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Review Article

The relationship between chronic kidney disease and soft tissue inflammation in the oral cavity

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ABSTRACT

Chronic kidney disease (CKD) is a systemic condition marked by progressive loss of renal function, often accompanied by complications beyond the kidneys. Among these, oral soft tissue inflammation remains under-recognized despite its growing clinical relevance. Individuals with CKD frequently experience gingivitis, periodontitis, oral mucosal lesions, and xerostomia, all of which may exacerbate systemic inflammation. The bidirectional relationship between renal dysfunction and oral disease is mediated through immune suppression, accumulation of uremic toxins, and microbial dysbiosis. Neutrophil impairment, altered cytokine profiles, and changes in salivary composition contribute to a compromised oral environment, allowing chronic inflammation to persist in soft tissues. The inflammatory burden from oral tissues may influence CKD progression by elevating systemic markers such as interleukin-6 and C-reactive protein. Moreover, patients with advanced CKD or those receiving dialysis often report nutritional challenges due to oral discomfort, which further complicates disease management. Periodontal pathogens and their byproducts may enter systemic circulation, potentially aggravating endothelial dysfunction and cardiovascular risks associated with renal impairment. Despite these associations, oral health is seldom integrated into CKD care protocols. Preventive strategies involving routine screening, patient education, and interdisciplinary collaboration are essential for early detection and intervention. Coordinated care models involving nephrologists and dental professionals can facilitate timely treatment and reduce systemic complications. Enhancing awareness of the oral-systemic link and prioritizing oral health within CKD management may improve long-term outcomes and quality of life for affected individuals.

Keywords: Chronic kidney disease, Oral inflammation, Periodontal disease, Systemic inflammation, Interdisciplinary care

INTRODUCTION

Chronic kidney disease (CKD) is a global health burden affecting over 10 percent of the adult population and is associated with increased risk of cardiovascular disease, infections, and overall mortality.[1] While much attention has been given to systemic complications of CKD, its impact on oral health, particularly soft tissue inflammation, is increasingly recognized. Individuals with CKD often present with gingivitis, periodontitis, and mucosal lesions, all of which can diminish quality of life and complicate disease management. Uremia-related metabolic imbalances and immune dysfunction contribute significantly to this oral pathology, creating a bidirectional relationship between kidney health and oral inflammation.[2]

The pathophysiological mechanisms linking CKD to oral soft tissue inflammation are multifactorial. Accumulation of nitrogenous waste products, such as urea and creatinine, alters the composition and pH of saliva, promoting a shift in the oral microbiota toward more pathogenic species.[3] Additionally, systemic inflammation, commonly observed in CKD, is mirrored in the oral cavity by elevated levels of pro-inflammatory cytokines like interleukin-6 and TNF-alpha. These mediators not only damage gingival and mucosal tissues but may also influence systemic disease progression. Impaired neutrophil function and altered immune surveillance further weaken the host defense in the oral environment, rendering patients more susceptible to chronic inflammation.[4]

Dialysis, often necessary in advanced stages of CKD, introduces further complications affecting the oral cavity. Xerostomia is frequently reported among dialysis patients and is associated with increased mucosal irritation and inflammation. Moreover, the medications commonly used in managing CKD have documented adverse effects on oral tissues. Studies have shown that dialysis patients with poor oral health have elevated levels of systemic inflammatory markers and lower serum albumin, a known predictor of morbidity in this population.[5] This highlights the clinical importance of managing oral inflammation not just for local health, but for its broader implications in CKD outcomes.

The overlooked nature of oral health in CKD care represents a missed opportunity for early intervention. Oral soft tissue inflammation may serve as a modifiable source of systemic inflammation, and treating it could potentially slow CKD progression. Despite increasing evidence, routine dental evaluations are rarely integrated into nephrology care protocols. Collaborative care between dental and renal professionals is essential to address this gap. A comprehensive understanding of the biological and clinical links between CKD and oral inflammation can inform more effective, interdisciplinary treatment strategies that enhance both systemic and oral outcomes.[6]

CKD

CKD contributes to a state of persistent systemic inflammation and immunosuppression, which predisposes patients to soft tissue inflammation in the oral cavity. Altered immune responses, including reduced neutrophil activity and disrupted cytokine regulation, make the oral mucosa more vulnerable to infections and chronic inflammation. Clinical studies have reported a higher prevalence of oral lesions, gingival enlargement, and mucosal ulcerations among CKD patients, particularly those undergoing hemodialysis.[7] These oral manifestations are not merely localized issues but may have systemic implications, contributing to the overall inflammatory burden that characterizes CKD progression. In addition, xerostomia and salivary gland dysfunction, commonly observed in CKD, disrupt the normal antimicrobial functions of saliva. This shift in the oral environment facilitates colonization by pathogenic bacteria, exacerbating soft tissue inflammation. Evidence suggests that periodontal and mucosal inflammation in CKD patients may influence cardiovascular and renal outcomes due to the systemic spread of inflammatory mediators. Management of oral inflammation through routine dental care and targeted periodontal therapy has shown potential in reducing systemic inflammation and improving renal biomarkers.[8] Addressing oral health in CKD patients is therefore essential not only for symptom control but also for supporting broader disease management strategies.

MECHANISMS LINKING CKD AND ORAL INFLAMMATION

The interaction between CKD and oral soft tissue inflammation is driven by complex immune, metabolic, and microbial mechanisms. CKD alters both innate and adaptive immunity, impairing cellular responses necessary for maintaining oral mucosal integrity. Neutrophil dysfunction, which includes reduced chemotaxis and oxidative burst capacity, is well-documented in individuals with declining renal function and has been linked to increased susceptibility to oral pathogens. Inflammatory lesions in the gingiva and other soft tissues are often more persistent in these individuals due to the inability of immune cells to effectively clear microbial insults.[9]

Uremic toxins, which accumulate as glomerular filtration declines, contribute to a systemic inflammatory state that impacts the oral environment. Compounds such as indoxyl sulfate and p-cresol sulfate have been shown to modulate the expression of pro-inflammatory cytokines and increase oxidative stress at epithelial barriers. This favors a microenvironment in which oral soft tissues, particularly gingival and mucosal tissues, experience sustained low-grade inflammation. In vitro models have demonstrated that epithelial cells exposed to uremic serum express higher levels of interleukin-1 beta and tumor necrosis factor-alpha, which are also implicated in

periodontal disease progression.[10] These mediators contribute to collagen breakdown and vascular permeability in the soft tissues of the oral cavity.

Salivary gland dysfunction is another key consequence of CKD that influences soft tissue inflammation. Reduced salivary flow not only diminishes mechanical clearance of microorganisms but also impairs the delivery of antimicrobial peptides and immunoglobulins essential for mucosal defense. The resulting xerostomic environment promotes colonization by acidogenic and proteolytic bacterial species, including *Porphyromonas gingivalis* and *Fusobacterium nucleatum*, both known to drive inflammatory responses in gingival tissues. These shifts in microbiota, referred to as dysbiosis, are often seen in CKD patients even in the absence of overt oral disease and may precede clinical signs of soft tissue damage.[11]

In CKD, mineral metabolism disturbances also influence oral tissue health. Secondary hyperparathyroidism, common in advanced stages of renal dysfunction, can induce changes in oral bone and adjacent soft tissues, creating structural instability and increasing susceptibility to inflammation. Calcification of soft tissue structures, although less commonly discussed in dental contexts, has been reported in patients with severe CKD and may contribute to discomfort, ulceration, or delayed healing following minor trauma. Additionally, oxidative stress related to systemic metabolic dysregulation can impair fibroblast function and collagen synthesis, weakening the structural resilience of gingival tissues.[12]

CLINICAL IMPACT ON CKD MANAGEMENT

The intersection of CKD and oral soft tissue inflammation is increasingly recognized in clinical management due to shared inflammatory pathways and reciprocal effects on disease progression. Periodontal disease, oral mucosal lesions, and salivary dysfunction not only contribute to local discomfort but also elevate systemic inflammatory markers known to influence renal outcomes. Among patients with moderate to advanced CKD, C-reactive protein and interleukin-6 levels tend to be higher in the presence of active oral inflammation, suggesting that oral disease may serve as a nontraditional risk factor for accelerating renal decline.[6]

Clinical observations have shown that patients with untreated periodontal inflammation frequently present with poorer nutritional status, hypoalbuminemia, and elevated cardiovascular risk. These complications are already prevalent in CKD and can be compounded by unrecognized oral inflammation. Inflammatory cytokines originating in the oral cavity enter the systemic circulation, contributing to endothelial dysfunction and vascular injury. The cumulative burden of systemic inflammation is not isolated to the kidneys but affects broader metabolic and cardiovascular systems, leading to poorer dialysis outcomes and increased hospitalization rates.[13]

Nutritional challenges in CKD may also be exacerbated by oral discomfort. Mucosal inflammation and xerostomia can impair mastication, reduce appetite, and limit dietary intake, particularly in elderly patients with comorbidities. Painful lesions or fungal superinfections may deter consumption of high-protein foods, which are often recommended to counter muscle wasting. In dialysis populations, where protein-energy wasting is a known concern, soft tissue inflammation in the oral cavity further restricts the ability to meet nutritional goals. Without timely identification and management of oral complications, these patients risk entering a cycle of malnutrition and inflammation that further compromises renal function.[14]

Dental management of patients with CKD presents unique challenges, especially in the context of medication interactions and bleeding risk. Many patients are prescribed anticoagulants, antiplatelet agents, or immunosuppressive therapies, which can complicate both routine dental procedures and emergency interventions. Inadequate awareness among nephrologists and dentists regarding these risks often leads to delays in oral care, allowing soft tissue inflammation to progress unchecked. Coordination between specialties remains limited, despite growing evidence that treating periodontal disease in CKD can modestly reduce systemic inflammation and improve clinical parameters such as serum albumin and estimated glomerular filtration rate. Delayed healing and increased susceptibility to infection following dental procedures are also more prevalent in this population due to impaired immune function and altered wound response.[15]

STRATEGIES FOR PREVENTION AND CARE

Effective management of oral soft tissue inflammation in CKD involves an integrated care model that bridges nephrology and dental services. Traditional healthcare systems often treat oral and systemic diseases in silos, which results in missed opportunities for early detection of oral pathology in individuals with CKD. Dental professionals can serve a critical role in screening for mucosal lesions, periodontal inflammation, and salivary dysfunction, particularly in patients attending regular dialysis sessions. Early identification of inflammation in gingival or mucosal tissues may serve as a warning sign of immune dysregulation or worsening metabolic control. Routine oral health assessments, when aligned with nephrology care, have the potential to prevent progression of inflammatory lesions and reduce systemic inflammatory burden.[16]

Preventive care must prioritize patient education alongside clinical interventions. Individuals undergoing dialysis often receive complex medication regimens and dietary restrictions, but oral hygiene instructions are rarely incorporated into treatment plans. Structured educational programs led by dental hygienists or nurses can improve patient knowledge and encourage consistent

oral hygiene behavior. Evidence supports that tailored instruction on brushing techniques, use of chlorhexidine rinses, and proper denture hygiene significantly reduces plaque accumulation and soft tissue irritation in renal populations.[5] These efforts are most effective when repeated over time and adapted to the changing needs of patients as CKD progresses.

Infection control is a central component of care in this group, given the elevated risk of systemic infections due to immunosuppression. Prophylactic antimicrobial protocols may be required before dental procedures, especially for individuals with vascular access devices or prior episodes of infective endocarditis. However, the use of antibiotics must be carefully balanced to avoid resistance and consider renal clearance rates. Salivary substitutes, fluoride varnishes, and topical antifungal agents are also commonly used to manage complications like xerostomia and candidiasis, both of which are frequently reported among patients with reduced salivary flow or altered oral flora.[17] These interventions, while considered basic, can dramatically improve oral comfort and reduce soft tissue trauma in vulnerable individuals.

Interprofessional collaboration remains essential but often underdeveloped. Communication between nephrologists, dentists, primary care providers, and pharmacists can help coordinate treatment plans, particularly around timing of interventions and medication adjustments. For instance, scheduling dental treatments on non-dialysis days, reviewing anticoagulant use prior to surgery, and ensuring proper wound care all require joint planning. Despite logistical barriers, models of shared care have shown promise. Clinics integrating dental screening into nephrology visits have reported higher rates of periodontal referrals and earlier intervention for mucosal lesions.[18] These results indicate that structural integration, rather than isolated service delivery, may be key to improving outcomes. The burden of oral disease in CKD reflects broader challenges in healthcare accessibility, patient engagement, and clinical integration. Addressing these challenges requires more than isolated dental procedures. Long-term preventive strategies must include regular monitoring, coordinated care pathways, patient empowerment, and systems designed to support oral health as a core element of chronic disease management.

CONCLUSION

CKD and oral soft tissue inflammation are interconnected through shared inflammatory and metabolic pathways. Recognizing this link is essential for improving patient outcomes and quality of life. Integrated care approaches can help manage both conditions more effectively. Preventive strategies and interdisciplinary collaboration remain vital in clinical practice.

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