Oral Microbial Co-infection in Patients with Coronaviruses

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Abstract

A global epidemic of coronaviruses has caused a negative effect on human health. The microbial domain linked with the SARS-CoV-2. The co-infection of the SARS-CoV-2 with other microbes is an actual vital issue in coronaviruses disease, which might complicate the precise detection, handling, prediction and increase the death rates. Local and systemic illnesses are linked to the oral cavity. This review shows the role of co-infection in COVID-19.

Introduction

The first case of the novel coronaviruses was observed in China in December of 2019 (1). The family Coronaviridae includes the genera Beta-coronaviruses to which this virus belongs. Acute respiratory distress syndrome (ARDS) and multiple organ failure can be caused by the rapid release of multiple cytokines into body fluids due to SARS-CoV-2 infection (2, 3). Hundreds of thousands of individuals have life-threatening outcomes not only in medically compromised persons, but also in perfectly healthy young individuals with an immune system. The coronaviruses must have special abilities to spread and compromise the immune mechanisms in humans (4).

There is evidence that supports the excessive incidence of co-infections among COVID-19 patients. One of the reasons for high mortality in COVID-19 is the fact that the immune system is not functioning properly. Reactivation of herpes viruses in patients is evidence of its association with an immune disorder (5) Poor oral hygiene is prepare to be a prime ecological pressure that leads to the development of dysbiosis. An increase in the propogation of oral microbes is the outcome of ecological transfers. Daily activities inclusive of mastication, flossing and tooth brushing can cause bacteraemia, which can cause inflammation in few patients. People with periodontitis appeared to be more likely to develop bacteraemia because of the sulcular epithelia and damaged gum tissues (6).

Bacterial and Fungal Co-infection in SARS-CoV-2

In severely unwell sufferers, bacterial co-infection is not unusual (7). There are between 11 and 35 percent cases of primary co-infection or secondary bacterial pneumonia among patients with respiratory viruses. The negative bacilli and Candida are in particular the most common varieties of microorganism in patients

with the disease (8, 9). However, there is an occurrence of microbial joint infection. A retrospective case series study found that the joint infection rate in all 221 patients is 7.7 percent. The co-infection rate is 3.2 percent (10).

Acinetobacter baumannii, Klebsiella pneumoniae, and Candida albicans are some of the co-infecting microorganisms reported in the ninety-nine patients of novel coronaviruses in China (11). The immune system can be damaged by the disease, especially B cells, T cells, and NK cells. The lower of immune function can be the principle purpose for the co-contamination (12, 13). The death rate in severe cases is greater than in the non-severe group because of the higher co-infection rate (14). Moreover, the co-infection was related with a 2.5-fold increase in the risk of death in the disease indicates that there is a positive interplay between the two (15).

.Patients with COVID-19 have frequently reported high reads of cariogenic and periodontopathicbacteria. Prevotella intermedia, Fusobacterium nucleatum, and gingivalis support the idea of a connection between the oral microbiome and COVID-19 (16). Evidence shows that periodontopathicbacteria are involved in the pathogenesis of respiratory diseases and are associated with chronic inflammatory systemic diseases. There is an increased risk of death and serious injuries from these diseases (17).

In recent Iraqi study, qPCR process was successfully utilized to determine and count *P. gingivalis* in saliva and blood specimens. The result showed that *P. gingivalis* was determined in COVID-19 patients and healthy subjects with significantly higher detection rate in saliva of the patients. Moreover this study demonstrated that salivary level of bacteria in patients increases in number with the disease severity, which indicates that bacterial infections contribute to the spread of the disease (18). Other local study found that the total viable count of salivary bacterial flora was significantly higher in patients group than to the COVID-19 free group (19). However, another study showed that there was non-significant difference in total viable count of salivary *Mutans streptococcus* between COVID-19 group and controls free group, while total viable count of salivary *candida* there was high significant difference between two groups (20).

However, Soffritti et al., (21) found that the bacterial composition appeared to be diverse between the two groups. A topobium was increased in COVID-19 compared to controls, and the relative abundance of the bacterial genera Streptococcus, Veillonella, Prevotella, Lactobacillus, Capnocytophaga, Porphyromonas, Abiotrophia and Aggregatibacter was increased. Also, Herrera et al. (22) has been established that in periodontitis sufferers, a bacterial-viral synergy can encourage penetration of the disease, such an interaction can assist viruses keep away from the immune reaction, consequently permitting its front to gingival capillaries and endovascular transmission immediately to the pulmonary vessels (23).

Viral Co-infection in SARS-CoV-2

Viral co-infection might also have a tremendous have an effect on remedy and prognosis of the disorder. The need for a higher level of care, increased length of

stay and development of ARDS are connected with co-infection (24). Many clinical studies have shown that the co-infection with other viruses is common in respiratory diseases (25).

Respiratory syncytial virus, human rhinoviruses, human metapneumoviruses, parainfluenza virus type 2 and human CoVs were all simultaneously detected. The data is consistent with a study that found entero/rhinoviruses and non-SARS-CoV-2 are the most common co infections (26). Co-infection with other respiratory viruses may be an important reason for the early misdiagnosis of COVID-19 (27).

In (2021) Adham and AL-Ghurabi (28) revealed that there is significant increase in the levels EBV IgG and CMV IgM among COVID-19 patients compared with controls. There were no differences in the levels of anti-viral antibodies in patients with good and bad oral hygiene. So, they concluded that a higher level of the IgG in patients pointing to that it is more common in patients. An increase in the percentage of CMV IgM pointing to reactivation of latent infections is related to the severity of the disease suggesting that COVID-19 can motive cellular immune disorder.

The exacerbation of COVID-19 may be related to oral hygiene. Populations that have suffered high mortality rates from the disease such as northern Italy, China and Spain have high rates of CMV sero-positivity (29). People from lower socio-economic groups seem to have higher mortality rates from the disease (30).

The poor effect of CMV on immune function in older humans might explain the correlation between the chance of mortality from SARS-CoV-2 and growing age is. The event of reminiscence inflation' is related to the gradual accumulation of CMV-specific effector and memory cells (31). The reduction in the naive T cell pool is associated with this (32). The naive T cell pool can be attritioned by CMV by 20 years and it is exactly this naive repertoire that will be required for generating adaptive immune responses against a novel virus (33). Recently, the pathological record of the COVID-19 deceased patient recommended that there was over-activation of T cells, which result in severe immune injury in the COVID-19 cease (34). There are similar symptoms such as fatigue, myalgia, and sore throat, which are indicative of a potential association (35).

It has been found that COVID-19 patients who had a positive test for the EBV had a higher risk of having a symptom than those who did not. The result refers a co-infection of the two viruses in patients (36). C-reactive protein is also produced in infections (37). There was a strong inflammatory response in the COVID-19 patients who had higher C-reactive protein than the patients who did not. Several reports have claimed that the severe group had higher levels of C-reactive protein than the non-severe group (34, 38).

Conclusion

The correlation of co-infections with the severity of the disorder indicated that

COVID-19 could cause a defect in immunity. The review shows that the oral hygiene may play a role in the exacerbation of COVID-19.

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