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Oral microbiota in hemodialysis patients; a narrative review on recent ideas

Mayyadah Hameed Rashid[1*], Haifaa Abdulameer Radhi[2], Aseel Abdulameer Radhi[3]

[1]Department of Dental Techniques, College of Health and Medical Technology, Ashure University, Baghdad, Iraq
[2]Department of Dentistry, Alamal College for Specialized Medical Science, Karbala, Iraq
[3]Department of Prosthodontics College of Dentistry, University of Karbala, Karbala, Iraq

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ABSTRACT

Alterations in oral microbiota can significantly affect systemic health in hemodialysis (HD) patients. Oral dysbiosis contributes to inflammation, production of uremic toxins, and microbial translocation, which can exacerbate cardiovascular disease, chronic kidney disease (CKD), malnutrition, and cognitive impairment. In addition, oxidative stress from periodontal bacteria exacerbates CKD by promoting systemic inflammation, increasing renal oxidative damage, and accelerating functional decline. Conversely, the persistent oxidative stress caused by periodontal bacteria creates a hostile microenvironment in the kidneys, characterized by mitochondrial impairment, membrane damage, disrupted calcium homeostasis, DNA and protein damage, and chronic inflammation. These factors collectively inhibit renal tissue repair mechanisms, accelerating renal failure progression and worsening of kidney outcomes. Managing oral health through good oral hygiene, dietary modifications, and interventions such as probiotics and prebiotics is crucial for improving the overall health and quality of life for HD patients.

Review

Implication for health policy/practice/research/medical education:
Oral dysbiosis is common in hemodialysis (HD) patients, characterized by reduced diversity and increased pathogenic species, contributing to poor oral health and potentially impacting systemic outcomes. In addition, periodontal pathogens significantly contribute to systemic inflammation in chronic kidney disease (CKD) patients by entering the circulation, triggering immune responses, and elevating inflammatory mediators, all of which can worsen renal function and overall health outcomes. Regular dental care and monitoring of oral health are especially important in this population. Factors such as dialysis catheters, dietary restrictions, medications, and oral hygiene practices contribute to these alterations. Modalities like good oral hygiene, probiotics, prebiotics, and dietary modifications can help improve oral microbiota composition in HD patients.

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Introduction

Hemodialysis (HD) patients experience significant alterations in their oral microbiota compared to healthy individuals, primarily due to the underlying chronic kidney disease (CKD) and the effects of dialysis itself (1). There is a notable imbalance (dysbiosis) in the oral microbiota of HD patients, with a significant reduction in microbial diversity and a shift in the relative abundance of specific taxa (2). This dysbiosis is associated with a pro-inflammatory state and may contribute to both oral and systemic complications (3). HD patients also show an increased prevalence of pathogenic bacteria linked to periodontal disease and dental caries (4). Besides, greater concentration of *Porphyromonas gingivalis*, *Treponema denticola*, *Haemophilus*, *Actinomyces*, *Streptococcus mutans*, *Lactobacillus salivarius*, *Lactobacillus fermentum*, *Lactobacillus vaginalis*, *Scardovia wiggsiae*, and *Actinomyces naeslundii* have been reported (5). These changes favor the development of periodontitis (6). Meanwhile, factors such as uremia, altered salivary composition, and systemic inflammation disrupt the balance between commensal and pathogenic microorganisms in the oral cavity (7). The severity of periodontal disease and the degree of oral microbiota alteration tend to increase with the

***Corresponding author**: Mayyadah Hameed Rashid, Email: mayadah.hameed@au.edu.iq

duration and severity of CKD and HD (8). Conversely, the dysbiotic oral microbiota in renal failure patients is linked to the worsening of periodontal health, which may accelerate CKD progression or contribute to additional complications (9). Oral pathogens can also influence the gut microbiome, potentially exacerbating systemic inflammation (10). Some studies show that oral bacteria rarely cause HD vascular access infections (11). The most common pathogens in such infections are *Staphylococcus* and *Enterococcus* species, not typical oral flora (11). Furthermore, certain medications administered in HD, such as iron-based phosphate binders, can induce patient-specific shifts in the oral microbiome, though the overall group effect may be minimal (12,13).

Systemic inflammation by periodontal pathogens in CKD

Periodontal pathogens play a significant role in driving systemic inflammation among patients with chronic renal failure, contributing to both the progression of this disease and the worsening of periodontal health (14). Periodontal pathogens such as *P. gingivalis*, *Tannerella forsythia*, and *Treponema denticola* can enter the bloodstream through ulcerated gingival tissue (15). Once in circulation, these bacteria and their products like lipopolysaccharide activate the immune system, leading to the release of pro-inflammatory cytokines, including interleukin (IL)-1, IL-6, tumor necrosis factor alpha (TNF-α) and acute-phase proteins like C-reactive protein (CRP) (15,16). This systemic inflammatory response can damage the kidneys and further exacerbate CKD progression as mentioned above (17). Several studies show a strong association between the presence of periodontal pathogens and increased markers of systemic inflammation detected as elevated CRP, IL-6 and TNF-α in CKD patients too (18). Recent investigation found, higher serum levels of IgG against *P. gingivalis* correlate with increased CRP levels in HD patients, independent of other causes of inflammation (19). Also, the severity of periodontitis and the abundance of these pathogens are greater in CKD patients compared to healthy controls, since this correlates with worse kidney function (20). However, this relationship is bidirectional, since CKD-related immune dysregulation and metabolic disturbances can worsen periodontal disease, while periodontitis-induced systemic inflammation can further impair kidney function (14). Each 10% increase in periodontal inflammation is associated with a 3% decrease in renal function (21). Accordingly, systemically disseminated periodontal pathogens and their inflammatory mediators can exacerbate renal inflammation and oxidative stress, across with inducing endothelial dysfunction and arteriosclerotic changes in the kidney (22). This condition also promotes persistent, low-grade inflammation that accelerates CKD progression (22).

CKD progression by oxidative stress from periodontal bacteria

Numerous studies found that, the oxidative stress generated by periodontal bacteria plays a crucial role in the progression of chronic renal failure by amplifying systemic inflammation and directly damaging renal tissues (21). Periodontal pathogens such as *P. gingivalis* can enter the bloodstream through ulcerated gum tissue (23); then, their components, especially lipopolysaccharide (LPS), trigger the systemic immune response, resulting in the production of reactive oxygen species (ROS) and pro-inflammatory cytokines (e.g., IL-1, IL-6 and TNF-α) (15,24). This condition, directed to a systemic oxidative stress state, defined by an imbalance between pro-oxidant and antioxidant systems, with increased ROS and reduced antioxidants like glutathione peroxidase (25,26). Experimental models show that periodontitis increases oxidative stress markers like malondialdehyde and decreases antioxidants such as glutathione in renal tissues, causing histological kidney damage such as injury to the renal tubules (25,27). Furthermore, the inflammatory and oxidative environment promotes endothelial dysfunction, cellular apoptosis, and persistent low-grade inflammation in the kidneys, accelerating CKD progression (28). Interestingly, structural equation modeling in human studies confirms a causal link; since a 10% increase in periodontal inflammation results in a 3% decrease in renal function, mediated by systemic oxidative stress (21). Hence, patients with both CKD and periodontitis have lower systemic antioxidant capacity and higher oxidative burden than those with either condition alone (29). Similarly, systemic oxidative stress induced by periodontal bacteria profoundly impairs renal tissue repair processes through multiple interconnected mechanisms (21). Periodontal pathogen-induced ROS inhibit the mitochondrial electron transport chain in kidney cells, causing mitochondria to produce excessive ROS (30). This mitochondrial dysfunction forwarded to energy deficits and further oxidative damage within renal cells, undermining their ability to repair and regenerate (30). Likewise, excess ROS react with polyunsaturated fatty acids in renal cell membranes, triggering lipid peroxidation (22). This damages membrane integrity and fluidity, disrupting cellular homeostasis critical for tissue repair (18). Correspondingly, ROS disrupt endoplasmic reticulum calcium pumps, causing calcium overload and unfolded protein responses (14). These stress responses impair protein folding and cellular metabolism, hindering renal cell recovery and repair (31). In parallel, ROS modify intracellular proteins and induce DNA damage, which interferes with essential signaling pathways and gene expression involved in cell proliferation, differentiation and repair (18,32). This compromises the regenerative capacity of renal tissue and promotes progression of kidney damage (30). Besides, oxidative stress from periodontal inflammation elevates systemic

inflammatory mediators like IL-1β, TNF-α, which further exacerbate renal oxidative damage and fibrosis, impeding effective tissue healing (22). Similar to periodontal tissues, excessive ROS can induce apoptosis and dysfunction in renal cells, reducing the pool of viable cells necessary for repair and regeneration (25).

Focus on salivary microbiome in HD

Chronic kidney disease and HD are linked to an imbalance in oral microbiota, which can induce a micro-inflammatory state and systemic inflammatory responses (8). Recently Duan et al showed that this dysregulation may lead to a higher prevalence of periodontitis and an increased risk of oral dysbiosis in end-stage renal disease (ESRD) individuals undergoing HD (33). Prior studies demonstrated that, HD cases generally show higher salivary microbial diversity but lower richness, indicating a shift in the microbial community structure (33). The study by Duan et al, who compared 108 HD patients with 100 healthy controls found that HD patients had a lower Chao index (richness estimator), across with a higher Shannon index (diversity index). They showed a distinct separation of microbial communities between HD patients and healthy controls (33). Moreover, the taxonomic composition at the phylum level varies significantly between HD patients and healthy individuals (33). While Firmicutes, Proteobacteria, Bacteroidetes, Actinobacteria, and Fusobacteria are dominant in both groups, the relative abundances of Firmicutes, Bacteroidetes, spirochaetae, Synergistetes, Tenericutes, and Gracilibacteria are significantly higher in HD patients (33). Conversely, Proteobacteria and Actinobacteria abundances tend to decrease (33). They also found, at the genus level, Ruminococcaceae, Capnocytophaga, Porphyromonas, Veillonellaceae, and *Granulicatella* are enriched in HD patients, while *Lautropia*, *Prevotella*, *Actinomyces*, *Veillonella*, *Rothia*, and *Leptotrichia* are more abundant in healthy controls (33). An increase in Firmicutes has also been observed in the gut microbiome of ESRD patients during HD (33). Furthermore, the core salivary microbiome in HD patients differs from that of healthy controls (33). On the other hand, *Actinomyces odontolyticus* and *Rothia aeria* are more prevalent in healthy individuals, while *Porphyromonas gingivalis*, *Neisseria elongata, Catonella morbi, Porphyromonas endodontalis*, and *Capnocytophaga leadbetteri* are primarily detected as core salivary microbiome in HD patients (33). Notably, they detected the presence of *Porphyromonas gingivalis*, a bacterium linked to periodontitis, is significantly elevated in the HD group (33). This finding suggests that ESRD patients under HD creates an oral environment rich in pathogens, potentially leading to a high prevalence and severity of periodontitis in this population (33). In fact, the functional analysis of the salivary microbiome predicts that microbes with higher relative abundances in healthy individuals are mainly associated with cellular processes, signaling (e.g., ABC transporters, secretion systems, pores ion channels), and energy metabolism (e.g., oxidative phosphorylation) (33). In contrast, microbes in HD patients are more likely to be involved in various metabolic processes, including carbohydrate metabolism (e.g., fructose and mannose metabolism), amino acid metabolism (e.g., valine, leucine, isoleucine degradation and beta-alanine metabolism), xenobiotics biodegradation, and lipid metabolism (33). Sporulation-related microbial functions also increase in HD patients (33). Porphyromonas, which is more abundant in HD patients, degrades amino acids into compounds like short-chain fatty acids, sulfur compounds, ammonia, and indole/skatole, all of which contribute to the virulence in periodontitis (33).

Oral health problems in HD patients

Patients on HD frequently report oral problems, including dry mouth, tooth sensitivity, oral ulcers, and gum bleeding (34). Recently Miyata et al showed that poor oral hygiene and a higher prevalence of periodontitis and inflammation compared to control groups (35). They also detected that, specific oral alterations like tooth mobility, malocclusion, crowding, and severe surface erosions are also observed (35). Others also demonstrated a high chance of fungal infections due to colonization by yeasts, particularly Candida, in patients with stage 5 CKD undergoing HD (36). Despite optimal dialysis, oral disease may increase due to lower uptake of public dental services, increased malnutrition, and inflammation (37). However, oral bacteria rarely cause vascular access infections in HD patients (11).

Periodontal health and dialysis duration

Chronic kidney disease is a risk factor for periodontitis, and HD patients tend to have higher levels of periodontal pathogens (38). While the overall structure of the microbial community might not be significantly altered by the duration of HD, certain specific species (39), like, Lachnospiraceae sp. HMT_096 shows a positive correlation with both the duration of HD and the community periodontal index (CPI), indicating its potential role in worsening periodontal status over time (33). Other species, such as *Agrobacterium tumefaciens, Pseudomonas marincola, Vibrio gigantis, Streptococcus anginosus, Pseudomonas orientalis,* and *Arthrospira platensi*, show correlations with the CPI index (33). The severity of periodontal disease can slightly worsen as the HD duration prolongs (35).

Increased metabolic processes in oral microbiota

The oral microbiota in HD patients is predicted to be more involved in carbohydrate metabolism (8). This includes processes such as fructose and mannose metabolism. Bacterial metabolites, which are by-products of carbohydrate metabolism, include short-chain fatty

acids, amines, and gases (8). Moreover, amino acid metabolism pathways are increased in the oral microbiota of HD patients (33,40). Examples include the degradation of valine, leucine, isoleucine, and beta-alanine metabolism (33). Porphyromonas, which is more abundant in HD patients, contributes to the virulence in periodontitis by degrading amino acids into compounds such as short-chain fatty acids, sulfur compounds, ammonia, and indole/skatole (41). Xenobiotics biodegradation is another metabolic process that shows increased activity in the oral microbiota of HD patients (8). This suggests that the microbial community is adapting to process foreign compounds or waste products that may accumulate in patients with impaired kidney function (42). Likewise, lipid metabolism is among the increased metabolic functions in the oral microbiota of HD patients (43). Bacterial metabolites from lipid metabolism also include short-chain fatty acids, amines, and gases (44). In addition to the aforementioned metabolic processes, sporulation-related microbial functions also increase in HD patients (43,45). This indicates a potential adaptation of certain microbial species to stress conditions prevalent in the oral environment of these patients (8).

Therapeutic modalities

Antioxidants such as melatonin have been shown in experimental models to reduce oxidative stress and inflammation, protecting kidney function in the context of periodontitis (46). Periodontal therapy may serve as a non-pharmacological strategy to lower systemic oxidative stress and improve CKD outcomes, though more clinical trials are needed to confirm this benefit (21).

Conclusion

Hemodialysis patients often experience oral microbiota dysbiosis, characterized by an imbalance in the composition of oral microbial communities. Hemodialysis patients often exhibit poor oral health. Oral health issues, such as periodontal disease, are more prevalent in HD patients compared to healthy individuals. Several factors contribute to this, including xerostomia resulting from salivary changes, compromised immunity and the accumulation of uremic toxins. The complex relationships among periodontal disease, HD duration, and systemic conditions like diabetes further exacerbate oral health problems. Salivary changes are common in HD patients and can significantly impact their oral health. There are alterations in saliva urea and calcium levels, as well as pH values, in HD patients compared to healthy controls. Reduced saliva flow can lead to an increased risk of dental caries and periodontal infections. Furthermore, a decrease in saliva pH during HD is related to poor oral hygiene, especially in diabetic patients. Managing oral microbiota dysbiosis requires maintaining good oral hygiene is essential for preventing and managing oral health issues in HD patients. Regular brushing, flossing, and professional dental cleanings can help reduce the risk of oral infections and inflammation. In some cases, antimicrobial therapies may be necessary to treat specific oral infections. Finally, probiotics and prebiotics may help restore a healthy balance of gut microbiota.

Authors' contribution

Conceptualization: Mayyadah Hameed Rashid.
Data curation: Haifaa Abdulameer Radhi, Aseel Abdulameer Radhi.
Investigation: Aseel Abdulameer Radhi, Haifaa Abdulameer Radhi.
Project administration: Mayyadah Hameed Rashid.
Resources: Aseel Abdulameer Radhi.
Software: Haifaa Abdulameer Radhi.
Supervision: Mayyadah Hameed Rashid.
Validation: Aseel Abdulameer Radhi.
Visualization: Haifaa Abdulameer Radhi.
Writing–original draft: Mayyadah Hameed Rashid.
Writing–review & editing: Haifaa Abdulameer Radhi, Aseel Abdulameer Radhi.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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