

Finding the Link Between Increased Oral Cancer Cases and Covid 19 Pandemic

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The coronavirus disease 2019 (COVID-19) is the newest respiratory contagious disease sweeping the globe rapidly [1]. The virus responsible for it is the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The disease quickly spread over the world after the first case was detected in Wuhan, China, in December 2019, and the WHO labelled it a public health emergency of international concern (PHEIC) and a pandemic in March 2020 [2]. By November 17, 2020, COVID-19 has caused almost 54 million confirmed cases and 1,324,249 deaths [3].

Comorbidities such as cerebrovascular disease, diabetes, coronary heart disease, and hypertension have been linked to an increased risk of fatal result following viral infection [4]. Patients with cancer or those receiving treatment are immunocompromised and are more susceptible to COVID-19, which has been linked to poorer outcomes. Patients with a history of cancer were also found to be more likely to develop COVID-19 as a result of their chronic immunosuppressive status, and to have a poor prognosis [5,6].

Because the ACE-2 receptor and the furine enzyme are upregulated in tobacco-induced oral cancer patients, they are more likely to contract COVID-19. Nicotine chemicals upregulate ACE2 receptors in the oral mucosa via the 7 acetylcholine receptors (7-nAChR) in tobacco-addicted oral cancer patients. The Ribosomal protein S3 (RPS3) and SRC genes have been demonstrated to increase viral gene replication, viral assembly, and entrance into host cells when ACE2 receptors are increased. The upregulation of T-cell-mediated responses (T-helper-2), cytotoxic reactions and neutrophil inflammation in oral cancer patients could be explained by an increase in ACE 2 receptor expression. Tobacco users may increase inflammation-induced damage in malignant oral mucosa infected with the SARS-CoV-2 virus because nicotine causes abnormal T-cell cytokine gene production (i.e. primary and secondary T-cell response). Nicotine-induced T-cell dysfunction increases the chance of an abnormal immunological response in these patients. In oral cancer patients infected with SARS-CoV-2, higher production of Furine in malignant oral mucosa might promote viral fusion. Patients with tobacco-related oral cancer had increased levels of ACE 2 and Furine, indicating a greater sensitivity to the SARS-CoV-2 virus [7,8].

The COVID-19 epidemic has resulted in a number of lifestyle and behavioural changes that can have a significant impact on the incidence, identification, and treatment of oral cancer. Treatment delays meant to reduce COVID-19 transmission are simultaneously causing increasingly severe oral cancer presentations at the time of diagnosis. As a result, in the setting of oral cancer, health care practitioners must be cautious in screening for these risk factors and lifestyle modifications [9]. Across the country, comprehensive guidelines should be implemented based on a coordinated three pronged approach that encompasses prevention/education, service delivery and research [7].

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