World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(REVIEW ARTICLE)

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Analysis of salivary pH changes induced by metabolic acidosis in chronic kidney disease and the impact on oral microbiota and periodontitis: Narrative review

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World Journal of Advanced Research and Reviews, 2025, 28(03), 1800-1803

Publication history: Received 17 November 2025; revised on 23 December 2025; accepted on 25 December 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.28.3.4261

Abstract

Background: To be able to regulate the balance between acids and bases in the body, physiologically the human body has a regulatory mechanism that can control acid-base concentration. Disorders that occur in the mechanism of regulating this balance can cause diseases that can occur in the oral cavity.

Purpose: This narrative review aims to provide an explanation of the relationship between changes in salivary pH due to metabolic acidosis of chronic kidney disease, periodontitis and the oral microbiota.

Methods: Articles in PubMed, Google Scholar, and ScienceDirect were searched using the keywords metabolic acidosis, periodontitis, chronic kidney disease, salivary pH, and oral microbiome.

Results and Discussion: Chronic kidney disease causes the salivary pH to become more alkaline resulting in a change in the composition of the oral microbiota and an increase in the pathogenic bacteria that cause periodontitis.

Conclusion: Changes in salivary pH due to chronic kidney disease lead to an increased risk factor of someone to be diagnosed with periodontitis and an increase in the diversity of the oral microbiome.

Keywords: Metabolic Acidosis; Periodontitis; CKD (Chronic Kidney Disease); Salivary Ph; Oral Microbiome

1. Introduction

All organ systems require a stable pH to function optimally. Systemic arterial pH is normally maintained between 7.35 and 7.45. Conditions related to disturbances in blood pH balance may manifest as acidosis or alkalosis. Acidosis is a condition in which the blood pH becomes acidic because it falls below the normal range, whereas alkalosis is a condition in which the blood pH becomes alkaline because it rises above the normal range.

The human body can produce acids as metabolic by-products derived from various physiological processes, including respiration and fat breakdown. Excess acid in the body can lead to numerous health problems, such as rapid and deep breathing, and in severe cases may result in coma. To regulate acid-base balance, the human body physiologically possesses mechanisms that control acid and base levels.

The body forms a bicarbonate buffer solution (a base) in the blood, which functions to neutralize acidic conditions resulting from increased acid production through metabolism. The body eliminates excess acid through respiration in the form of carbon dioxide and through the kidneys in the form of ammonia. Disturbances in these regulatory

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mechanisms can lead to acidosis or excess acid accumulation, whether in the form of metabolic acidosis or respiratory acidosis.

2. Material and Methods

This study is a narrative review that examines several journals from multiple databases, including PubMed, ScienceDirect, and Google Scholar, using the keywords: metabolic acidosis, periodontitis, CKD (Chronic Kidney Disease), saliva pH, and oral microbiota. A total of 57 articles were identified, and 15 were selected based on content relevance.

3. Results and Discussion

3.1. Metabolic Acidosis and Chronic Kidney Disease (CKD)

Acidosis is a condition in which the blood contains excessively high amounts of acid. Acidosis caused by a primary decrease in HCO_3^- concentration is referred to as metabolic acidosis. When the increase in acidity exceeds the pH buffering system, the blood becomes acidic. As blood pH decreases, breathing becomes deeper and faster as the body attempts to reduce the excess acid by lowering carbon dioxide levels. Eventually, the kidneys also try to compensate by excreting more acid in the urine. However, these mechanisms can be overwhelmed if the body continues to produce excessive amounts of acid, leading to severe acidosis [5].

Chronic Kidney Disease (CKD) is a disorder in which the kidneys lose their function, and an increasing number of nephrons progressively decline, reducing overall kidney function. Normally, the body produces about 50–80 mmol more metabolic acids than metabolic bases. Therefore, when the kidneys lose function, acid accumulates in body fluids. The body's fluid buffers are usually capable of buffering 500–1000 mmol of base without increasing the concentration of H^+ in the extracellular fluid. However, when buffering capacity decreases, blood pH drops drastically, and a person may fall into a coma or even die [11], [12].

Metabolic acidosis in patients with CKD is associated with faster loss of kidney function. Several studies have shown slowed progression of CKD in patients with CKD and metabolic acidosis treated with oral alkali or dietary acid reduction. Some of these studies include measurements of renal hormones and biomarkers that reveal underlying adaptive mechanisms to correct acidosis. These studies support the idea that direct kidney damage is a consequence of acid retention, which leads to persistently elevated levels of gastric acid. Angiotensin II, aldosterone, and ET-1 cause inflammation and fibrosis, which are further exacerbated by continuous ammoniagenesis that can damage kidney tissue through complement activation [15].

3.2. Prevalence of Periodontitis in Patients with Chronic Kidney Disease (CKD)

A survey conducted by Rodríguez-Godoy et al. in 2019 involving 135 dialysis patients found that only 19 individuals (14.07%) did not have periodontitis. Meanwhile, 116 subjects had periodontitis, consisting of 15 individuals (11.11%) with mild periodontitis, 39 individuals (28.89%) with moderate periodontitis, and 62 individuals (45.93%) with severe periodontitis based on the CDC-AAP index. In addition, based on the Biofilm-Gingival Interface (BGI) index, 4 individuals (2.96%) were classified as healthy, 20 individuals (14.81%) had gingivitis, 10 individuals (7.41%) had mild pocket bleeding, 20 individuals (14.81%) had moderate pocket bleeding, and 81 individuals (60%) had severe pocket bleeding [13].

A second survey conducted by Cholewa et al. on 128 hemodialysis patients—25 of whom were edentulous—showed that only 1 person did not have periodontal disease, 62.14% had gingivitis, and 36.9% had moderate or severe periodontitis. From these data, it can be concluded that there is a high prevalence of periodontal disorders among hemodialysis patients [2].

3.3. Effects of Chronic Kidney Disease (CKD) on Salivary pH as a Biomarker

Biomarkers play an important role in diagnosing oral diseases for screening, determining prognosis, and evaluating disease activity and severity, in this case periodontitis. Biomarkers can be derived from microbial products, immune-inflammatory components, and host-cell enzymes produced during tissue degradation or bone resorption. In saliva, specimens that can be used as biomarkers include whole saliva, gingival crevicular fluid (GCF), dental plaque, and serum. In periodontitis, saliva as a biomarker provides information about the activity, extent, and severity of periodontal disease. Components in saliva that have the potential to serve as biomarkers for periodontitis include calcium, phosphorus, alkaline phosphatase, and pH [6], [9].

Studies have found that patients with periodontitis exhibit lower salivary pH compared to healthy individuals [7]. However, other studies have reported that periodontitis can lead to a more alkaline salivary pH [9], [10].

In patients with chronic kidney failure or chronic kidney disease, salivary pH levels increase. This is due to heightened proteolytic activity of microorganisms and phosphate deposition, which enhances mineralization of dental plaque [10]. This is consistent with other studies stating that remineralization potential increases, resulting in a more alkaline salivary pH. Additionally, the high urea content in saliva, leading to the formation of carbon dioxide and ammonia, may also contribute to the elevated salivary pH [14].

3.4. Chronic Kidney Disease (CKD) and the Oral Microbiota

In patients with chronic kidney disease, the concentration of urea in saliva increases due to metabolic disturbances. Patients experience an increase in salivary pH and flow rate. An alkaline pH creates a more conducive environment for pathogenic bacteria, allowing them to colonize the oral cavity more easily [8].

The oral microbiota in patients with chronic kidney disease shows increased diversity. Bacteria that increase in CKD patients include *Streptococcus*, *Actinomyces*, *Leptotrichia*, and 11 other genera. Meanwhile, the concentrations of six bacterial genera decrease, including *Prevotella* and *Haemophilus* [4]. Other studies have reported an increase in *Lautropia* and *Pseudomonas*, but a decrease in *Actinomyces*, *Prevotella*, *Prevotella* 7, and *Trichococcus* [8].

3.5. Sodium Bicarbonate Therapy and Chronic Kidney Disease (CKD)

A study was conducted to determine whether Sodium Bicarbonate therapy could treat metabolic acidosis. Parametric tests, non-parametric tests, and survival analyses were used to evaluate the effects of Sodium Bicarbonate on these outcomes. The results showed that in individuals with CKD stages 3–5 without advanced chronic heart failure, treatment of metabolic acidosis with sodium bicarbonate was safe and improved both kidney and patient survival [3].

Another study involved 35 patients (14 women) with an average age of 57 (±15) years, with 18 randomized to the intervention group. The mean T50 time was 275 (±64) minutes. After 4 weeks, the average change in T50 time was 4 (±69) minutes in the intervention group and 18 (±56) minutes in the rescue group (β = −25; 95% CI: −71 to 22; p = 0.298). Additionally, changes in each patient's serum bicarbonate level were not associated with changes in T50 time, based on regression analysis. Changes in serum phosphate had a significant impact on changes in T50 time (β = −145; 95% CI: −237 to −52). In this study, sodium bicarbonate showed no effect on T50 time in patients with metabolic acidosis and chronic kidney failure [1].

4. Conclusion

This review indicates that alterations in salivary pH associated with chronic kidney disease (CKD) increase the risk of periodontitis and are accompanied by greater oral microbiome diversity. CKD-related metabolic acidosis may further contribute to the pathogenesis of periodontitis. Sodium bicarbonate has been proposed as a therapeutic approach; however, existing studies report mixed findings regarding its efficacy. Notably, no published evidence has specifically addressed the effect of metabolic acidosis on the shift toward alkaline salivary pH and its potential role in the development of periodontitis.

Compliance with ethical standards

Acknowledgments

The authors would like to acknowledge the support of the Faculty of Dental Medicine, University of Airlangga, for providing the facilities required to complete this study.

Disclosure of conflict of interest

All authors declare that there are no conflicts of interest related to this publication.

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