

## Case Report

## Oral Health in Chronic Kidney Disease

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Chronic kidney disease (CKD); Periodontitis

## Abbreviations

CKD: Chronic Kidney Disease

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Chronic Kidney Disease (CKD) presents a major public health challenge with an estimated global prevalence of 11-13% [1]. Around 0.1% of the populations of developed countries live with end-stage renal disease, so that in the UK there are 60,000 patients dependent on renal replacement therapy in the form of dialysis or a kidney transplant [2]. In the earlier stages of CKD patients may be asymptomatic and unaware of their diagnosis, but demonstrate proteinuria and reduced renal excretory function [3] and are exposed to significant risks of cardiovascular disease and other complications [4].

The relationship of CKD and oral health is varied and complex. CKD has been said to protect against caries, because of the effect of salivary urea in increasing oral pH [5]. However many patients with CKD, particularly in advanced disease, complain of a range of oral symptoms including altered (often metallic) taste, halitosis, stomatitis and xerostomia [6]. Objectively both in patients and experimental models, CKD has been shown to reduce salivary flow rates and alter salivary biochemistry, with increased concentrations of urea and creatinine and high oral pH [7-9]. Such oral disease may have implications for the quality of life, nutritional status and the general health of patients living with CKD [10]. Furthermore, some research suggests that patients with CKD may have limited access to dental care, and have poor oral hygiene routines [11,12].

Importantly, the most robust evidence available associates CKD with a high prevalence of gingival disease and periodontitis. Pooled data suggest that roughly 20% of dialysis patients have no teeth, and over half of those that remain have significant periodontitis, with mean periodontal probing depths of 2.3mm and clinical attachment loss of 3.5mm [6]. Another meta-analysis found a similar, consistent association between CKD and periodontitis with an odds ratio of 1.65 compared to the general population [13]. Data from the RIISC cohort patients in the UK found that CKD patients had an odds ratio of 4.0 compared to community-matched controls for all forms of periodontitis and 3.9 for the severest form of the disease [14]. In the

NHANES III dataset from the US, subjects with all stages of CKD had a combined prevalence of periodontitis of 12.9% compared to 7.5% in subjects without CKD, and a trend was seen towards a dose-dependent increase in periodontitis rates in more severe stages of CKD that reached statistical significance in non-white populations [15].

There are well established associations in the general population between periodontitis and both cardiovascular disease and all-cause mortality [16,17]. This association holds true in CKD, as CKD patients with periodontitis have an increased mortality compared to CKD patients without periodontitis, a deleterious effect similar in size to the effect of suffering diabetes as a comorbidity [18]. A similar increase in mortality in CKD patients with periodontitis compared to those without was found in a recent meta-analysis, although they were not able to establish a specific link with cardiovascular disease [19]. Furthermore, CKD patients with periodontitis have been shown to have higher levels of systemic inflammation and malnutrition compared to those with better oral health [20,21], and this systemic inflammatory milieu, driven by periodontal inflammation, with oxidative stress and endothelial dysfunction, may account for some of the excess cardiovascular disease seen in the CKD patient population.

Periodontitis is a complex inflammatory disease, and it is unclear exactly what factors cause its increased prevalence in CKD patients. Patient factors, including smoking, poor oral hygiene and limited engagement with dental screening programmes may be contributory. It is possible that CKD mineral and bone disease, in which rapid bone turnover driven by secondary hyperparathyroidism disrupts normal bony architecture, may contribute to periodontal bone loss and loosening of tooth attachment. CKD is associated with functional abnormalities of the immune system, including of neutrophil activity, which are cells known to be important in maintaining periodontal health [22,23]. It is also possible that alterations in salivary biochemistry cause changes in bacterial populations in the oral cavity, resulting in a different level of microbial pathogenicity to the gingival tissues.

In the general population there is evidence to suggest that treatment of periodontal disease improves cardiovascular and inflammatory parameters [24,25]. These results have been mirrored in the CKD population [8,26], although evidence of the long-term outcomes of such interventions is presently lacking.

As the incidence of CKD increases, the proportion of patients with CKD-associated gum disease looks set to rise accordingly. It will be important to deepen our understanding of the pathological mechanisms involved and of the benefits of periodontal treatment not just on periodontal health but also on critical patient end-points such as cardiovascular events and mortality.

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