



**QUALITY IMPROVEMENT IN
DENTAL AND MEDICAL
KNOWLEDGE, RESEARCH, SKILLS
AND ETHICS FACING GLOBAL
CHALLENGES**

Edited by
Armelia Sari Widyarman, Muhammad Ihsan Rizal,
Moehammad Orliando Roeslan & Carolina Damayanti Marpaung



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Comparison of periodontal disease severity in COVID-19 survivors and non-COVID-19 individuals

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ABSTRACT: In December 2019, SARS-CoV-2 was detected, causing a considerable amount of people diseased. Around ten to twenty patients experienced post-COVID-19 symptoms that persevered, surpassing three weeks after the initial infection, which then was referred to as long-COVID. Long-COVID symptoms primarily are prolonged symptoms encountered in the acute phase, but there is a high potential of other unidentified long-COVID symptoms. To this point, clinical studies regarding the effects of COVID-19, particularly in regard to the long-term, are still scarce. This study is carried out to discover the long-term effects of COVID-19 on gingivitis and periodontitis. This study included 40 subjects, consisting of half of COVID-19 survivors (diagnosed by a history of at least one positive PCR result) and the rest were non-COVID-19 survivors, each group consisting of 10 gingivitis patients and 10 periodontitis patients. The COVID-19 group filled out a questionnaire to discern long COVID (at least one persisting symptom 4 weeks to 6 months after the initial infection). A complete periodontal examination (probing depth, bleeding on probing, simplified oral hygiene index, clinical attachment loss, and panoramic examination) was done on all participants, and data analysis was performed to discern gingivitis and periodontitis severity between each group. No significant difference was found in the distribution of gingivitis (localized/generalized) between COVID-19 survivors and non-survivors ($p>0.05$). There was also no significant difference in the distribution of periodontitis (localized/generalized) between COVID-19 survivors and non-survivors ($p>0.05$). Similarly, this study also showed that there were no differences in periodontitis staging and grading between COVID-19 survivors and non-survivors ($p>0.05$), as well as there was no significant difference between COVID-19 survivors and non-survivors' periodontal clinical parameters. Accordingly, this pilot study has excluded long COVID impact on gingivitis and periodontitis severity measured by clinical parameters.

1 INTRODUCTION

In December 2019, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was detected in China and spread throughout the world, causing 565.207.160 people diseased with 6.373.739 deaths according to WHO data as of July 22nd, 2022 at 7.12 PM CET (Lopez-Leon *et al.* 2021; Organization 2022) Around 10%–20% patients experience symptoms that persist surpassing 3 weeks after SARS-CoV-2 infection (Ladds *et al.* 2021). This incident is then referred to as long COVID-19 or post-COVID-19 syndrome (Burhan *et al.* 2020; Maltezou *et al.* 2021).

The most common manifestations of long COVID are fatigue, pain, headaches, joint pain, anosmia, ageusia, and so forth. Pathogenesis of post-COVID-19 syndrome is still undetermined, but existing studies show that prolonged inflammation contributes to most

post-COVID-19 syndrome manifestations (Maltezou *et al.* 2021). Increased laboratory parameters among others are interleukin-6 (IL-6), procalcitonin, serum ferritin, C-reactive protein (CRP), N-terminal (NT)-pro hormone BNP (NT-pro BNP), and D-dimer (Lopez-Leon *et al.* 2021).

Cytokine storm (inflammation response mediated by uncontrolled cytokines) was first found in 1980, and was found to be caused by infection or certain drugs. Infected cells activate leukocytes, natural killer cells, macrophages, dendritic cells, and monocytes, causing inflammatory cytokines release, which then activates a large sum of leukocytes. Cytokine storms occur locally after primary infection, then spread throughout the body through systemic circulation, causing classic signs of inflammation. After a cytokine storm occurs, a repair process begins and could either restore organ function or cause fibrosis which is characterized by persistent organ malfunction. Studies found that cytokine storm is closely related to uncontrolled inflammation and incline in mortality attributed to elevated pro-inflammatory cytokines, among others found in COVID-19 patients were interferons (IFN), tumor necrosis factor (TNF), and interleukin (IL) groups (Ladds *et al.* 2021).

Gingivitis is an inflammatory disease which cause is attributable to the accumulation of dental biofilm, distinguished by the presence of gingival redness and edema without loss of periodontal attachment (Trombelli *et al.* 2018). A condition is defined as gingivitis if there is a $\geq 10\%$ bleeding on probing (BOP) with a $\leq 3\text{mm}$ probing depth. Based on the extent of the disease, gingivitis can be divided into localized and generalized. Localized gingivitis occurs when there are 10%–30% of BOP and generalized gingivitis occurs when the BOP exceeds 30% (Chapple *et al.* 2018).

Periodontitis, on the other hand, is an inflammatory dental supporting tissue disease caused by infection by certain microorganisms, consequently prompting periodontal ligament and alveolar bone destruction to a greater extent, accompanied by pocket formation, gingival recession, or both. (Kodir *et al.* 2014; Könönen *et al.* 2019) Periodontitis customarily originates from gingivitis, but not all gingivitis advances into periodontitis. Plaque accumulation at the gingival margin triggers an inflammatory response that causes gingival inflammation, where chronic inflammation of the gingiva can progress to periodontitis. The most distinguishable mark of periodontitis is osteoclastogenesis activation which then causes alveolar bone destruction and loss of attachment (Könönen *et al.* 2019). Periodontitis is classified into three forms: necrotizing periodontal disease, periodontitis as a manifestation of systemic disease, and periodontitis. The classification for periodontitis is further characterized by a staging and grading system.

Data shows that around $>80\%$ of COVID-19 patients experience one or more persisting symptoms two weeks after the acute infection phase. Most of the symptoms are the continuation of symptoms experienced in the acute phase (old symptoms), but there is still a big possibility pertaining to the appearance of other unidentified symptoms (Lopez-Leon *et al.* 2021). The presence of an increased similarity of pro-inflammatory cytokines in periodontal inflammation and COVID-19 suggests a possible association between the two diseases (Shrestha *et al.* 2017).

COVID-19-induced cytokine storm is similar to cytokine imbalance transpiring in periodontitis, so it is assumed that there is a possible relation between COVID-19 and periodontitis complication (Martu *et al.* 2020). This proposition was further supported by a study conducted by Anand *et al.*, in which they found that gingival bleeding, dental plaque, probing depth, mobile teeth, and clinical attachment loss were more frequently noted in COVID-19 patients. These findings prompt COVID-19 patients' awareness of maintaining periodontal health and oral hygiene (Anand *et al.* 2022). The diagnostic parameters previously stated (probing depths, bleeding on probing/BOP, clinical attachment level, plaque index) are useful for dentists, in connection to periodontal disease type, location, and severity, as well as a basis for periodontal disease treatment planning and monitoring (Taba *et al.* 2005). To date, there are few clinical studies on the effect of COVID-19 on periodontal disease, especially regarding the long-term effects of COVID-19. One case-control study by Anand *et al.*

discovered an interrelation between periodontitis and COVID-19, in which bleeding and dental plaque accumulation were found to occur more often in COVID-19 patients (Anand *et al.* 2022). Furthermore, a study in 2020 also found that some COVID-19 patients manifested generalized erythematous and edematous gingiva, necrotic interdental papillae in both maxilla and mandible, with gingival sulcus bleeding, and no attachment loss detected (Gofur 2020). This research is done to ascertain the long-term effect of COVID-19 on gingivitis and periodontitis.

2 RESEARCH METHOD

The present study was carried out between September 2021 and January 2022 in RSGM FKG Trisakti University, Jakarta, and was approved by the Ethical Committee (Number 014/S3/KEPK/FKG/10/2021). Subject recruitments were obtained from patients visiting the researcher's private dental practice and RSGM FKG Trisakti. This pilot study included 40 participants aged 21–66 years, consisting of 13 males and 27 females who did antigen swabs.

2.1 Inclusion and exclusion criteria

The inclusion criteria were applied as follows: (1) gingivitis patients with a probing depth of 3 mm and BOP score of $\geq 10\%$ (Chapple *et al.* 2018), (2) periodontitis patients with clinical attachment loss (CAL) on ≥ 2 non-adjacent teeth or ≥ 3 mm CAL at buccal/oral surface on ≥ 2 teeth (Chapple *et al.* 2018), (3) subjects with a minimum number of 20 teeth (Gokul 2012), (4) moderate to severe oral hygiene index-simplified (OHI-s) with a score of 1,3-6,0 (there is no significant difference between COVID-19 survivors and non-survivors), (5) COVID-19 survivors with persisting symptoms for 4 weeks up to 6 months following the first time symptoms appeared (Nalbandian *et al.* 2021), (6) survivors with a minimum of one persisting symptoms, among others: coughing, breathlessness, anosmia, ageusia, headaches, body pain, diarrhea, nausea, fatigue, and cognitive decline (Burhan *et al.* 2020), and (7) patients have not been vaccinated after COVID-19 recovery. We excluded patients who were active smokers, patients with type II diabetes, breastfeeding/pregnant women, subjects who ever received periodontal treatments during the pandemic, and patients with grade C periodontitis (Gokul 2012).

2.2 Subject division

Before commencing the study, all of the participants agreed to written informed consent. The 40 participants were divided into two groups: COVID-19 survivors and non-survivors. Each group encompassing 20 subjects (10 gingivitis patients and 10 periodontitis patients). Subjects' COVID-19 histories were taken by seeing subjects' medical history, whether they had been confirmed COVID-19 positive (through PCR test) or not. COVID-19 survivors are defined as having a minimum of one persisting symptom, lasting 4 weeks up to 6 months since symptoms first appeared. (Nalbandian *et al.* 2021) The COVID-19 group filled out a questionnaire regarding COVID-19 history (time of initial illness and recovery, symptoms experienced, treatment received, and so forth). A complete periodontal examination (probing depth, BOP, OHI-s, clinical attachment loss, and panoramic examination) was performed on all participants to confirm the diagnosis of gingivitis and periodontitis.

2.3 Gingivitis subject criteria and scoring

Gingivitis is defined as gingival inflammation indicated by a probing depth of ≤ 3 mm and $\geq 10\%$ BOP score (Chapple *et al.* 2018). The examination was done using a Williams probe and mouth mirror. Probing depth is defined as the distance between the sulcus base/pocket

and gingival margin (Do *et al.* 2019). Examination of probing depth was done using a Williams probe which was inserted into gingival sulcus, with measurement results in millimeters (mm). Bleeding on probing scores indicates the proportion of gingival bleeding sites during gingival stimulation with periodontal probe (Chapple *et al.* 2018). Williams probe is inserted into the base of the sulcus in six insertion points (mesiobuccal, buccal, distobuccal, mesio lingual/mesio palatal, lingual, disto lingual/disto palatal). The score is obtained by measuring the number of bleeding gingival surfaces while probing divided by the number of all surfaces in the mouth, then subsequently multiplied by 100%. Gingivitis is considered as localized if BOP scores are within the range of 10–30%, and considered as generalized if BOP exceeds 30% (Chapple *et al.* 2018).

2.4 Periodontitis subject criteria, grading, and staging

Periodontitis is defined as periodontal tissue inflammation marked by clinical attachment loss (CAL) on ≥ 2 non-adjacent teeth or ≥ 3 mm loss of attachment on buccal/oral surfaces of ≥ 2 teeth. Clinical attachment loss is the distance between the cemento-enamel junction with the base of the pocket (the length of recession added by pocket depth). Examination of CAL is done using a Williams probe inserted into periodontal pockets, with measurement results in mm.

Based on the new 2017 classification, periodontitis is categorized into staging and grading (G. Caton *et al.* 2018). Periodontitis staging depends on the disease severity and complexity, determined by CAL, bone loss, tooth loss on radiographs, and furcation involvement (Parikh *et al.* 2020). Staging is divided into Stage I (early stage of clinical attachment loss), Stage II (development of periodontitis), Stage III (attachment apparatus damage), and Stage IV (breakdown that leads to tooth loss and the loss of masticatory function) (Babay *et al.* 2019).

Periodontal disease grading shows and considers the pace of disease progression, response toward periodontal disease treatment, and systemic impacts on periodontal disease. Grading aims to use any information available to decide disease progression possibility and respond to therapy. Grading depicts periodontitis scoring based on direct evidence (longitudinal observation such as patient's previous radiographic imaging) and indirect evidence (percentage scoring of bone loss divided by patient's age) (Tonetti *et al.* 2018). Grades A, B, and C are considered as slow rate, moderate rate, and rapid rate, respectively. Grade B is deemed as the expected rate, while grade C is a high-risk factor of periodontal disease progression (Babay *et al.* 2019) Smoking and diabetes were also considered risk factors that could alter periodontal disease staging (Parikh *et al.* 2020; Tonetti *et al.* 2018).

2.5 Data analysis

The normality of OHI-s data was analyzed by the Saphiro-Wilk test. Data analysis for OHI-s was done by using an independent t-test. Data analysis for gingivitis was done by using the Fisher test and data analysis for periodontitis was done by using the Fisher test for distribution and grade and Kolmogorov-Smirnov test for stage.

3 RESULTS

Every group has been subjected to statistical analysis of OHI-S values and there was no significant difference ($p > 0.05$). From this study, it was stated that there was no difference in the severity of gingivitis and periodontitis in survivors and non-survivors of COVID-19, which we can see from the data analysis below.

3.1 Gingivitis severity between COVID-19 survivors and non-survivors

The parameter for gingivitis severity used in this study was the disease distribution, whether the gingivitis was localized or generalized (Table 1). There was a total of 8 localized gingivitis subjects, consisting of five COVID-19 survivors and 3 non-COVID-19 survivors. There was a higher number of generalized gingivitis subjects, with a total of 12 patients, consisting of 5 COVID-19 survivor subjects and 7 non-COVID-19 survivor subjects. There was no significant difference in the distribution of gingivitis survivors and non-survivors ($p>0.05$).

3.2 Periodontitis severity between COVID-19 survivors and non-survivors

The parameters for periodontitis severity used in this study were: distribution, staging, and grading. Periodontitis distribution was also based on whether it is localized or generalized (Table 2). A total of 7 subjects had localized periodontitis: 3 COVID-19 survivors and 4 non-COVID-19 survivors. Similar to gingivitis subjects, a higher number of generalized periodontitis was observed, with a total of 13 subjects, consisting of 7 COVID-19 survivors and 6 non-COVID-19 survivors. Data showed that there was no significant difference in the distribution of periodontitis between COVID-19 survivors and non-survivors ($p>0.05$).

Table 1. Distribution of localized and generalized gingivitis among COVID-19 survivors and non-survivors.

| | | Gingivitis | | | | P |
|-------|----------------|------------|-----|-------------|-----|-------|
| | | Localized | | Generalized | | |
| Group | COVID Survivor | 5 | 50% | 5 | 50% | 0.141 |
| | Non-Survivor | 3 | 30% | 7 | 70% | |
| Total | | 8 | 40% | 12 | 60% | |

Table 2. Distribution of localized and generalized periodontitis among COVID-19 survivors and non-survivors.

| | | Periodontitis | | | | P |
|-------|----------------|---------------|-----|-------------|-----|------|
| | | Localized | | Generalized | | |
| Group | COVID Survivor | 3 | 30% | 7 | 70% | 1.00 |
| | Non-Survivor | 4 | 40% | 6 | 60% | |
| Total | | 7 | 35% | 13 | 65% | |

3.3 Periodontitis staging between COVID-19 survivors and non-survivors

Subjects with periodontitis were distributed into four stages (Table 3). There was a higher number of subjects in Stage II category compared to other stages. Stage I consists 4 subjects (2 COVID-19 survivors and 2 COVID-19 non-survivors), Stage II consists of 11 subjects (5 survivors and 6 non-survivors), Stage III consists of 4 subjects (2 survivors and 2 non-survivors), and lastly Stage IV consists of only 1 subject (1 COVID-19 survivor). Statistical analysis showed that there was no significant difference between periodontitis staging between COVID-19 survivors and non-survivors ($p>0.05$).

3.4 Periodontitis grading between COVID-19 survivors and non-survivors

Periodontitis subjects were also assessed by their grading, also based on the new 2017 classification (Table 4). There was a higher number in grade B periodontitis subjects. The sum of grade A and grade B subjects, consecutively, were 7 subjects and 13 subjects. Statistical analysis also indicated that there was no significant difference in periodontitis grading between COVID-19 survivors and non-survivors ($p>0.05$).

Table 3. Distribution of periodontitis staging among COVID-19 survivors and non-survivors.

| | | Periodontitis | | | | | | | | p |
|-------|----------------|---------------|-----|----------|-----|-----------|-----|----------|-----|------|
| | | Stage I | | Stage II | | Stage III | | Stage IV | | |
| Group | COVID Survivor | 2 | 20% | 5 | 50% | 2 | 20% | 1 | 10% | 1.00 |
| | Non-Survivor | 2 | 20% | 6 | 60% | 2 | 20% | 0 | | |
| Total | | 4 | 20% | 11 | 55% | 4 | 20% | 1 | 5% | |

Table 4. Distribution of periodontitis grading among COVID-19 survivors and non-survivors.

| | | Periodontitis | | | | P |
|-------|----------------|---------------|-----|---------|-----|------|
| | | Grade A | | Grade B | | |
| Group | COVID Survivor | 4 | 40% | 6 | 60% | 1.00 |
| | Non-Survivor | 3 | 30% | 7 | 70% | |
| Total | | 7 | 35% | 13 | 65% | |

4 DISCUSSION

In addition to systemic conditions, oral manifestations could also occur in COVID long haulers. A previously conducted study observed some manifestations, among others were altered taste and smell, salivary gland ectasia, white tongue, dry mouth, facial muscle weakness, oral ulcers, and temporomandibular disorders (France & Glick 2022). Other oral manifestations affecting periodontal structures have been found in COVID-19 patients, among others are ulcers, vesicles, necrotizing gingivitis, and desquamative gingivitis. However, the relation whether these periodontal findings related to COVID-19 were still undetermined (Drozdik 2022).

Markers of systemic inflammation were found remarkably escalated in COVID-19 patients with periodontitis, which leads to the belief that there is a synergic relation between COVID-19 and periodontitis (Andrade *et al.* 2021). COVID-19-induced cytokine storm is comparable to cytokine imbalance in periodontitis, pointing to a potential relationship between COVID-19 and periodontitis. Similar cytokines and chemokines found in both COVID-19 and periodontitis among others are C reactive proteins (CRP), acute phase proteins, TNF- α (Tumor necrosis Factor-Alpha), IL (interleukin)-1 β , IL-2, IL-6, and Interferon-gamma (IFN-gamma) (Fabri 2020).

Studies found that cytokine storm was found in COVID-19 patients, with higher interleukin levels found in severe COVID-19 patients. This exaggerated systemic inflammatory response could generate oral manifestations, especially in the periodontal area, in view of the fact that periodontal disease is also mediated by the immune response. Inflammation response caused by inflamed gingival tissue could spread into the systemic circulation and cause amplified systemic inflammation. Conversely, it is proposed that increased

inflammatory response caused by COVID-19 could trigger periodontitis. In a 2020 study, some oral manifestations were found in COVID-19 patients, namely generalized erythematous, edematous gingiva, and necrotic interdental papillae (Gofur 2020). This finding was in agreement with a study conducted by Anand et al, where they found that subjects with a history of COVID-19 had substantially higher plaque scores, mobile teeth, gingival bleeding, probing depth, recession, and clinical attachment loss compared to patients with no history of COVID-19 (Anand *et al.* 2022).

Long COVID/post-COVID syndrome affects COVID-19 survivors, with symptoms surpassing 2 weeks after the acute infection phase. Most of the symptoms of long COVID are old symptoms experienced in the acute infection phase, but there are a considerable number of long-term symptoms not yet identified (Lopez-Leon *et al.* 2021). This pilot study conducted aims to determine whether there is a distinction between gingivitis and periodontitis severity between COVID-19 survivors and non-survivors.

Gingivitis severity between COVID-19 survivors and survivors was distinguished clinically by further categorizing the groups into localized or generalized gingivitis. There was no significant difference in the distribution of gingivitis survivors and non-survivors ($p>0.05$). Periodontitis severity between the two groups was categorized into periodontitis distribution, staging, and grading. Data analysis was done, showing that there was no significant difference in the distribution of periodontitis between COVID-19 survivors and non-survivors ($p>0.05$). Similarly, following data analysis, this study also showed that there were no differences in periodontitis staging and grading between COVID-19 survivors and non-survivors ($p>0.05$).

This pilot study has confirmed the absence of long COVID impact on gingivitis and periodontitis severity measured by clinical parameters. This was proven by no significant differences shown between COVID-19 survivors and non-survivors. Limitations of the study were the small sample size (40 subjects), and the only parameter used in this study were clinical parameters. Future studies could incorporate a larger group of subjects and various parameters besides clinical parameters. Some clinical parameters commonly utilized to diagnose periodontal diseases among others are probing depths, BOP, clinical attachment levels, plaque index, and radiographs. Although these are practical, cost-effective, and non-invasive, they are limited to only searching disease history and not current disease progression.¹⁵ Other parameters, for example, samples of saliva or gingival crevicular fluid have proven to reflect periodontal condition and are beneficial to detect biomarkers (Ramenzoni *et al.* 2021).

5 CONCLUSION

Clinical parameters assessed on gingivitis and periodontitis patients, such as BOP, CAL, periodontal staging, and grading showed no significant differences between COVID-19 survivors and non-survivors. These findings indicate that long COVID had no significant effect on periodontal clinical parameters and severity of gingivitis or periodontitis. Further studies incorporating a larger sum of subjects, as well as assessing other parameters (e.g. laboratory parameters such as cytokines) are required to further assess the impact of long COVID on the severity of periodontal disease.

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