

Effect of Non-Surgical Periodontal Intervention in Chronic Kidney Disease Patients: A Pilot Study

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ABSTRACT

Background: Periodontal disease has been highlighted as an important potential risk factor for non-communicable diseases such as chronic kidney disease (CKD), diabetes mellitus, cardiovascular diseases, and pulmonary diseases. Studies have reported that patients suffering from end-stage renal failure (ESRF) and those receiving dialysis are more prone to compromised periodontal health status. Dialysis patients may form calculus more rapidly than healthy individuals possibly due to high salivary urea and phosphate levels. Studies prove that non-surgical periodontal therapy significantly improves the periodontal status and also reduces inflammatory mediators which ultimately improve the glomerular filtration rate.

Method: This was designed as a pilot study and was performed between 2018 and 2019. The inclusion criteria were CKD patients of age 18 and above. Data from medical records were collected, clinical oral examination was performed by recording periodontal pocket depth (PPD) p value: 0.001, clinical attachment loss (CAL) p value: 0.000, bleeding on probing (BOP) p value: 0.012, serum creatinine p value: 0.035, e-GFR p value: 0.009. Non-surgical periodontal therapy was given to CKD patients at baseline and clinical periodontal data were statistically analysed.

Result: Non-surgical periodontal therapy given to study group has resulted into significant improvement in all the measured periodontal clinical parameters.

Conclusion: this study demonstrates that CKD patients show effective response to Non-surgical periodontal therapy.

Keywords:

Chronic kidney disease, Dialysis, Periodontitis, Non-surgical periodontal therapy

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INTRODUCTION

Periodontal disease has been highlighted as an important potential risk factor for non-communicable diseases such as diabetes mellitus, cardiovascular diseases, pulmonary diseases, and Chronic Kidney Diseases (CKD).¹ It is a chronic inflammatory and infectious disease of oral cavity that causes destruction of the gingiva, periodontal ligaments, and alveolar bone, predominantly caused by

Gram negative bacteria residing in dental plaque biofilm. This dental biofilm plays the main role in initiating the inflammatory process which is associated with periodontal destruction. On the other hand, CKD, also known as chronic renal disease, is a progressive loss in renal function over a period of months or years. It is defined by National kidney foundation of USA as – “Kidney damage or glomerular filtration rate <60 mL/min/1.73 m² for more than three months”. CKD is classified into stage one to five based on glomerular filtration rate. There has been significant progress in validating the plausibility of oral disease disseminating to cause systemic diseases. Systemic diseases can be affected by the progression and

treatment of periodontitis and has been the focus of sustained interest in the field of Periodontology. There is enough evidence to suggest that periodontal infections can adversely affect systemic health with manifestations such as Coronary Heart Disease (CHD) and CHD-related events such as angina and infarction, atherosclerosis, stroke, diabetes mellitus, preterm labor, low birth weight delivery and respiratory diseases [1]. Various studies have shown a substantial relation between poor oral hygiene to increased susceptibility for various systemic disorders. Existing evidence points to a relationship between oral bacteria and systemic diseases such as bacterial endocarditis, atherosclerosis, lung disease and other cardiovascular diseases, and is well-documented in the literature.⁴ During the past two decades, the interplay between plaque induced gingivitis/periodontitis and other systemic conditions has been investigated. Periodontal disease also disproportionately affects racial and ethnic minorities and has been recently implicated as an independent risk factor for CKD. To date, two studies have found that severe periodontal disease (as defined by radiographic criteria among a cohort of Pima Indians with diabetes and by periodontal inflammation criteria in a cohort of elderly Japanese adults) is associated with an increased risk of kidney function decline over time. In the last 30 years there has been a lot of research on the pathogenesis of periodontitis and we now understand that the host response to the organisms causing periodontitis varies between individuals. Certain systemic disorders and conditions alter host tissues and physiology which may impair host barrier integrity and host defence to periodontal infection resulting in more destructive disease. The association between periodontitis and CKD has also been studied. The link between periodontal diseases and CKD may be bidirectional. In the last decade there has been an increasing evidence to show association between periodontitis and chronic kidney disease. It has been suggested that different forms of acute and chronic inflammatory processes can stimulate an inflammatory response in the kidneys, leading to CKD. CKD shares many risk factors with periodontitis, such as diabetes mellitus, declining age and smoking. It has also been suggested that periodontitis could be considered a non-traditional risk for CKD due to the systemic inflammation burden caused by periodontal inflammation and its locally produced inflammatory mediators, such as interleukin-1(IL-1), interleukin-6(IL-6), prostaglandin E2 (PGE2) and tumour necrosis factor (TNF-a) and the presence of bacteria and their products in the bloodstream. Recent evidence has shown that patients with periodontitis may have elevated levels of C-reactive protein (CRP) and, consequently, a mild acute-phase systemic inflammatory response when compared with healthy subjects. Since the link between chronic systemic inflammation and CKD is measured by the levels of CRP, periodontitis is considered as a source of "permanent inflammation" could contribute to CKD. A study reported that patients suffering from End-Stage Renal Failure (ESRF) and those receiving dialysis are more prone to periodontal disease and other oral health

problems. It revealed that the renal failure patients had higher gingival index and bleeding, probing depths, attachment loss, enamel hypoplasia, pulp obliteration, and less caries. Plaque was also found to be higher in the dialysis and predialysis groups, and it was concluded that the correlation between dialysis and ESRF and gingivitis, probing depth, attachment loss, and enamel hypoplasia is significant. Dialysis patients may form calculus more rapidly than healthy individuals possibly due to high salivary urea and phosphate levels, and a significant correlation between plaque scores and gingival inflammation in renal dialysis patients has been reported. However, the association of periodontal disease with kidney function decline over time among Indians has not been explored. This study included the subjects from central India suffering from Chronic Kidney diseases and periodontitis. This was the first interventional study done in India and in this region. Aim of this study was to gather data to inform a definitive study into the impact of successful periodontal treatment on the periodontal and renal health of patients with CKD.

METHODOLOGY

After obtaining ethical approval from the institutional ethical committee of Datta Meghe Institute of Medical Sciences, the purpose of the study was explained and a written informed consent was obtained from individuals who were willing to participate in the study. This study was conducted in the department of Periodontics, Sharad Pawar Dental College in collaboration with Acharya Vinobha Bhawe Rural Hospital, Sawangi, Wardha. A total of 20 CKD patients were recruited from the medicine department of Acharya Vinobha Bhawe Rural Hospital. Patients aged 18 years or older, and who were able to provide informed consent to participate in the study were selected. Patients with moderate to severe periodontitis, with total number of teeth present to be 20, Clinical attachment loss of >5mm and cumulative probing pocket depth of at least 30mm were included [2]. The exclusion criteria included patients with end stage renal disease, patients who have received specialized periodontal treatment in previous one year and patients who were receiving immunosuppressive agents. To calculate the glomerular filtration rate (GFR) values, the Cockcroft and Gault equation was used to estimate the clearance of creatinine (Ccr), as follows:

$$Ccr = \{(140 - \text{age}) \times \text{weight}\} / (72 \times \text{Scr})$$

where, Ccr is expressed in millilitres per minute, age in years, weight in kilograms, and serum creatinine (Scr) in milligrams per decilitre. Patients with diagnosis of chronic kidney disease who were receiving dialysis were examined for all periodontal parameters and laboratory parameters. A detailed dental and medical history was obtained from all patients. All volunteers received a full mouth periodontal clinical examination performed at six sites per tooth (excluding the third molars) by one trained examiner. Baseline measurements of the Plaque Index (PI), Papillary Bleeding Index (PBI), Pocket Depth (PD) and Clinical Attachment Levels (CAL) were recorded at baseline and at 3 months after therapy. Non-surgical

periodontal treatment consisting of oral hygiene instructions and supragingival scaling with ultrasonic scalers was done. Clinical periodontal parameters, GFR, and serum creatinine were assessed at baseline and 3 months post-therapy.

RESULTS

The evaluated parameters periodontal pocket depth , clinical attachment loss , bleeding on probing , serum

creatinine , estimated glomerular filtration rate at baseline and at an interval of 3 months obtained were submitted to paired-t test. The mean and Standard Deviation (SD) of the values are tabulated in Table no.1 and shown in the figure no.1. The results showed that study group experienced a significant improvement in all of the periodontal clinical parameters measured. Statistically significant improvements were seen in both the laboratory parameters- serum creatinine and e-GFR.

Clinical parameters	Study group (CKD PATIENTS)		P value
	PRE SCRP	POST SCRP	
PPD	3.23 + 0.78	2.91 + 0.85	0.001*
CAL	3.67 + 0.71	3.27 + 0.86	0.000*
BOP	1.82 + 0.40	1.72+0.40	0.012*
SERUM CREATININE	8.65 + 1.84	7.84 + 1.52958	0.035*
e- GFR	8.55 + 1.82	9.41 + 1.41	0.009*

Table1: Clinical parameters recorded in CKD group at baseline and after scaling (Mean + SD in mm).

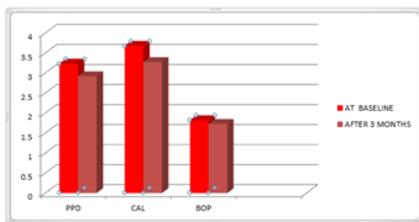


Figure1: Depicting the change in the clinical periodontal parameters from baseline.

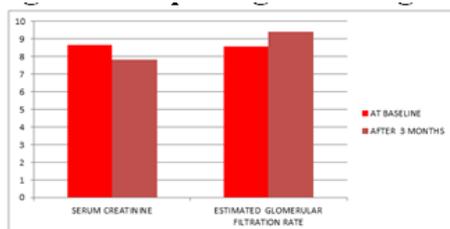


Figure2: Depicting changes in serum creatinine and e-GFR values from baseline.

DISCUSSION

Chronic Kidney Disease (CKD) is a debilitating systemic condition which causes chronic deterioration of nephrons. CKD is defined as Glomerular Filtration Rate (GFR) reduction or kidney damage, reflected as abnormal urine sediment or abnormalities in the renal anatomy. It leads to progressive and usually irreversible decline of the glomerular infiltration rate leading to an increase in serum creatinine and blood urea nitrogen levels. Periodontitis is a bacteria-driven chronic inflammatory disease that destroys the connective tissue and bone that supports the teeth and represents a potential source of

episodes of bacteraemia especially in immune compromised patients. The increasing incidence of renal failure and patients receiving renal replacement therapy including haemodialysis, peritoneal dialysis or renal transplantation comprise a large group of dental patients with increased levels of plaque, calculus and gingival inflammation and possible increased prevalence and severity of destructive periodontal disease. The link between CKD and periodontitis is this persistent chronic inflammation. Recently, this link between CKD and periodontitis has been the focus for research in the field of periodontology. However, the role of periodontitis as a risk factor for CKD is still not clear. Due to scarcity of research in this field, more evidence is required to highlight the connection between the two. Therefore, the main purpose of this study was to evaluate the effect of non-surgical periodontal therapy on chronic kidney disease patients. Periodontitis is initiated by bacterial accumulation between the gingivae and teeth, which triggers an inflammatory-immune response within the host [3]. Periodontitis has been found to contribute to systemic inflammatory burden with increase in CRP and its effect on systemic health.8 Periodontal inflammation contributes to the systemic inflammatory burden, through acute-phase and oxidative stress pathway, as evidenced by increases in CRP, IL-6 and biomarkers of oxidative stress in the serum of patients with periodontitis. Literature has shown 100% prevalence of periodontal disease in these patients; compared to the healthy individuals the release of these mediators plays an important role in inflammation. These lead to synthesis of prostaglandin-2(PG2), Nitric Oxide (NO), Platelet Activating Factor (PAF). These cause vascular changes in the endothelium and secretion of cytokines. Ultimately, the role of kidneys in clearance of cytokines is affected. Hence, increase in periodontal inflammation leads to down regulation of kidney function. Successful periodontal therapy is associated with reductions in

these inflammatory mediators. Chronic Renal failure has not only shown to affect the general health but also oral and periodontal health of the patients. In our study clinical periodontal parameters including Clinical Attachment Loss (CAL), Periodontal Pocket Depth (PPD) has significant improvement at 3 months from baseline. (p value-0.00,0.01,respectively).Comparable improvement was observed in the serum creatinine levels. With the help of serum creatinine values e-GFR values were calculated [5]. To calculate the Glomerular Filtration Rate (GFR) values, the Cockcroft and Gault equation was used to estimate the clearance of creatinine. The results obtained in this study are similar to the study done by, the study group by included predialysis patients. Statistically significant changes from baseline were found in the measured periodontal parameters after nonsurgical periodontal. The values obtained are given in table 1. Statistically significant changes were seen with all clinical parameters except for BOP. (P value: 0.012). Some studies observed significant reduction in bleeding on probing, however we did not. Study population included by artese included predialysis group of patients however in our study patients were undergoing dialysis. The significant changes we observed with respect to serum creatinine levels and e- GFR values might be the result of haemodialysis reported significant co-relation between plaque levels and gingival inflammation in dialysis patients has also postulated that periodontal disease is influenced by chronic renal failure because of insufficient bone metabolism.¹⁴ In the study by the results obtained were contradictory to their original hypothesis that stated CKD patients would respond poorly to periodontal therapy. They included predialysis patients in the study and statistically significant changes were seen in all the clinical periodontal parameters (PPD, BOP, CAL) $P < 0.05$. There was also significant impact on GFR post-SCRIP in both groups showing a statistically significant improvement in GFR in group 1 (predialysis) ($p = 0.04$) and group 2 (chronic periodontitis) ($p = 0.002$) Serum creatinine levels post therapy were improved when measured after 3 months. In the study by dialysis was not a factor in the improvement of laboratory parameters. (Serum creatinine and e-GFR). The study by showed pathogenic microorganisms persisted in high levels in non-responsive sites of CKD individuals compared with chronic periodontitis patients, however there was no significant difference in all periodontal parameters measured at baseline and post therapy. ($p > 0.05$)¹¹ Both groups showed significant clinical improvement in those sites for PD, CAL, and VP after treatment ($p < 0.05$, Wilcoxon test). In our study significant changes were observed in all the periodontal clinical parameters measured at baseline after 3 months ($p < 0.05$). Significant improvements were seen in serum creatinine levels and e-GFR measured at baseline and after 3 months ($p < 0.05$). However the laboratory parameters were measured soon after the dialysis, it is difficult to conclude that the

changes seen were due to non-surgical periodontal therapy. Our study include small sample size therefore, chances of confounding factor is more and it is difficult to conclude that the changes in glomerular filtration rate in chronic kidney disease patients due to nonsurgical periodontal therapy therefore, large sample size needed to be explored. However, further research is necessary to establish whether effective periodontal therapy will actually result in outcome in patients with chronic kidney diseases.

CONCLUSION

The CKD patients on dialysis with periodontitis are medically complex and present the dental practitioner with several challenges in the management of their periodontal condition. Consultation with the patient's nephrologist is required for the benefit of patient. First line of treatment includes non-surgical periodontal therapy (Scaling and root planning) followed by maintenance therapy. However, the presence of extensive pocket formation associated with severe osseous defects or exposure of anatomical features such as root furcations may inhibit attempts at effective oral hygiene or local root debridement. Therefore, for patients who have not resolved after initial periodontal therapy and who demonstrate adequate levels of plaque control, surgical pocket elimination, either by resection or regeneration, should be indicated. Non-surgical periodontal therapy along with dialysis helps improving periodontal parameters serum creatinine levels and e-GFR.

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