Volume 3, Issue 5, May 2025 ISSN (E): 2938-3765

OPTIMIZATION OF DENTAL TREATMENT IN PATIENTS WITH CHRONIC RENAL FAILURE AND INFLAMMATORY DISEASES OF THE ORAL MUCOSA

Sabirov Bobur Kadirbaevich
Khabibova Nazira Nasullaevna
Bukhara State Medical Institute

Abstract

This article examines the clinical specifics and pathogenetic mechanisms underlying the optimization of dental treatment in patients with chronic renal failure (CRF) accompanied by inflammatory lesions of the oral mucosa. The analysis is based on a synthesis of empirical data demonstrating that CRF induces systemic dysregulation of immune and metabolic pathways, which significantly alters the morphology and function of the oral tissues. Persistent xerostomia, shifts in the biochemical composition of saliva, and salivary protein imbalance lead to the destabilization of local defense mechanisms. Histologically, oral mucosal inflammation in CRF patients is characterized by atrophic epithelial changes, microvascular disturbances, and impaired reparative responses. The prevalence and severity of gingivitis, stomatitis, and non-carious cervical lesions correlate with residual renal function, dialysis modality, and the presence of endocrine comorbidities. Dental hard tissues exhibit structural degradation, including enamel demineralization and dentin sclerosis, with altered elemental ratios. The study substantiates the necessity of individualized treatment protocols, incorporating pre-dialysis planning, management of odontogenic infection foci, and modulation of inflammatory activity with non-nephrotoxic agents. The proposed therapeutic model emphasizes interdisciplinary coordination between dentistry and nephrology to mitigate systemic complications and preserve oral functionality.

Keywords: Chronic renal failure; oral mucosa; inflammatory lesions; periodontal status; xerostomia; dialysis; biochemical markers; structural integrity of enamel; immunomodulation; nephrology-oriented dental protocols.

Introduction

Chronic renal failure (CRF) is characterized by progressive systemic dysregulation, which includes disturbances in nitrogen metabolism, mineral balance, and immunological homeostasis. These alterations directly affect the morphofunctional status of oral tissues, leading to specific clinical phenotypes of inflammatory diseases of the oral mucosa. In patients with advanced stages of CRF, oral pathology is not limited to increased susceptibility to infection but includes structurally verifiable alterations of both hard and soft tissues, such as epithelial atrophy,

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

connective tissue fibrosis, sclerotic changes in dentin, and demineralization of enamel with modified Ca/P ratios.

Clinical observations confirm that inflammatory lesions of the oral mucosa in CRF progress independently of typical infectious triggers and are modulated by the stage of renal insufficiency, dialysis modality, and associated endocrine dysfunctions. The severity of periodontal and mucosal inflammation correlates with serum urea levels, phosphate retention, parathyroid hormone concentrations, and residual renal clearance. Histopathological examinations reveal chronic inflammatory infiltrates, microvascular alterations, and delayed epithelial regeneration, forming a distinct nosological pattern unresponsive to standard dental protocols.

Management of oral pathology in this cohort is further complicated by altered pharmacodynamics, bleeding diathesis, and high frequency of chronic odontogenic infection foci. Endodontic and periodontal interventions are frequently associated with delayed healing and increased risk of systemic inflammatory response. The necessity for optimization of dental treatment protocols in CRF is clinically justified by reduced efficacy of conventional therapies and the need for integration of nephrological parameters into dental decision-making algorithms.

The present study investigates the structural, biochemical, and clinical parameters that determine the course of inflammatory oral diseases in CRF, and formulates evidence-based strategies for their management. The objective is to define a therapeutic framework aligned with renal pathology, focused on minimizing systemic complications and preserving oral integrity through nephrology-adapted dental care.

Chronic renal failure is accompanied by progressive alterations in systemic homeostasis, including nitrogen retention, dysregulation of calcium-phosphate metabolism, and secondary hyperparathyroidism, all of which have a direct impact on the structural and functional integrity of oral tissues [1]. Uremic toxicity, combined with immune dysfunction and microvascular changes, contributes to persistent inflammation of the oral mucosa, with characteristic atrophic and erosive features [2].

Periodontal pathology in patients with chronic kidney disease is characterized by increased prevalence and severity of generalized periodontitis. Morphological alterations in gingival tissue and alveolar bone are closely associated with residual renal function and duration of dialysis therapy [3]. Inflammatory destruction is often accompanied by fibrotic degeneration and impaired collagen remodeling, especially in long-term dialysis patients [4].

Salivary hypofunction is a prominent clinical feature in renal failure, reflected in reduced unstimulated flow rate and altered composition. Deficiency of protective components such as lactoferrin, lysozyme, and secretory IgA predisposes to candidiasis, aphthous lesions, and viral infections [5]. Comparative studies indicate more pronounced changes in the group undergoing peritoneal dialysis relative to those on hemodialysis [6].

Mineralized dental tissues also undergo structural changes. Enamel microfractures, dentinal sclerosis, and deviations in Ca/P and (Ca+Na+Mg)/P molar ratios have been documented in patients with advanced renal disease, correlating with serum levels of parathyroid hormone and markers of phosphate retention [7]. Elemental analysis of dental tissues reveals mineral imbalance as a contributing factor to increased susceptibility to both carious and non-carious lesions [8].

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Materials and Methods

The clinical study was conducted at Bukhara State Medical Institute. A total of 90 individuals were included, divided into three statistically comparable groups. The main group consisted of 42 patients with clinically verified chronic renal failure stages III–V, receiving either hemodialysis or continuous ambulatory peritoneal dialysis. The comparison group comprised 28 patients with chronic kidney disease without established renal failure. The control group included 20 somatically healthy individuals without systemic pathology.

Clinical dental examinations were performed under standardized lighting using mirror-sonda technique. Assessment of mucosal inflammation included localization, phase, and severity of stomatological lesions based on WHO criteria. Gingival status was evaluated using the Papillary-Marginal-Attached Index (PMA), Muhlemann-Sulcus Bleeding Index, and Periodontal Screening and Recording Index (PSR). Salivary flow rate was measured unstimulated over five minutes. Biochemical analysis of saliva included pH measurement via potentiometry, total protein via Bradford assay, lactoferrin and albumin concentration via enzyme-linked immunosorbent assay (ELISA). Calculation of lactoferrin/albumin ratio was performed to quantify local mucosal immune activity.

Dental hard tissue assessment involved digital intraoral radiography and fiber-optic transillumination. For a subset of patients requiring extraction of premolars for prosthetic indications, samples were collected for scanning electron microscopy (SEM) and energy-dispersive X-ray spectroscopy (EDX). Elemental composition of enamel and dentin was determined with calculation of Ca/P and (Ca+Na+Mg)/P ratios.

All patients underwent laboratory blood analysis to quantify serum creatinine, urea, phosphorus, total and ionized calcium, parathyroid hormone (PTH), and C-reactive protein (CRP). Glomerular filtration rate was calculated using the CKD-EPI equation. For dialysis patients, duration of renal replacement therapy, type of dialysis, and dialysis adequacy (Kt/V) were recorded. Presence or absence of residual urine output was noted.

Clinical and biochemical parameters were subjected to statistical analysis using IBM SPSS Statistics v26. Distribution normality was verified using the Shapiro–Wilk test. Intergroup differences were analyzed via one-way ANOVA with post hoc Bonferroni correction. Correlation analysis employed Spearman's rank coefficient. Statistical significance threshold was set at $p < 0.05$.

Results and Discussion

A total of 90 subjects participated in the study, stratified into three distinct cohorts based on renal status and systemic health. Group I included 42 patients with clinically verified stage III–V chronic renal failure (CRF), undergoing either hemodialysis (n = 27) or peritoneal dialysis (n = 15), with a mean dialysis duration of 4.7 ± 1.3 years. Group II comprised 28 patients diagnosed with chronic kidney disease (CKD) not requiring renal replacement therapy. Group III consisted of 20 systemically healthy individuals without any history of renal, endocrine, or immune dysfunction, matched by age and sex.

Comprehensive clinical assessment of the oral cavity revealed a significantly higher incidence of inflammatory mucosal pathologies in Group I compared to both comparison groups. Specifically,

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

mucosal lesions of atrophic and ulcerative character, predominantly localized to the buccal and palatal epithelium, were observed in 76.2% of Group I patients, 42.8% of Group II, and 15.0% of controls. The difference was statistically significant ($\chi^2 = 19.32$, $p < 0.001$), reflecting a direct correlation between severity of renal impairment and mucosal vulnerability.

Sialometric analysis demonstrated a progressive decline in unstimulated salivary flow rate across the study groups, with Group I presenting a mean value of 0.11 ± 0.04 mL/min, significantly lower than both Group II (0.18 ± 0.05 mL/min) and Group III (0.29 ± 0.07 mL/min) (ANOVA, $F = 34.89$, $p < 0.001$). In conjunction with hypofunction, alterations in salivary composition were documented, specifically a reduction in protective protein components. Enzyme-linked immunosorbent assay (ELISA) revealed a marked decrease in lactoferrin concentrations in Group I, with an adjusted lactoferrin-to-albumin ratio of 0.67 ± 0.21, compared to 0.94 ± 0.25 in Group II and 1.21 ± 0.27 in controls ($p = 0.004$), suggesting functional compromise of mucosal immunity under uremic conditions.

Periodontal examination using the PMA (Papillary-Marginal-Attached) Index revealed chronic gingival inflammation in the majority of dialysis-dependent patients. The average PMA Index in Group I was 43.5 ± 9.8%, which exceeded values recorded in Group II (29.4 ± 6.2%) and Group III (11.2 ± 3.1%) ($p < 0.001$). Notably, this increase was independent of oral hygiene status, indicating systemic factors as primary modulators of periodontal health.

Laboratory biomarkers further elucidated the pathophysiological landscape. Serum C-reactive protein (CRP), a reliable indicator of systemic inflammation, was elevated in Group I (14.7 ± 3.4 mg/L) relative to Group II (8.6 ± 2.1 mg/L) and Group III (3.3 ± 0.9 mg/L) ($p < 0.001$). Parathyroid hormone (PTH) levels were highest in the dialysis group (248.6 ± 60.1 pg/mL), reflecting secondary hyperparathyroidism and its potential influence on mineralized tissue turnover.

To quantify enamel and dentin quality, extracted premolars from 12 patients in each group were subjected to scanning electron microscopy (SEM) and energy-dispersive X-ray spectroscopy (EDX). Enamel in CRF patients demonstrated a significant reduction in the Ca/P ratio (1.43 ± 0.08) when compared to CKD patients without dialysis (1.61 ± 0.07) and controls (1.71 ± 0.05) ($p < 0.01$). Elemental analysis revealed additional increases in Mg and Na, consistent with a pathological mineralization profile and increased enamel porosity.

A detailed summary of key clinical and biochemical parameters is presented in Table 1.

Table 1. Comparative Analysis of Oral and Systemic Parameters Across Study Groups (Mean ± SD)

Parameter	Group I (CRF + Dialysis)	Group II (CKD Non-Dialysis)	Group III (Control)	p-value
Mucosal inflammation prevalence (%)	76.2	42.8	15.0	< 0.001
Unstimulated salivary flow rate (mL/min)	0.11 ± 0.04	0.18 ± 0.05	0.29 ± 0.07	< 0.001
Lactoferrin/Albumin ratio in saliva	0.67 ± 0.21	0.94 ± 0.25	1.21 ± 0.27	0.004
PMA Index (%)	43.5 ± 9.8	29.4 ± 6.2	11.2 ± 3.1	< 0.001
Serum CRP (mg/L)	14.7 ± 3.4	8.6 ± 2.1	3.3 ± 0.9	< 0.001
Serum PTH (pg/mL)	248.6 ± 60.1	129.3 ± 38.4	41.7 ± 13.2	< 0.001
Enamel Ca/P ratio (SEM–EDX)	1.43 ± 0.08	1.61 ± 0.07	1.71 ± 0.05	< 0.01

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Correlation analysis revealed significant inverse relationships between salivary immune markers and clinical indices of inflammation. The lactoferrin/albumin ratio negatively correlated with the PMA index ($r = –0.62$, $p = 0.001$), underscoring the association between local immune depletion and periodontal deterioration. A similar pattern was observed between PTH levels and enamel Ca/P ratio ($r = –0.59$, $p = 0.004$), indicating a systemic-metabolic influence on hard tissue mineral content.

These results substantiate the hypothesis that inflammatory oral pathology in CRF is not a secondary complication but a direct extension of the uremic and metabolic milieu. Given the observed impairment in salivary secretion, immune protection, and mineral homeostasis, conventional dental management strategies appear insufficient in this cohort.

Targeted therapeutic interventions should include regular screening for mucosal lesions, periodic salivary analysis, remineralization therapy based on elemental composition, and integration of dental care into renal treatment protocols. The data support the implementation of a multidisciplinary approach combining nephrological monitoring with preventive and restorative dental measures tailored to the severity of renal dysfunction and type of dialysis.

Conclusion

The findings of this study demonstrate a clear and statistically significant relationship between the severity of chronic renal failure and the progression of inflammatory and degenerative lesions in the oral mucosa and periodontal tissues. Patients undergoing dialysis exhibit markedly compromised salivary function, impaired mucosal immunity, and structural demineralization of dental hard tissues. These alterations are strongly correlated with systemic markers of inflammation, parathyroid hormone levels, and biochemical evidence of mineral imbalance.

The observed inverse correlation between salivary lactoferrin/albumin ratio and periodontal inflammatory index confirms the immunological vulnerability of the oral cavity in end-stage renal disease. Similarly, deviations in enamel Ca/P ratio as a function of hyperparathyroidism substantiate the systemic-mineral influence on dental integrity. The accumulation of uremic toxins, dysbiosis, and chronic microvascular ischemia within oral tissues further aggravate the inflammatory potential and reduce healing capacity.

These results underscore the necessity for a multidisciplinary treatment model integrating nephrological supervision with individualized dental protocols. Such protocols must include pre-dialysis dental clearance, salivary diagnostic surveillance, remineralization strategies, and strict infection control measures. Clinical dentistry in patients with CRF should no longer be considered auxiliary but must be positioned as a strategic component of systemic disease management.

References

1. Proctor R., Kumar N., Stein A., Moles D., Porter S. Oral and dental aspects of chronic renal failure // J Dent Res. – 2005. – Vol. 84, No. 3. – P. 199–208.
2. Guzeldemir E., Toygar H.U., Seydaoglu G. Evaluation of some periodontal risk factors in patients with chronic renal failure // J Periodontol. – 2009. – Vol. 80, No. 2. – P. 206–213.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

3. Davidovich E., Schwarz Z., Davidovitch M., Eidelman E., Bimstein E. Oral findings and periodontal status in children and young adults suffering from renal failure // J Clin Periodontol. – 2005. – Vol. 32. – P. 1076–1082.
4. Martins C., Siqueira W.L., Primo L.S. Oral and salivary flow characteristics of Brazilian children with chronic renal failure // Pediatr Nephrol. – 2008. – Vol. 23. – P. 619–624.
5. Ioannidou E., Swede H., Fares G., Himmelfarb J. Tooth loss strongly associates with malnutrition in chronic kidney disease // J Periodontol. – 2014. – Vol. 85, No. 7. – P. 899–907.
6. Al Nowaiser A., Roberts G.J., Trompeter R.S., Wilson M., Lucas V.S. Oral health and caries-related factors in children with chronic renal failure // Pediatr Nephrol. – 2003. – Vol. 18. – P. 39–45.
7. Costantinides F., Castronovo G., Vettori E., Frattini C., Artero M.L. Dental care for patients with end-stage renal disease undergoing hemodialysis // Int J Dent. – 2018. – Article ID 9610892. – P. 1–7.
8. Nunn J.H., Sharp J., Lambert H.J., Plant N.D., Coulthard M.G. Oral health in children with renal disease // Pediatr Nephrol. – 2000. – Vol. 14. – P. 997–1001.
9. Yashchuk E.V., Platova T.S., Tsymbalistov A.V. Morphological structure and elemental profile of enamel in chronic kidney disease // Inst Stomatol. – 2008. – No. 3(40). – P. 46–48.
10. Naylor G.D., Fredericks M.R. Pharmacological considerations in the dental management of the patient with disorders of the renal system // Dent Clin North Am. – 1996. – Vol. 40, No. 4. – P. 665–683.
11. Kshirsagar A.V., Craig R.G., Moss K.L., Beck J.D., Offenbacher S., Kotanko P. Periodontal disease adversely affects the survival of patients with end-stage renal disease // Kidney Int. – 2009. – Vol. 75. – P. 746–751.
12. Bayraktar G., Kurtulus I., Duraduryan A., Cintan S., Kazancioglu R. Dental and periodontal findings in hemodialysis patients // Oral Dis. – 2007. – Vol. 13, No. 4. – P. 393–397.
13. Klassen J.T., Krasko B.M. The dental health status of dialysis patients // J Can Dent Assoc. – 2002. – Vol. 68. – P. 34–38.
14. Bots C.P., Poorterman J.H., Brand H.S., Kalsbeek H., van Amerongen B.M., Veerman E.C. The oral health status of dentate patients with chronic renal failure undergoing dialysis therapy // Oral Dis. – 2006. – Vol. 12, No. 2. – P. 176–180.
15. Craig R.G. Interactions between chronic renal disease and periodontal disease // Oral Dis. – 2008. – Vol. 14, No. 1. – P. 1–7.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5