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LETTER TO THE EDITOR

Vascular events induced by SARS-CoV-2 and their impact in oral health

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Some works have called attention to the occurrence of oral vascular abnormalities and Kawasaki disease-like symptoms in patients infected with SARS-CoV-2 (Martelli Júnior et al. 2021a). To our understanding, most of these vascular alterations and oral manifestations are resulting from complex physiological cascades whose pathogenesis still require further elucidation. Due to the hematogenic nature of COVID-19, we highlight other alterations as relevant as Kawasaki disease that can affect the oral cavity in diverse ways and add important contributions to current gaps of the literature.

A remarkable characteristic observed in histological tissues of patients with COVID-19 is the presence of thrombosis. It has been discussed that the new coronavirus causes thrombosis in response to endothelial damage, mainly, as immediate consequence of its proinflammatory and prothrombogenic action (Cruz Tapia et al. 2020). Through its mutagenic profile, SARS-CoV-2 is able to activate the pathways involved in antiphospholipid syndrome, that may affect the behavior of salivary glands in some situations (Basu et al. 2019, El Hasbani et al. 2020). Indeed, disorders in salivary glands including cases of xerostomia and Sjogren's syndrome have been observed in patients with COVID-19 (Martelli Júnior et al. 2021b).

Antiphospholipid syndrome is recognized by causing blood clots inside arteries, veins, and organs, and despite that several cases have been reported in SARS-CoV-2- positive patients, it is not an exclusivity of COVID-19 (El Hasbani et al. 2020). As in Kawasaki disease, antiphospholipid syndrome may appear associated with vasculitis and provoke hemodynamic unbalance in the carotid artery (Svenungsson & Antovic 2020).

It has been demonstrated in rabbits that reduced arterial blood supply in the carotid artery may directly impact the functional activity of salivary glands (Stamers et al. 2010). The intimate contact that the glandular parenchyma maintains with the blood flow allows plasma extraction for salivary synthesis. Besides, recent investigations have demonstrated that saliva is a potential source for contamination by SARS-CoV-2, and high replications rate of the virus trigger the local inflammatory response that, occasionally, could lead to damage acinar cells provoking subsequent fibrotic scarring and impairing organ functionality (Mercer & Chambers 2013, Basu et al. 2019, Matuck et al. 2021).

Coincidentally, the presence of the new coronavirus in salivary gland tissues has been confirmed through immunohistochemistry showing high positivity for ACE2 receptors in both biopsy and necropsy samples (Matuck et al. 2021).

As vasculitis is a common inflammatory process for both Kawasaki disease and COVID-19, it could be assumed that most of the oral alterations associated to SARS-CoV-2 are linked to this hematogenous disorder. Consequently, this event reduces blood flow and may result in occasional formation of ulcers and local ischemia (Shanmugam et al. 2017).

Therefore, we emphasize the need for a critical eye to oral findings in COVID-19 patients that can constitute pathognomonic features of the disease. Moreover, we highlight the role of the virus as the primary agent in the pathogenesis of these disorders as well as its capacity of activate mechanisms and pathways until then "silenced".

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