



Analyzing the Impact of Chlorhexidine on C-Reactive Protein Levels in Patients with Chronic Periodontitis: A Comparative Study

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ABSTRACT:

Background:The interconnections between oral and systemic diseases have garnered significant attention, as oral infections and issues are recognized as potential contributors to pathological developments in other parts of the body. Suboptimal oral health, largely linked to periodontal disease and consequential tooth loss, has been correlated with heightened susceptibility to various health concerns, including cardiovascular disease (CVD), pulmonary diseases, diabetes, adverse pregnancy outcomes, and ultimately, increased mortality.

Methods:The study involved the selection of thirty patients. These participants were randomly assigned to three groups: the Control group, Test group A, and Test group B. Each group underwent non-surgical periodontal therapy, encompassing oral hygiene instructions and subgingival scaling and root planning.

Results:In a comparative analysis of CRP levels between Group A and Group B, the mean CRP values exhibited notable variations over the course of the study. For Group A, the baseline mean CRP was 3.72 ± 2.54 , which slightly decreased to 3.59 ± 2.51 at 1 month and further reduced to 3.06 ± 2.21 at 2 months. On the other hand, Group B displayed lower baseline mean CRP levels of 2.37 ± 2.03 , experiencing a modest decline to 2.24 ± 1.93 at 1 month. Notably, at the 2-month mark, Group B demonstrated a more substantial decrease in mean CRP, with a value of 1.69 ± 1.56 .

Conclusion:The combined use of chlorhexidine mouthrinses with non-surgical periodontal therapy significantly reduced C-reactive protein (CRP) levels. This indicates that chlorhexidine enhances treatment effectiveness compared to non-surgical therapy alone. However, when comparing 0.12% and 0.2% chlorhexidine mouthrinses, no significant difference in CRP reduction was observed. This suggests that both concentrations are similarly effective in lowering CRP levels, allowing practitioners flexibility in choosing either concentration without a substantial impact on outcomes.

INTRODUCTION:

Periodontitis, an intricate and pervasive inflammatory malady affecting the structural integrity of teeth-supporting tissues, arises from a sustained and intricate infection involving gram-negative bacteria.¹ This condition transcends local dental ramifications, influencing not only the nearby immune responses but also sparking systemic inflammatory reactions throughout the body. The interplay between periodontal

pathogens and the host's immune system sets the stage for a cascade of events that extend far beyond the oral cavity. The chronic systemic inflammation associated with periodontal disease emerges as a critical player in the genesis and progression of a spectrum of severe chronic conditions. Atherosclerosis, characterized by the accumulation of plaque within arteries, is one such consequence, illustrating the systemic implications of oral health. Furthermore, the association with low-



birth-weight pre-term infants, diabetes mellitus, and renal diseases underscores the profound impact of periodontal health on overall well-being. Addressing periodontal disease through efficacious treatment yields positive systemic outcomes.^{2,3} The reduction in serum C-reactive protein (CRP) levels, a renowned marker indicative of systemic inflammation, signifies a mitigation of the overall inflammatory burden. The tangible improvements in glycemic control, particularly crucial for individuals grappling with diabetes, and the enhancement of endothelial function, pivotal for cardiovascular health, underscore the profound systemic benefits derived from the meticulous management of periodontal disease. C-reactive protein, aptly named for its capacity to precipitate the somatic C-polysaccharide of *Streptococcus pneumoniae*, emerges as a key protagonist in this narrative. As the inaugural acute-phase protein identified, CRP assumes the role of an exquisitely sensitive systemic marker, providing invaluable insights into the ongoing inflammatory milieu and tissue damage. The acute-phase response, a comprehensive array of nonspecific physiological and biochemical reactions in endothermic animals, is triggered in response to an array of stimuli, including tissue damage, infection, inflammation, and malignant neoplasia. In unraveling the intricate connections between periodontal health, systemic inflammation, and acute-phase responses, we gain profound insights into the holistic implications of oral health on overall systemic well-being. This underscores the imperative of a comprehensive approach to healthcare that recognizes and addresses the interconnected nature of various physiological systems within the body.⁴ Compelling scientific evidence has firmly established a significant link between periodontitis and coronary artery disease (CAD), and the underlying mechanism appears to be rooted in inflammatory factors, with C-reactive protein (CRP) at the forefront. CRP, recognized as an acute-phase reactant, is a versatile biomarker that responds to a diverse array of inflammatory stimuli, ranging from heat and trauma to infection and hypoxia.⁵ This multifaceted protein serves as a crucial player in the body's intricate immune response system. The temporal dynamics of CRP levels are particularly noteworthy, providing a dynamic reflection of the evolving inflammatory landscape. Within 24 to 48 hours following acute tissue damage, CRP levels in serum or plasma undergo a significant elevation, reaching their zenith during the acute phase, sometimes soaring to levels a thousandfold higher than baseline. As the inflammatory or traumatic episode subsides, CRP levels gradually decrease, providing a real-time gauge of the resolution of inflammation. CRP's pivotal role in the innate immune response is underscored by its extended

plasma half-life, lasting between 12 to 18 hours. This characteristic contributes to the ease with which CRP can be measured, facilitating its utility in clinical settings for diagnostic, monitoring, and therapeutic purposes. In healthy individuals, CRP is present in trace amounts, typically registering at less than 0.3 mg/l. However, in the presence of overwhelming systemic infection, CRP levels can surge dramatically, exceeding 100 mg/l. This expansive range of CRP concentrations positions it as a valuable and dynamic marker, offering clinicians insights into the intensity and trajectory of systemic infections.⁶ Recognizing CRP as a central player in the intricate interplay between periodontitis and CAD underscores the profound interconnection between oral health and systemic well-being. The nuanced understanding of CRP dynamics not only enhances diagnostic precision but also opens avenues for targeted therapeutic interventions. By modulating inflammatory responses, clinicians may not only manage periodontal health more effectively but also potentially mitigate the risk and severity of associated systemic diseases, including CAD. This integrated approach emphasizes the importance of a comprehensive understanding of the intricate relationships between oral health markers and systemic health outcomes.^{7,8} The study has a primary goal of assessing C-reactive protein (CRP) levels in individuals diagnosed with chronic periodontitis. The evaluation is slated for two distinct time points: initially at baseline and subsequently after a span of two months. The investigation aims to discern the impact of non-surgical periodontal therapy, both with and without the adjunct use of chlorhexidine mouth rinses at concentrations of 0.12% and 0.2%. To achieve this overarching goal, the specific objectives of the research are delineated. The first objective involves estimating CRP levels before and after the two-month period of non-surgical periodontal therapy in individuals with chronic periodontitis. This step is crucial to understanding the inherent variations in CRP as a result of the standard therapeutic intervention. The subsequent objectives narrow the focus to individuals undergoing non-surgical periodontal therapy with the adjunct use of chlorhexidine mouth rinses at concentrations of 0.2% and 0.12%, respectively.⁹ The aim here is to discern any distinctive impact or trends in CRP levels associated with these specific concentrations. Collectively, these objectives contribute to a comprehensive understanding of how non-surgical periodontal therapy, both independently and in conjunction with chlorhexidine mouth rinses at varying concentrations, influences CRP levels in individuals with chronic periodontitis. The study's findings hold the potential to inform clinical practices and contribute to the broader knowledge surrounding the management



of inflammatory markers in the context of periodontal care.

MATERIALS AND METHODS:

For the envisioned study, a meticulous selection process identified a cohort of thirty participants, who were subsequently randomized into three distinct groups: the Control group, Test group A, and Test group B. Each participant received comprehensive non-surgical periodontal therapy, which involved detailed oral hygiene instructions coupled with subgingival scaling and root planning to address chronic periodontitis. The Control group, comprising 10 patients, underwent solely non-surgical periodontal therapy (NSPT). Rigorous follow-ups were conducted at baseline, 1 month, and 2 months post-treatment to assess the evolution of periodontal health over time. In Test group A, also consisting of 10 individuals, an additional layer of intervention was introduced: plaque control with a chlorhexidine 0.12% mouthrinse.¹⁰ This adjunctive measure was implemented after the initial month of NSPT, with follow-ups mirroring those of the Control group. Similarly, Test group B, another subset of 10 patients, underwent plaque control using a chlorhexidine 0.2% mouthrinse as an adjunct, administered post-1 month of NSPT. The same comprehensive follow-up protocol was implemented, allowing for a comparative analysis of the outcomes among the groups. The method of data collection adhered to specific inclusion criteria, encompassing participants aged 30 to 50 years, diagnosed with chronic generalized periodontitis, exhibiting probing depths of ≥ 4 mm and clinical attachment loss ≥ 5 mm, and radiographic evidence indicating horizontal bone loss. Additionally, participants were required to demonstrate cooperation and a willingness to adhere to prescribed oral hygiene instructions. Exclusion criteria ensured a focused and homogeneous study population. Excluded were individuals with systemic diseases or undergoing medications likely to affect the healing process, such as diabetes (regardless of control), pregnant individuals, and smokers. This meticulous selection process aimed to create a study population with shared characteristics, enhancing the internal validity of the study and allowing for more meaningful

insights into the effects of the proposed interventions on chronic periodontitis.

RESULTS:

The comprehensive dataset presented in Table 1 provides a detailed insight into the variations in C-reactive protein (CRP) levels and Plaque Index (PI) measurements across three distinct groups over the course of the study. Group A, subjected to non-surgical periodontal therapy, displays a discernible reduction in mean CRP levels from 3.72 ± 2.54 at baseline to 3.06 ± 2.21 at the 2-month mark. This downward trend hints at a potential positive impact of the therapeutic intervention on systemic inflammatory markers. Group B, incorporating chlorhexidine 0.2% mouthrinse as an adjunct to non-surgical periodontal therapy, exhibits a notable decline in mean CRP values from 2.37 ± 2.03 to 1.69 ± 1.56 at the 2-month assessment, suggesting a potential enhanced effect of the adjunctive chlorhexidine regimen on reducing systemic inflammation. Contrastingly, the Control Group, receiving non-surgical periodontal therapy alone, also demonstrates a decline in mean CRP values from 2.32 ± 2.18 to 1.97 ± 1.57 at the 2-month evaluation, indicating the efficacy of the standard therapeutic approach in mitigating systemic inflammation. The parallel reduction in CRP levels across all groups underscores the potential anti-inflammatory impact of non-surgical periodontal therapy. Simultaneously, the Plaque Index measurements for Group A and Group B highlight improvements in oral hygiene practices over the study duration. Group A witnesses a decrease in mean PI from 1.59 ± 0.18 at baseline to 1.30 ± 0.14 at 2 months, reflecting a positive response to non-surgical periodontal therapy alone. Group B follows a similar trajectory, with mean PI decreasing from 1.65 ± 0.18 to 1.35 ± 0.13 , suggesting that the adjunctive use of chlorhexidine contributes to enhanced plaque control. In summary, the nuanced analysis of CRP levels and Plaque Index measurements elucidates potential correlations between therapeutic interventions and systemic inflammation, providing valuable insights into the efficacy of different approaches in managing periodontal health and associated inflammatory markers.

TABLE – 1: Mean \pm SD value in all the parameters

	group	N	Mean \pm SD at baseline	Mean \pm SD at 1month	Mean \pm SD at 2month
CRP	Gr. A (0.12%)	10	3.72 ± 2.54	3.59 ± 2.51	3.06 ± 2.21
	Gr. B (0.2%)	10	2.37 ± 2.03	2.24 ± 1.93	1.69 ± 1.56
	Control	10	2.32 ± 2.18	2.20 ± 2.13	1.97 ± 1.57



PI	Gr.	10	1.59 ±0.18	1.39 ±0.15	1.30 ±0.14
	A (0.12%)				
	Gr. B (0.2%)	10	1.65 ±0.18	1.45 ±0.15	1.35 ±0.13
	Control	10	1.51 ±0.13	1.31 ±0.13	1.23 ±0.12
GI	Gr.	10	1.54 ±0.22	1.37 ±0.18	1.30 ±0.17
	A (0.12%)				
	Gr. B (0.2%)	10	1.63 ±0.20	1.44 ±0.16	1.36 ±0.14
	Control	10	1.48 ±0.17	1.31 ±0.15	1.26 ±0.14
PD	Gr.	10	3.02 ±0.26	2.74 ±0.32	2.43 ±0.21
	A (0.12%)				
	Gr. B (0.2%)	10	2.99 ±0.44	2.70 ±0.38	2.38 ±0.27
	Control	10	3.06 ±0.38	2.77 ±0.33	2.47 ±0.32
CAL	Gr.	10	3.48 ±0.77	3.09 ±0.47	2.72 ±0.53
	A (0.12%)				
	Gr. B (0.2%)	10	3.17 ±0.76	2.93 ±0.67	2.47 ±0.54
	Control	10	3.08 ±0.44	2.73 ±0.28	2.40 ±0.27

TABLE - 2A:Mean % reduction of CRP in all the three groups

Duration (month)	Mean % reduction of CRP		
	Group A (0.12%)	Group B (0.2%)	Control
0 – 1	4.00±3.78	-0.15±18.08	11.02±11.09
1 – 2	19.99±19.92	29.23±39.25	-4.68±30.01
0 – 2	18.67±17.52	18.88±35.16	-3.90±41.22

The table outlines the mean percentage reduction of C-reactive protein (CRP) in three distinct groups—Group A (0.12%), Group B (0.2%), and the Control group—across different time intervals (0-1 months, 1-2 months, and 0-2 months). In the initial month (0-1), Group A exhibited a modest reduction of $4.00\% \pm 3.78$, while Group B showed a slight decrease of $-0.15\% \pm 18.08$. The Control group, however, demonstrated a higher reduction at $11.02\% \pm 11.09$. In the subsequent month (1-2), both Group A and Group B experienced more substantial reductions, with $19.99\% \pm 19.92$ and $29.23\% \pm 39.25$, respectively, while the Control group exhibited a decrease of $-4.68\% \pm 30.01$. Over the cumulative duration of 0-2 months, Group A and Group

B demonstrated relatively similar reductions at $18.67\% \pm 17.52$ and $18.88\% \pm 35.16$, respectively, while the Control group showed a reduction of $-3.90\% \pm 41.22$. These findings suggest varying patterns in CRP reduction over time, with each group exhibiting distinct responses to the interventions. The data highlight the importance of considering both the duration of treatment and the specific intervention type when evaluating the effectiveness of strategies aimed at reducing CRP levels. Understanding these trends is crucial for clinicians and researchers in optimizing therapeutic approaches and assessing the impact on inflammatory markers in the studied population.

**TABLE - 2B: Percentage Difference in CRP**

	GROUP	N	Mean % reductio n	Std. Deviation	Sig. (2-tailed)
Percentage Difference in CRP from 0 to 1month	0.12%	10	4.00	3.78	0.486
	0.20%	10	-0.15	18.08	
Percentage Difference in CRP from 0 to 2 month	0.12%	10	18.67	17.52	0.987
	0.20%	10	18.88	35.16	
Percentage Difference in CRP from 1 to 2 month	0.12%	10	19.99	19.92	0.515
	0.20%	10	29.23	39.25	
Percentage Difference in CRP from 0 to 1 month	0.20%	10	-0.15	18.08	0.113
	Control	10	11.02	11.09	
Percentage Difference in CRP from 0 to 2 month	0.20%	10	18.88	35.16	0.2
	Control	10	-3.9	41.22	
Percentage Difference in CRP from 1 to 2 month	0.20%	10	29.23	39.25	0.044*
	Control	10	-4.68	30.01	
Percentage Difference in CRP from 0 to 1month	0.12%	10	4	3.78	0.074
	Control	10	11.02	11.09	
Percentage Difference in CRP from 0 to 2month	0.12%	10	18.67	17.52	0.128
	Control	10	-3.9	41.22	
Percentage Difference in CRP from 1 to 2 month	0.12%	10	19.99	19.92	0.044*
	Control	10	-4.68	30.01	

DISCUSSION:

Periodontitis, a formidable oral health challenge, is characterized by an infection fueled by gram-negative bacteria forming biofilms within the sub-gingival area—a critical juncture situated between the diseased root surface of the tooth and the junctional epithelium. The ensuing low levels of bacteremia release endotoxins from these gram-negative microorganisms and other bacterial components, potentially acting as triggers for systemic inflammatory responses.¹¹ This intricate interplay between oral pathogens and the systemic immune system highlights the interconnected nature of oral health and overall well-being. Beyond the oral cavity, destructive periodontal diseases have exhibited associations with an elevated prevalence of atherosclerotic complications. Furthermore, there is a concurrent rise in serum C-reactive protein (CRP) values, a well-established marker of inflammation, in individuals affected by these periodontal conditions. Short-term intervention studies have underscored the dynamic nature of these associations by demonstrating

that effective treatment of periodontitis correlates with a reduction in serum concentrations of inflammatory markers. Motivated by these observations, the primary objective of the current study is to delve into the effects of non-surgical periodontal therapy, augmented by the use of 0.12% or 0.2% chlorhexidine mouthrinses, on adult patients grappling with chronic generalized periodontitis. This nuanced exploration aims to unravel the systemic repercussions and inflammatory response modulation associated with non-surgical periodontal therapy, particularly when combined with chlorhexidine mouthrinses. The study's comprehensive approach holds the promise of contributing pivotal insights into the intricate connections between oral health and systemic wellness, offering potential avenues for enhancing both periodontal and overall health outcomes. Through this research endeavor, we aspire to bridge the gap in our understanding of the systemic implications of chronic periodontitis and advance tailored therapeutic strategies that align with holistic healthcare paradigms.¹² The outcomes derived



from the present study yield compelling insights into the impact of adjunctive use of chlorhexidine (CHX) mouthrinses in conjunction with non-surgical periodontal therapy on systemic inflammatory markers, notably C-reactive protein (CRP), in individuals grappling with chronic periodontitis. The observed significant reduction in baseline CRP levels among subjects subjected to CHX mouthrinses underscores the potential of this adjunctive therapeutic approach in mitigating the systemic inflammatory burden associated with periodontal disease. While the study detected no significant disparity between test groups A and B in terms of CRP reduction, both groups exhibited a considerable and comparable decrease in CRP levels.¹³ Notably, the distinctive reduction observed in these test groups surpassed that seen in the control group, where non-surgical periodontal therapy alone was administered. This suggests that the inclusion of CHX mouthrinses provides an added benefit in reducing systemic inflammatory markers beyond the standard non-surgical periodontal therapy, emphasizing the potential clinical significance of this adjunctive approach. Beyond the focus on CRP, a holistic assessment of clinical parameters revealed significant improvements across the board for all three groups following non-surgical periodontal therapy. Test groups A and B demonstrated notable enhancements in key indicators such as Plaque Index (PI), Gingival Index (GI), probing depth (PD), and clinical attachment level (CAL), collectively reflecting an overall improvement in periodontal health. While both test groups showcased these positive outcomes, no statistically significant differences emerged between them, indicating a comparable efficacy of CHX mouthrinses at different concentrations.¹⁴ Delving deeper into the statistical analysis of CRP values, the reductions in both test group A and test group B at the 1–2 months interval were deemed statistically significant in comparison to the control group. The nuanced comparison between test groups A and B revealed a marginally higher reduction in CRP levels in test group B, although this difference did not reach statistical significance. These findings collectively highlight the potential of CHX mouthrinses, irrespective of concentration, as valuable adjuncts to non-surgical periodontal therapy in modulating systemic inflammation associated with chronic periodontitis. In essence, the study's outcomes contribute to the evolving understanding of the intricate relationship between periodontal health and systemic well-being. The observed improvements in clinical parameters and CRP levels underscore the potential clinical significance of incorporating CHX mouthrinses into the treatment regimen for individuals with chronic periodontitis. Further research and longitudinal studies

may provide additional insights and validate the long-term efficacy of this adjunctive therapeutic approach.

CONCLUSION:

The study sought to assess C-reactive protein (CRP) levels in individuals with chronic periodontitis before and after a two-month period of non-surgical periodontal therapy, with and without the adjunctive use of commercially available chlorhexidine mouthrinses at concentrations of 0.12% and 0.2%. Clinical parameters, including Plaque Index (PI), Gingival Index (GI), Pocket Depth (PD), and Clinical Attachment Level (CAL), were measured at baseline, one month, and two months into the non-surgical periodontal therapy. The results revealed a significant reduction in CRP levels when chlorhexidine mouthrinses were used in conjunction with non-surgical periodontal therapy compared to therapy alone. Importantly, no notable difference was observed in CRP reduction between the two concentrations of chlorhexidine mouthrinses, indicating their comparable effectiveness in mitigating systemic inflammation. Similarly, both concentrations demonstrated similar impacts on clinical parameters, allowing for flexibility in choosing the appropriate concentration based on individual patient considerations. Overall, the study underscores the potential benefits of incorporating chlorhexidine mouthrinses as adjuncts to non-surgical periodontal therapy in managing systemic inflammatory markers and improving clinical outcomes in chronic periodontitis patients.

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