Original Research Paper

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Subgingival Plaque as a reservoir for SARS COV-2 VIRUS. A Cross sectional study.

Abstract:

Aim: To evaluate the presence of SARS COV-2 virus in subgingival plaque in previously infected patients diagnosed with Chronic Generalized Periodontitis.

Material & Method: Subjects with chronic generalized periodontitis who had suffered from COVID-19 Disease (both male and female) in age group of 18-65 yrs total 100 patients were included in the study . Patients diagnosed with systemic illnesses,antibiotics/antiviral drug usage in past 6 months and undergone scaling were excluded. Subgingival plaque samples were collected with the help of curette by following sterilization guidelines. Collected samples were sent for Reverse transcription polymerase chain reaction (RT-PCR) to determine the presence of SARS-COV-2 virus.

Results: RT-PCR results showed no sample positive for SARS-CoV-2 virus in subgingival plaque samples of patients who had COVID-19 disease in past 6 months.

Conclusion: Within the limitation of the study, we conclude that Subgingival Plaque from previously infected COVID-19 patients doesn't harbour SARSCoV-2 RNA and it might not be a potential reservoir with an essential role in COVID-19 transmission.

Key-words: Covid 19, SARSCoV-2,Subgingival Plaque

Introduction:

Coronavirus disease 2019 (COVID-19), caused by the SARS-CoV-2 virus, has affected millions of people worldwide, constituting one of the most challenging public health issues in human history.[1] Being a never before encountered pathogen, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has afforded no luxuries to those attempting to build knowledge in the face of this continuously mounting challenge. Various interventions—such as social distancing, face masks, hygiene measures, massive diagnostic efforts, contact tracing and quarantines—remain the best option to try to interrupt the spread of the virus, thus lowering the risk of contagion between people until there are enough vaccines to prevent further spread of the virus (Lewnard & Lo, 2020; Wiersinga et al., 2020).[2,3]

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Understanding the sites within the human body capable of harbouring SARS-CoV-2 RNA is crucial for understanding the virus's points of entry and reducing its spread. A meta-analysis evaluating the presence of SARS-CoV-2 RNA in different clinical samples detected viral RNA in samples from nasopharynges and oropharynges, secretions from the lower respiratory tract, bronchoalveolar lavage fluid, rectal swabs, blood and faeces (Bwire et al., 2021).[4] The literature also

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identifies saliva as one of the most critical routes of interindividual SARS-CoV-2 transmission.[5,6,7] (Cheng et al., 2020; Li et al., 2020; Wang et al., 2020), and viral RNA was recently observed in the salivary glands.[8] (Huang et al., 2020). However, saliva may not be the only intra-oral niche capable of harbouring viruses. Therefore, it is essential to investigate the presence of SARS-CoV-2 RNA in other intra-oral sites. The teeth, gingival sulcus, tongue, cheek mucosa, hard and soft palates, and tonsils are all niches that harbour several types of microorganisms.[9,10] (Dewhirst et al., 2010; Teles et al., 2013), including viruses.[11,12] Gingival sulcus, a well-established microbial niche has been associated with respiratory diseases.[13,14] Enzymes such as mannosidase, fucosidase, hexosaminidase, and sialidase released by the gingival sulcus microbes, can modulate the respiratory surfaces, promoting microbial colonization.[2] These gingival microbes can also cause loss of fibronectin from oral, oropharyngeal and respiratory mucosa, increasing adherence of microorganisms.[13,15]

Recently, SARS-CoV-2 RNA was detected in gingival crevicular fluid.[16] (Gupta et al., 2021). In addition, the tooth structure and adjacent tissues might represent a potential virus-harbouring site in the context of the present pandemic. According to the literature, viruses from dental biofilm may also infiltrate the bloodstream (Slots, 2015).[11] SARS-CoV-2 depends on the presence of angiotensin-converting enzyme II (ACE2) receptors—which exhibit high expression on the epithelial cells of the oral mucosa (Xu et al., 2020).[17]—to attach, enter and multiply, leading to infection. Alternatively, it can also remain at the virion stage (i.e. a complete viral particle constituting an infective form of a virus outside the host cell; Lodish et al., 2000).[18]

A few studies based on patient data have also been published, which generally point towards periodontal disease as a determinant of poorer COVID-19-related outcomes. In recent studies, SARS-CoV-2 was detectable in junctional epithelium, adjacent oral epithelium, and underlying connective tissue of COVID-19 patients. The plaque biofilm on the non-shedding tooth surface harbors micro-organisms in a complex environment that can resist host immune response and other pharmaceutical agents unless it is mechanically disrupted.[19,20] The microorganism can detach from the plaque biofilm and gain systemic entry during the process of plaque maturation. Furthermore, the process of biofilm formation and maturation presupposes a detachment phase, during which microorganisms can travel to other parts of the human body (Talsma, 2007).[21] Hence, the present study was conducted to evaluate the presence of SARS COV-2 virus in subgingival plaque in previously infected patients diagnosed with Chronic Generalized Periodontitis.

Material and methods:

This cross-sectional study was conducted at the Department of Periodontics, RUHS College of Dental Sciences and Hospital, Jaipur between November 2023 to February 2024 to evaluate the presence of SARS COV-2 virus in subgingival plaque in previously infected patients diagnosed with Chronic Generalized Periodontitis who were not admitted in any healthcare institute and home quarantined. The study protocol was approved by the Institutional Ethics Committee (IEC), and was conducted in accordance with the Helsinki criteria.[22] A prior written informed consent was obtained from the study participants.

Inclusion criteria:

100 Subjects with previous infection with SARS COV-2 virus in last 6 months to one year with chronic generalized periodontitis (Stage II& grade B) in age range between 18 and 65 years not undergone scaling/professional oral prophylaxis after SARS-COV-2 Infection reporting to the department were included in the study.

Exclusion criteria:

Patients diagnosed with systemic illness, using antibiotics/antiviral in past 6 months. Infected with immunodeficiency virus, acquired immunodeficiency syndrome or any co-morbid illness & lactating or pregnant patients were excluded from the study.

The patient's demographic data, medical history& severity of COVID-19 infection was recorded using a structured proforma by a single investigator to prevent bias. Plaque index, gingival index were recorded to assess their oral hygiene status. A thorough periodontal examination was conducted & periodontal parameters recorded were clinical attachment level (CAL) 3-4mm& probing pocket depth (PPD) $\leq$ 5 mm.

Subgingival plaque samples were collected using Gracey curettes from the all four quadrants following adequate sterilization guidelines (Figure 1). All the samples were immediately transferred into a viral transport medium (Fig.

2) and were sent for Reverse transcriptase real-time qualitative polymerase chain reaction (RT-PCR) (Fig.3)for the presence of SARS-CoV-2 RNA. Data analysis was carried out using the SPSS 18 (IBM) software.

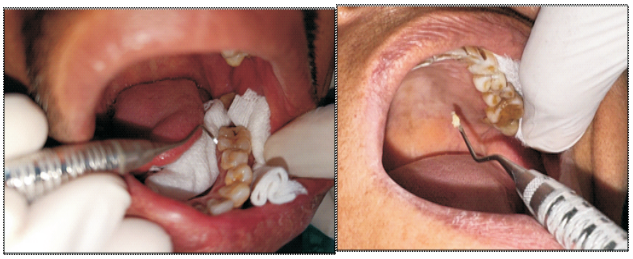


Figure1: Subgingival plaque sample collection from periodontal pockets

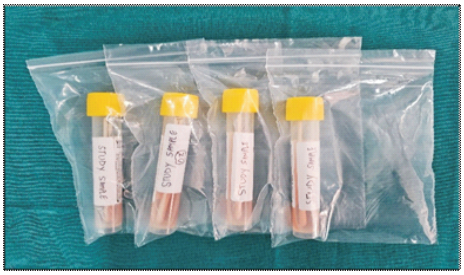


Figure 2 : Collected plaque sample transferred vials containing viral transport medium

Figure 3: Qualitative polymerase chain reaction equipment

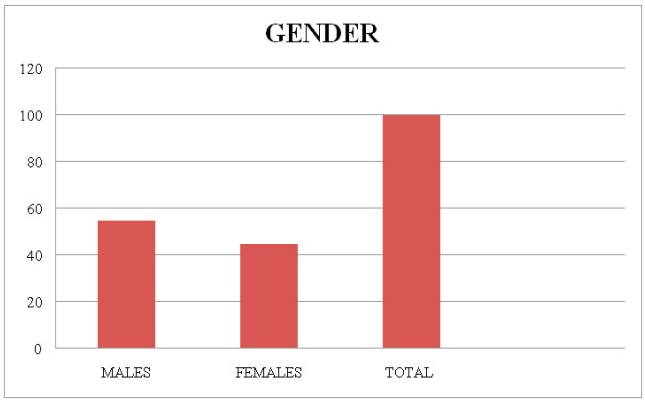
Results:

A total of 100 subjects were recruited for the study out of which 55 were male and 45 were female. (Fig. 4) The demographic details are presented. The mean plaque index was 2.20 ± 0.36 ,mean Gingival Index was 1.61 ± 0.22. The mean PPD was 3.47 ± 0.52mm of the subjects.

The plaque samples were assessed for the presence of SARS-CoV-2 RNA using RT-PCR, and the results were negative for all the samples, based on cycling threshold values that were considered negative if more than 35 according to the Indian Council of Medical Research (ICMR) guidelines.

Figure 4- Demographic Data

	Mean =SD
Age	40.5 = 7.63 years
Gender (percentage)	Male – 55 (55%) Females – 45 (45%)
Plaque Index	2.20 = 0.36
Gingival Index	1.61 = 0.22
PPD	3.47 = 0.52mm
CAL	3.47 = 0.52mm



Discussion:

Gingival sulcus has also been implicated as a favored site for the proliferation of pathogenic viruses like Human papillomavirus and Human herpesviruses 6 and 7[23-26]. Symbiotic relationship between gingival microbes could be a potential reason for the high viral proliferation in the gingival sulcus. It is presumed that the virus modulates the local environment aiding in the retention of bacterial colonies, which in turn aid in triggering the reactivation of viruses[27].

A previous study has suggested that the SARS-CoV RNA has been reported to be present in the saliva even before pulmonary changes were detected.[28] This underlines the potentially strong correlation between viral presence in the oral cavity and the development of respiratory pathologies. Angiotensin-converting enzyme 2 (ACE2), the major receptor for the SARS-CoV2 virus is present in many tissues in the body, including the epithelium of the salivary glands and gingival.[29,30] Thus, considering the conducive symbiotic local microbial environment, and the presence of ACE2 inhibitors, it can be hypothesized that the gingival

sulcus is a potential ecological niche for the SARS-CoV-2 virus. A recent publication has reported salivary SARS-CoV-2 positivity in a patient who was in convalescence.[31] In such cases, it is plausible that the positive result could be at least partly be attributed to the SARS-CoV-2 viral load in the gingival crevicular fluid. To confirm the hypothesis, future studies must assess the presence of SARS-CoV-2 in the gingival crevicular fluid and salivary samples of COVID-19 patients both during the disease progression and post-recovery. It is plausible that even in patients in convalescence the SARS-CoV-2 could be detected in the saliva/gingival fluid.

The simple presence of SARS-CoV-2 in the dental biofilm, irrespective of the viral load, may define the oral cavity as a potential reservoir for the virus. A study by Gomes et al.[32] hypothesized that dental biofilm might be a potential reservoir for SARS-CoV-2 RNA in symptomatic COVID-19 subjects and could have an essential role in COVID-19 transmission. In the future, researchers should always consider the oral cavity's role in the transmission of SARS-CoV-2.[33] (Herrera et al., 2020), pay close attention to the richness of ACE2 receptors at the dorsum of the tongue.[29] (Xu et al., 2020), and continue to define colonization pathways and mechanisms.

The present study result shows an absence of SARS-CoV-2 RNA in subgingival plaque samples of patients who recovered from COVID-19 infection in the last 6 months. A conceivable reason could be that the study population involved subjects with mild and moderate COVID-19 infections. Hence, the viral load could be potentially less as it correlates with disease severity.[34] It included the sample population who completely recovered from the infection, currently presenting with no related symptoms, and have been vaccinated against COVID-19 infection.

To date, there have been very few recorded cases of viral transmission and infection in the dental clinic setting. In November 2020, Estrich and colleagues reported a prevalence rate of 0.9% among dentists in the United States, based on a cross-sectional study that included over 2000 practising dentists.[35] The CDC guidelines highlight the precautions to be taken regarding aerosols generated during prophylactic procedures for cleaning gingival crevices.[36,37] Hence, it is emphasized that there is a need to formulate safe therapeutic strategies to reduce the overall oral SARS-CoV-2 load.

Conclusion:

Dental plaque was not observed to be a potential reservoir,of SARS-CoV-2 virus in the subgingival plaque sample of patients recovered from COVID-19 infection. Hence, all necessary personal protective measures must be taken by the clinicians while treating the patients recovered from COVID-19 infection. A change in the subgingival environment must be anticipated, leading to alteration in periodontal disease progression. Hence, a modification in periodontal maintenance care is suggested.

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