access is gained multiplication occurs resulting in suppressing the immune response of the body against the virus. Henceforth, the equilibrium of the host is disrupted leading to manifestation of the disease. The Periodontal pocket also presents with pathology very much similar to COVID-19 and a possibility of dual role can be thought of pertaining to aspects of Periodontal Medicine.

Sir,

In accord with the hypothesis by V. Sahni [1] and considering the abundance of literature centralizing on the pathogenesis of SARS-CoV-2 and its cynical impact on the immune system, I would like to correlate the concept of periodontal medicine with this contemporary pandemic. With due honors, I would like to remember Steve Offenbacher, “Father of Periodontal medicine” for his unprecedented work [2]. The notorious SARS-CoV (Severe acute respiratory syndrome coronavirus) with its spike mediated entry into target cell through ACE-2 receptors (Angiotensin-converting enzyme receptor-2), affecting lungs is the game resolver for COVID-19. Pascolo et al. (2020) demonstrated concomitant expression of ACE-2 and trans-membrane serine protease TMPRSS2 in salivary glands are required for facilitating viral ingress into cell. Inevitably, Saliva lodges numerous SARS-Co Viruses and periodontal pocket is in close vicinity. Breach of the pocket epithelium results in direct contact of the virus and activation of the host immune response with release of pro-inflammatory cytokines like CL8, CXCL10, C3a, C5a stimulating macrophages, granulocytes, and natural killer cells thus releasing IL-1 β , IL-6, IL-8, TNF- α , IFN- γ [3]. These aggravate the pocket formation by downward displacement of junctional epithelium. “Focal infection theory” by William Hunter states that oral foci of infections spread through the bloodstream affecting systemic organs [4]. As with SARS-CoV-2, the major pathology behind the lung damage is “Cytokine storm” or “Macrophage activated syndrome” which is auto-amplifying cascades of host immune response [5]. ACE-2 receptors in the lungs aggravate the entry of SARS-CoV-2 thereby activating cytokines damaging the respiratory epithelium and lung parenchyma. Cytokine storm suppresses the innate and adaptive immunity against SARS-CoV-2. IL-6 and TNF- α are noticed to increase excessively in severely ill hospitalized individuals. Sir, a hypothesis which could be also thought of is a relationship of COVID-19 to periodontal pocket wherein the cytokine responses happen to be common and the increased cytokines in the periodontal pocket could further aggravate the COVID induced destruction of lung. Research focusing on this issue could additionally

prove the authenticity of the hypothesis.

Declaration of competing interest

None.

Source of support

Nil.

CRediT authorship contribution statement

Nirupa Elisetti: Conceptualization.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.mehy.2020.110355>.

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Nirupa Elisetti

Nirupa's DENTAL ZONE, Nallagandla, Hyderabad, Telangana 500019, India

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E-mail address: nirupasentalzone@gmail.com.