

Editorial

The impact of oral health on high recurrences of long-COVID-19

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Respected Editor,

The SARS-CoV-2 was formally identified on 7 January 2020 by the Chinese Centre for Disease Control and Prevention (CCDC). The COVID-19 symptoms are primarily age-dependent and may be more severe in cases where co-morbidities are present. Both the direct and indirect health repercussions of the epidemic are still being felt today. The COVID-19 pandemic had devastating effects on people's oral and mental health (Nijakowski et al., 2022). Since oral health is associated with both COVID-19 severity and time to recovery, it may be important to evaluate oral health as an independent risk factor for COVID-19 patients (Cardiology, 2021). The oral microbiome has a significant impact on both health and disease. A healthy oral microbiome may also help prevent COVID-19 infection and long-term symptoms. These systemic disorders typically co-occur with skin and mucosal sores. Oral lesions seen in COVID-19 individuals include herpes simplex virus (HSV), candidiasis, geographic tongue, aphthous-like ulcers, hemorrhagic ulcerations, necrotic ulcerations, white hairy tongue, reddish macules, erythematous surfaces, petechiae, pustular enanthema. It is yet unclear if these symptoms are a direct effect of the viral infection, the result of systemic deterioration, or a negative drug reaction (Etemad-Moghadam and Alaeddini, 2021).

In a variety of body sites, such as the oral cavity and lungs, humans harbor immense communities of bacteria, fungi, and viruses. In a healthy condition, the virome and microbiome communities are maintained "in check" via effective and competent immune response and remain in a steady state of equilibrium, called homeostasis. However, dysbiosis, an imbalance in the virome/microbiome ecosystem, has been linked to numerous chronic diseases. Major changes in the diversity and composition of organism communities are a hallmark of virome/microbiome dysbiosis, which may provide a favorable environment for the development of opportunistic infections (Proal and VanElzakker, 2021).

Newly invading viruses, like SARS-CoV-2, may encourage and enhance pathobiont pathogenicity and overall virome/microbiome dysbiosis by disrupting or impairing the immune response. The influenza virus, for instance, compromises the host immune response by disrupting the function of the respiratory tract mucosal barrier and reducing the activity of macrophages (particularly alveolar macrophages) and neutrophils. The expression of the ACE2 receptor in the case of COVID-19 is associated with the potential risk of infection. The oral mucosa and tongue epithelial cells have high expression of ACE2, which can slow the mucosal immune system's response (Xu et al., 2020). Various research demonstrates COVID-19 as a potential agent of virome/microbiome dysbiosis, similar to the influenza virus (Proal and VanElzakker, 2021). Shen et al. (2020) discovered an increase in the number of oral commensal bacteria and enrichment of pathogenic bacteria in

the microbiome of bronchoalveolar lavage fluid from persons with COVID-19 (Shen et al., 2020). This dysbiosis remained regardless of whether respiratory symptoms improved, and SARS-CoV-2 was eradicated (Proal and VanElzakker, 2021).

A dysbiosis of virome /microbiome community may potentially influence the initial risk and virulence of SARS-CoV-2 infection, which complicates the interpretation of the previous COVID-19 investigations. This is because the initial host vulnerability and persisting control of SARS-CoV-2 can be affected by the constitution and function of virome/microbiome communities at any given host organ or site. By either stimulating the immune system to control pathogen invasion more effectively, producing substances that disable pathogens, or simply occupying ecological niche space in a manner that hinders the colonization of body tissue by pathogens, organisms in virome /microbiome communities assist the host defense mechanisms. These defense mechanisms may be impaired when a virome/microbiome community is dysbiotic (Proal and VanElzakker, 2021). It has been shown that inflammation commonly related to poor oral hygiene (Cardiology, 2021), which in turn was found to be associated with virome /microbiome dysbiosis, can cause malfunction or decomposition of oral barriers such as the gingiva. Because of the permeability in the epithelium or oral barrier, pathogens in these communities can enter the bloodstream and contribute to various systemic inflammatory diseases (Proal and VanElzakker, 2021). A previous meta-analysis found that 75% of SARS-CoV-2 positive patients experienced a specific oral symptom, including ulcerations (21.43%), xerostomia (37.58 %), and changes in taste (54.73 %) (Nijakowski et al., 2022). In addition, COVID-19 may lead to a wide variety of lesions in the oral mucosa such as leukoplakia, edema, enanthems, mucositis, hemorrhagic crusts, change in pigmentation, ecchymosis, spontaneous bleeding, erythema, ulcers, and other symptoms (Fakhruddin et al., 2022). It is not feasible to definitively demonstrate the possible relationship between SARS-CoV-2 infection and an increased incidence of oral symptoms because of the possibility of other variables, such as environmental or individual factors (Nijakowski et al., 2022).

Although some COVID-19 patients show no symptoms, others may require intensive care and ventilation. The duration of a typical COVID-19 infection is between one and four weeks. However, in a minority of cases, individuals with acute SARS CoV-2 infections are showing a wide variety of long-term symptoms that do not resolve even after months. Approximately 30% of COVID-19 patients who were followed up for 9 months following the disease were reported with chronic symptoms. Long COVID-19, chronic COVID-19 syndrome, persistent post-COVID syndrome, post-acute sequelae of COVID-19 (PASC) or post-acute COVID-19 syndrome (PACS) are terms given to this group of patients' conditions (France and Glick, 2022; Proal and VanElzakker, 2021). Similar to myalgic encephalomyelitis and other diverse syndromes, long COVID-19 might present differently in different people in terms of the precise symptoms, severity, timing, and effect (France and Glick, 2022). Overall, patients demonstrate the following symptoms: fatigue (80%), psychological problems (34%), respiratory problems (59%), neurological symptoms (59%), difficulties in activities of daily living (ADLs) (34%), menstrual disorders, palpitations, muscle and joint pain, skin rashes, loss of smell and taste, and other symptoms (Nielsen et al., 2022).

Long COVID-19 presents with symptoms that are similar to those of other post-viral syndromes, including weakness, mental difficulties, headaches, an increased risk of venous thromboembolism, and impaired lung function (Brandini et al., 2021). In the absence of active SARS-CoV-2 replication, individuals with a long COVID-19 are no longer contagious. People with persistent COVID can be reinfected and may have a revival of early symptoms as the COVID-19 pandemic progresses and as other strains of the SARS-CoV-2 evolve and disseminate. Patients who have been immunized against COVID-19 still have the risk of contracting the virus through so-called "breakthrough" infections that can lead to chronic COVID syndrome even after the acute stage of the disease has been resolved (Brandini et al., 2021).

Furthermore, long COVID-19 may be caused by an excessive immunological response. As with other inflammatory syndromes, patients exhibit high levels of interleukin-6 and transforming growth factor- β . The CDC has also identified a multisystem inflammatory syndrome following SARS-CoV-2 acute infection that correlates with long COVID-19 and can cause hospitalization and serious symptoms. SARS-CoV-2, like other viruses, may stay dormant in the brain system, producing neurologic symptoms. Acute COVID-19 can damage organs and cause some of the symptoms of chronic COVID-19 syndrome (Brandini et al., 2021; Sampson et al., 2020). Oral inflammation can be made worse when resident oral microbiota combines synergistically with SARS-CoV-2, which can trigger an overt immune response. In response to oral infections, the body produces

a variety of inflammatory mediators such as prostaglandins, cytokines, and histamines that may have profound impacts on local cells and promote the infiltration of adaptive as well as innate immune cells, therefore boosting the immune response. About 10% of those infected with SARS-CoV2 have mild to severe symptoms, while the vast majority show no symptoms. Proinflammatory cytokines, such as TNF- α , IL-6, IL-12, IL-1, Gal-9, IL-8 and IFN- γ have been frequently found in blood /plasma and infected tissues. Cytokine storm refers to the rapid development of inflammatory cytokines that is frequently described in people with severe COVID-19 and raises the risk of chronic periodontitis, multiorgan failure and vascular hyperpermeability (Sampson et al., 2020).

Symptoms of PASC may originate from microbiome/virome dysbiosis-driven changes in epithelial barrier permeability and host signaling. The severity of the disease would increase because of the redundancy in the molecular systems associated with these processes and the activity of SARS-CoV-2. Adams et al. (2019) showed that *Porphyromonas gingivalis* along with other bacteria that enter the circulation can cause abnormally high levels of clot formation (Adams et al., 2019). Secreted substances from SARS-CoV-2 also have a role in modulating blood coagulation cascades (Adams et al., 2019).

The severity of COVID-19 has been associated with an inflammatory response, according to a previous study which was conducted in Egypt. Particularly for individuals with cardiovascular illnesses, the researchers expected that poor oral health status would be associated with elevated COVID-19 severity. It was shown that poor oral health was inversely related to COVID-19 severity, as well as to the length of recovery time and to C-reactive protein (CRP) levels. Increased CRP values and longer times to recover were seen in individuals with cardiac conditions whose poor health was poor (Abubakr et al., 2021; Cardiology, 2021).

Recurrent herpetic lesions are presented in the case reports of COVID-19 patients. It is generally established that human herpesviruses (HHV) reactivation can be triggered by inflammation, stress, and corticosteroid usage. It is hypothesized that HHV can recur and function synergistically with bacterial etiological factors to exacerbate oral diseases. Indeed, HHV members increase in several oral illnesses such as periapical periodontitis, peri-implantitis, periodontitis, inflamed pulp, and so on. If COVID-19 infection in the tissues and oral mucosa provides an ecological niche for opportunistic viral and/or bacterial infections is uncertain, however, symptoms suggest a slight alteration in some pathogens (Brandini et al., 2021). A current metagenomic investigation of COVID-19 individuals showed unusually high bacterial readings of *Prevotella intermedia* and other common microorganisms (such as *Treponema*, *Fusobacterium*, *Streptococci*, and *Veillonella* species) involved in oral disease initiation and progression (Chakraborty, 2020). Coinfected animals with the periopathic bacteria *P. intermedia* and *Streptococcus pneumoniae* had a high mortality rate, severe bacteraemia, and elevated levels of inflammatory cytokines. *P. intermedia* also boosted *Streptococcus pneumoniae* adherence within human alveolar epithelial cells A549 (Nagaoka et al., 2014). These findings highlight the potential role of periopathic bacteria in exacerbating the systemic respiratory illness symptoms that are characteristic of COVID-19. It has been proven that better dental health reduces the likelihood of contracting acute viral respiratory infections such as pneumonia (Brandini et al., 2021).

In conclusion, Recent evidence supports the existence of the virus in the oral cavity, which includes periodontal tissues and saliva and relates to the presence of viral targeted receptors within these tissues. Based on the aforementioned findings, a relationship between the oral microbial environment and COVID-19 has been identified, with unsuitable oral hygiene considered one of the most important risk factors for severe COVID-19. To minimize the risk of bacterial superinfection and the bacterial burden in the oral cavity, oral hygiene must be preserved, if not enhanced, during a SARS-CoV-2 infection. Heart disease hypertension and Diabetes patients with COVID-19 infection require special dental health precautions.

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