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Original Research

Assessment of the Biochemical Indicators of Kidney Function in Patients with Gingivitis

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Abstract

Gingivitis, a chronic inflammatory condition of the gums is caused by the accumulation of bacterial plaque. While once considered primarily a localised oral disease, recent research indicates that it can also be systemic - especially when associated with systemic inflammatory conditions, such as chronic kidney disease (CKD). Chronic kidney disease (CKD) is characterised by the loss of kidney function, systemic inflammation and immune dysregulation, which may impact on gum inflammation via common pathways. There seems to be a plausible link between oral health and the health of the kidneys, with both diseases sharing features such as increased inflammatory cytokines, oxidative stress and dysbiosis. The researchers studied 43 women with gingivitis to determine whether there was an association between the gum condition and early indicators of chronic kidney disease (CKD). They examined key biochemical and haematological markers in these patients. To study markers of systemic inflammation and renal disease, we determined the white blood cell (WBC) count, blood urea and serum creatinine concentration in the blood samples. The statistical analysis showed that the white blood cell count at $10.36 \times 10^3/\mu L$ is high, and it may mean that there is systemic inflammation, which may be linked to both gingival and renal disease. This was found to be significantly greater than the test value ($P>0.05$) despite being in the high-normal range. The average of 2.48 mg/dL of serum creatinine was higher than the recommended value of 1.3 mg/dL which suggests a reduction in glomerular filtration. Moreover, there was a significant impairment in renal function, with the average blood urea level of 40.45 mg/dL, which is well above the normal range of 7-20 mg/dL. These findings support the theory that renal failure and gingivitis go

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hand in hand, where inflammation in the mouth could worsen systemic immune response and CKD. Or, the destruction of periodontal tissue may be expedited by uremic toxins and immunosuppression in CKD. This study demonstrates that patients with renal disease or at risk of renal disease should receive a dental assessment and treatment.

Keywords: Gingivitis, Biochemical Indicators, Kidney Function, Patients

Introduction

is a bacterial infection resulting in an inflammation of the gums and gingival tissue. The disease is characterised by swollen gums, but the connective tissue that supports the tooth (i.e., attachment) remains. The condition is confined to the soft-tissue region's connective tissues and gingival epithelium, according to Eknoyan (2013). Gingivitis is considered to be the most common periodontal disease. Gingivitis, which has multiple forms depending on the aetiology, duration of infection, severity and clinical manifestations (Marchesan JT et al., 2020; Shimada Y et al., 2013), is a public health problem globally. The Clinical Practice Guideline for the assessment and management of chronic renal disease (Eknoyan, G, 2013) reports that 9.1% of the global population currently suffers from chronic renal disease (Jazmati, N, 2024; Trzcionka, A et al., 2021). The World Health Organization (WHO) lists chronic kidney disease (CKD) as the 14th leading cause of death. (Wu H et al., 2024; Chaudhry A et al., 2022). Chaudhry et al. (2022) and Martin-Cabezas R. et al. (2019) report that the World Health Organization estimates chronic kidney disease (CKD) to become the sixth leading cause of death by 2040 in place of smoking. This is because treatment is costly due to high morbidity and mortality associated with the disease such as cardiovascular diseases, severe infections and diabetes mellitus (Mahendra J et al., 2022; Schütz JDS et al., 2020).

There is some evidence that gum disease can influence chronic kidney disease. Patients with chronic kidney disease (CKD) frequently suffer from generalised gingivitis. In addition, studies have shown that compared with healthy individuals, adults with CKD has an altered oral and gut microbiome. However, it is still unclear how the oral microbiome dysbiosis occurs, how this is connected to the altered immune response in patients with CKD and the clinical symptoms that result (Trzcionka, A et al., 2021; Jazmati, N., 2024). As a result, this study aimed to address the question, "How does gingivitis affect kidney function?" by exploring the association between the inflammatory disease and renal function of patients with a clinical diagnosis of gingivitis. Gingivitis is usually caused by bacterial infection. Since the condition only affects the gingival epithelium and the connective tissue, it does not result in attachment loss and epithelial migration as periodontitis. Gingivitis can be classified in many ways, according to the cause, severity, duration of infection and clinical presentation, and is one of the most common periodontal diseases. The common type of plaque-induced chronic gingivitis is the most common. Clinical signs include swollen, red, tender, shiny, and bleeding on gentle probing gingival tissues. Gingivitis is generally asymptomatic and rarely bleeds spontaneously, so many patients don't seek treatment (Rathee, M., & Jain, P. 2023).

The kidneys regulate numerous functions of the body. They are vital for the disposal of metabolic waste products and toxins, regulation of acid-base balance and fluid balance. They are involved in gluconeogenesis and the production or regulation of a variety of hormones. The kidney contains more than a million nephrons. Nephrons consist of a glomerulus and a tubule. The glomerulus generates an ultrafiltrate from other, smaller constituents after filtering out large proteins and cells from the blood. The specialised tubule has several entry points for the ultrafiltrate. Secretion, reabsorption and filtration maintain the delicate balance of electrolytes, minerals, water and

hydrogen ions in the body. The well-established primary hormonal functions of the kidney affect red cell production, calcium balance and blood pressure. The kidneys' capacity to respond to acute and chronic disease is based on their role in regulating the physiological response. Imenez Silva, P. H. I., & Mohebbi, N. (2022) and Preuss, H. G. (1993) found that a diseased kidney will have a diminished ability to perform these functions.

Link Between Gingivitis and Kidney Disease

There is no doubt that the inflammatory cytokines and the periodontal bacteria at the site of infection can enter the general circulation, induce systemic inflammatory reaction and may be related with some systemic diseases. Biological theories show a strong bi-directional relationship between periodontitis and CKD. Periodontitis may slow down renal functions by allowing the spread of germs and inflammatory cytokines from the site of local inflammation, as demonstrated by Abbud. Gingivitis occurs due to increased plaque formation, mineralisation and retention time because of decreased saliva flow. Patients with chronic kidney disease (CKD) and end-stage renal disease (ESRD) are more likely to have periodontal disease. This may be partly explained by the acidic saliva. In a 2021 study, Nazir et al. In addition to inflammation and poor oral hygiene accelerating periodontal disease, which leads to pocket formation, gum recession and ultimately bone and tooth loss, renal osteodystrophy, increased urea levels, saliva composition changes, and host factors associated with systemic diseases also contribute to the regulation of the host response to the periodontal infection (Preuss, H. 1993). Periodontitis has been recently associated with oxidative stress in chronic renal disease patients. Periodontal infections may cause atherosclerosis and reduced vascular relaxation by attaching to, penetrating and then proliferating in coronary endothelium cells. Because of the high degree of overlap in the risk factors for cardiovascular disease and chronic kidney disease, it is plausible that periodontitis has similar effects on the blood vessels of the kidneys. Periodontitis-related chronic low-grade inflammation might cause endothelial dysfunction, which might contribute to chronic kidney disease (CKD). Both conditions have inflammatory markers such as C-reactive protein (CRP). The underlying cause of periodontitis is an uncontrolled inflammatory response to the imbalance of the microbiome of the periodontium. This causes a plethora of downstream effects; increased risk of death and a plethora of NCDs, including CKD, to name a few. Zou et al. (2024) have discovered that periodontitis is a non-conventional risk factor for diseases such as chronic renal disease and that there are shared risk factors between periodontitis and NCDs.

Material and methods

Table 1. Kits were used in this study.

Kits	Origin
Urea Kit and Creatinine	Zybio,Inc , China

Sampling of Blood

In particular, this research examined the results of 43 blood samples from those with gingivitis (20 to 70 years of age) and compared them to 25 healthy individuals (20 to 70 years of age) who were used as a control group. The participants who were diagnosed at the dental clinic through a certain clinical test had 5 ml of blood taken from a vein, using a syringe. The blood was then divided into two tubes for two different types of tests: a complete blood count (CBC) test which required 2 ml of blood in an EDTA tube, and a separate tube to draw a sample of blood for serum extraction (3 ml in a gel tube). The gel tube was spun for 5

minutes to separate the serum. It was used to test the kidney functions (urea and creatinine).

Assessments of Blood and Kidney Functions

An whole blood count

The blood was first mixed to ensure it didn't clot and then placed into an EDTA tube. It was then analysed with a full set of tests by the complete blood count (CBC) machine for several markers.

Evaluation of Renal Function

The blood was collected in a gel tube and allowed to clot. Then the tube was centrifuged to isolate the serum. Once separated, the serum was carefully returned to the Automatic Biochemistry Analyser to be analysed using new analytical tubes; urea and creatinine kits were used according to the manufacturer's instructions. The instruments used to measure urea and creatinine levels measured colour or light intensity changes that occurred due to chemical reactions between the target molecules and the chemicals in the kits.

Results

The use of a one-sample t-test of the mean white blood cell (WBC) count

The One-Sample T-Test statistical test was used to compare the average white blood cell (WBC) count in the sample to the hypothetical value of 5. Descriptive data (Table 2) shows that the sample size was 43 patients. The mean white blood cell count was 10.3633, the standard deviation was 3.47644 and the standard error of the mean was 0.53015.

Table 2. The one sample statistics test will show the difference between the patients and the control group in terms of white blood cell count.

	N	Mean	St. Deviation	St. Error mean
WBC	43	10.3633	3.47644	.53015

The T-Test results show that the mean of the sample is significantly different from the reference value. The result was highly significant ($p < 0.05$) with the two-tailed significance value of ($p = 0.000$) and the value of t calculated as 10.116 with 42 degrees of freedom (df = 42). The difference between the sample and reference value was 5.36326 with a 95% confidence interval between 4.2934 and 6.4331 (Table 3).

Table 3. One sample test of word count (WBC).

	t	df	Siq.(2-tailed)	Mean difference	95% confidence interval of the difference	
					Lower	upper
WBC	10.116	42	.000	5.36326	4.2934	6.4331

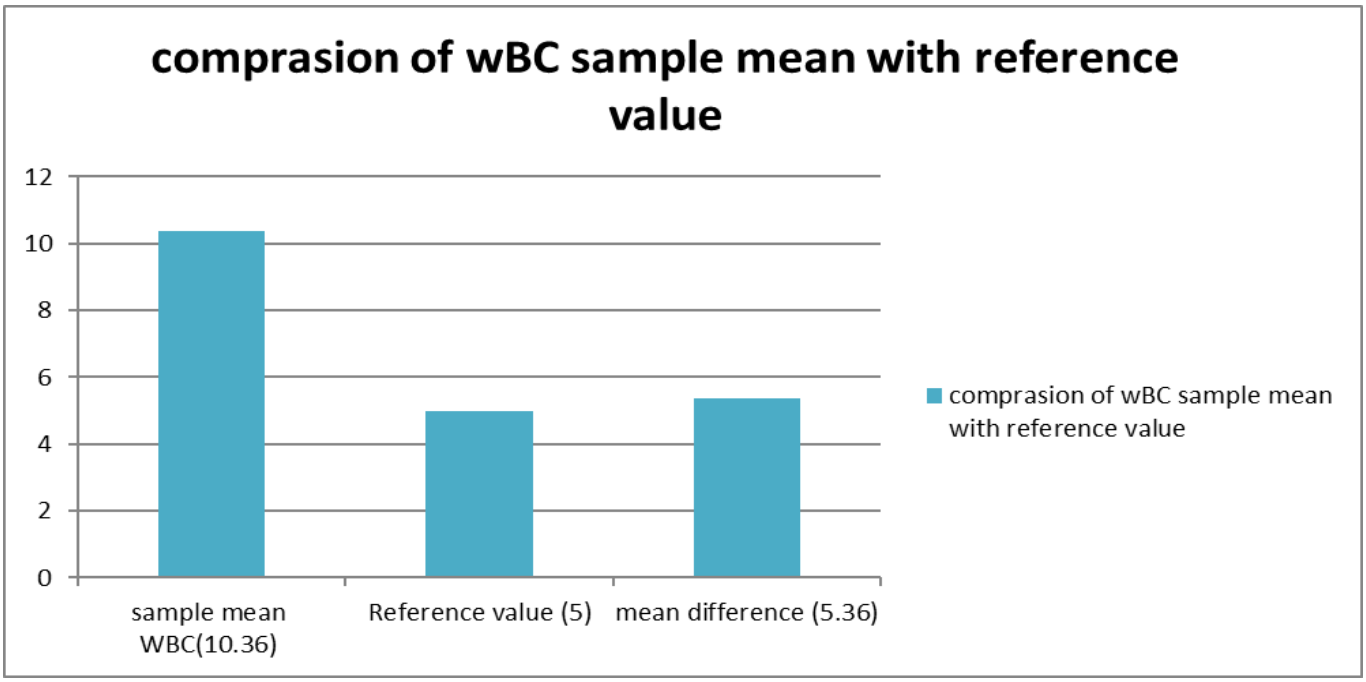

Figure 1: Comparison of the mean white blood cell (WBC) to reference values.

A One-Sample T-Test of Urea Concentration

The statistical test used was the One-Sample T-Test, which compared the mean urea (UREA) concentration in the sample we analysed to a reference value of 12.5. From the descriptive statistics, we know that there were 43 patients in the sample and that their average urea concentration was 43.1326 with a standard deviation of 31.41827 and margin of error of 4.79124. Section 4

Table 4. Perform a one-sample t-test to analyse the difference in Urea between the patient and control.

	N	Mean	Std. deviation	Std. error mean
Urea	43	43.1326	31.41827	4.79124

The T-Test results shown that the sample's mean was significantly different from the reference value. The calculated t-value was 6.393, with 42 degrees of freedom (df = 42), and the two-tailed significance (p = 0.000), indicating a high level of significance (p < 0.05). The difference in means between the two values was 30.63256, with a 95% confidence interval ranging from 20.9634 to 40.3017. These findings confirm the claim made in Table 5 that the mean urea value is significantly higher than the reference value.

Table 5. Urine one-sample test.

	T	df	Siq. (2-tailed)	Mean difference	95% Confidence interval of the difference	
					Lower	upper
Urea	6.393	42	.000	30.63256	4.2934	6.4331

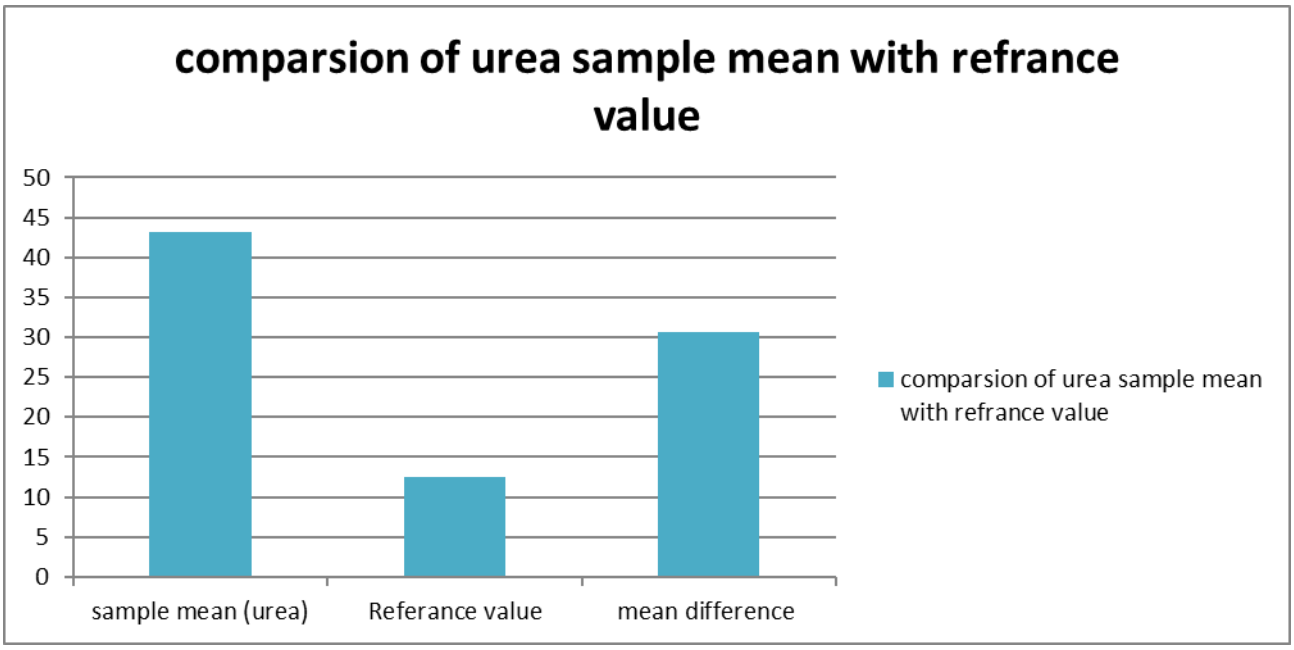

Figure 2. Average of the Urea sample with the reference value.

Analysis of the average of creatinine levels using one-sample t-test

A One-Sample T-Test was performed to compare the average blood creatinine level in the sample group to an estimated reference value of 12.5 milligrams per decilitre. The descriptive statistics for the sample (43 patients) are shown in Table 6.

The mean creatinine was 1.1321, the standard deviation was 0.94118 and the standard error of the mean was 0.14353.

Table 6. Describe the difference in creatinine levels, using a one-sample statistics test, between the patient group and the control group.

	N	Mean	St. Deviation	St. Error mean
creatinine	43	1.1321	.94118	.14353

The T-test results indicated that the mean of the sample was significantly different from the specified value. The estimated t-value was 79.203 (df = 42) with a p-value of less than 0.05 and the two-tailed significance (Sig. 2-tailed) was 0.000.

Table 7 shows that the 95% confidence interval for the difference between the actual and predicted values ranged between 11.6576 and 11.0783 with a mean difference of 11.36791.

Table 7. The creatinine one-sample test.

	t	Df	Siq. (2-tailed)	Mean difference	95%confidence interval of the difference	
					Lower	upper
creatinine	-79.203	42	.000	11.36791	11.6576	11.0783

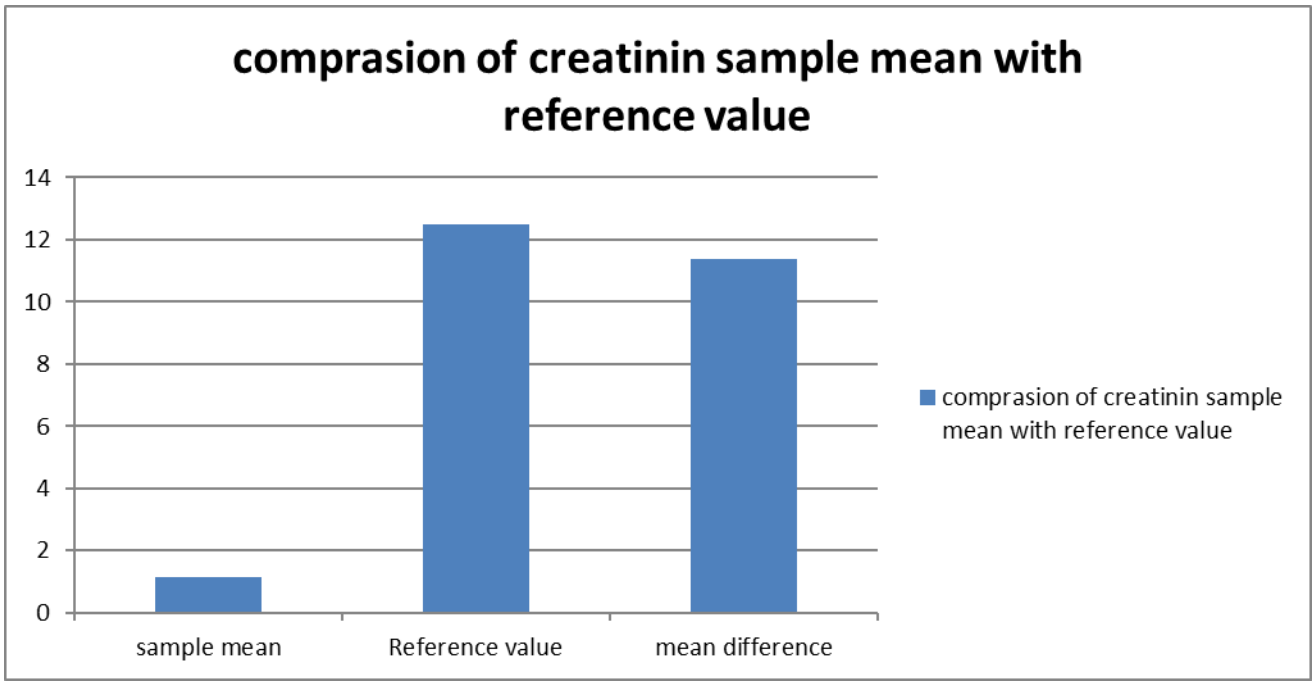

Figure 3. The average value of the sample creatinine versus the reference value.

Discussion

White blood cell count (WBC) and its link to CKD and gingival disease

White blood cells (WBCs) play an important role in the immune response to infection and inflammation. In this study the mean white blood cell (WBC) count in the patients was 10.36 x10^3/µL, which is above the reference range (4.0-10.0 x10^3/µL). Inflammatory status is a typical feature of patients with chronic kidney disease (CKD), such as this mild leukocytosis. Inflammation is a recently identified non-conventional risk factor for the development of chronic kidney disease (CKD). High white blood cell counts are associated with endothelial dysfunction, oxidative stress and the development of atherosclerosis, and are often found in renal patients (Vanholder, R., et.al. 2018). Similarly, inflammation is involved in the development of chronic kidney disease (CKD), perhaps through cytokines, which induce glomerulosclerosis and tubulointerstitial fibrosis. Importantly, inflammatory oral diseases, such as gingivitis, also affect white blood cell count. There is a lot of data from many studies that chronic kidney disease and periodontal disease are inversely related. Bacterial and inflammatory products which leak into the bloodstream from the gums can lead to low grade systemic inflammation and can cause kidney damage (Abbud, M. et al 2007). The vicious cycle is further reinforced when there is a higher risk of periodontal infections in chronic kidney disease (CKD) due to uremia and immunosuppression. Increased white blood cell counts (WBC) may be a common link in chronic kidney disease and periodontal disease. Systemic inflammatory markers, such as C-reactive protein (CRP) and white blood cell count (WBC), are commonly raised in chronic kidney disease (CKD) and periodontitis (Keller, A., et al. 2015). This would suggest that oral disease is a marker of systemic inflammation, which may affect renal disease.

Increased gingivitis risk, blood creatinine levels and chronic kidney disease

Serum creatinine is a biochemistry indicator that is often used to assess kidney function. It is normally filtered and excreted by the kidneys as a breakdown product of muscle creatine phosphate. This study's findings demonstrate reduced glomerular filtration ability as the average serum creatinine concentration was 2.48 mg/dL, which is above the normal range for adults (0.6-1.3 mg/dL). The high creatinine level is indicative of chronic kidney disease (CKD), where the accumulation of nitrogenous waste products in the

blood is due to impaired nephron function. A reliable indicator of the staging and progression of chronic kidney disease (CKD), the concentration of blood creatinine rises in direct proportion to the decline of glomerular filtration rate (GFR). Chronic hypercreatininemia (reflecting both acute and chronic kidney injuries) is often associated with systemic metabolic dysfunction, including acid-base disorder, anaemia and heart failure. It is interesting to note that there is recent evidence that potentially links biochemical changes associated with chronic renal disease with various mouth diseases such as gingivitis and periodontitis. Impaired immunity is often seen in advanced chronic kidney disease (CKD). This increases their risk of infection and impairs wound healing, both of which can have an effect on the gingival tissues (Chen, L. et al 2006). Also, the pathophysiology of periodontal inflammation may involve systemic oxidative stress and accumulation of uremic toxins, which may be reflected in high levels of creatinine. Gomes-Filho, I., et al. (2021) reported that patients with CKD and elevated creatinine levels were more likely to have severe gingival inflammation, increased pocket depth, and increased clinical attachment loss. Serum creatinine levels may be a secondary indicator of the inflammatory burden in the oral cavity, as well as a renal marker, in patients with systemic conditions. And, periodontal disease could contribute to renal dysfunction by impairing the renal microcirculation and systemic spreading of pro-inflammatory cytokines and endotoxins. High creatinine levels might be a cause or consequence of the reciprocal relationship between the kidneys and the periodontal tissues.

Urea levels, chronic kidney disease and gingevitis

Urea, also known as blood urea nitrogen (BUN), is a critical indicator of kidney function and protein metabolism. In this research, female patients with gingivitis (n = 43) had an average blood urea concentration of 40.45 mg/dL, which is much higher than the expected value of 7-20 mg/dL. There was a statistically significant rise in urea concentration when compared with the reference value of 17 mg/dL, which implied that renal excretion was insufficient, and a systemic metabolic disorder was present. Urea, a waste product of protein metabolism that forms in the liver, is primarily eliminated by the kidneys. A decrease in the glomerular filtration rate (GFR) and chronic kidney disease (CKD) results in an accumulation of urea and other nitrogenous wastes in the blood. This disease, known as uremia, can induce a variety of metabolic and systemic changes, including electrolyte imbalances, immune dysfunction and cardiovascular disease (Vanholder, R., 2018). There is now evidence that urea is not only an indicator of kidney function, but also enhances protein carbamylation, which alters their structure and function and possibly accelerates tissue injury, and it has direct toxic effects on endothelial cells. Increased urea levels being associated with increased mortality in patients with CKD means that urea is no longer considered a passive indicator of disease (Ni, Z., et al. 2017). Recent evidence reveals that elevated systemic urea levels can cause oral symptoms, particularly in CKD patients. The action of urease on urea by the oral microbiota results in the formation of ammonia and elevation of saliva pH, leading to calculus formation and causing gingivitis (D'souza, L., et al., 2023). In addition, the local immunity of the mouth is impaired in uremic patients because of immune dysfunction. The accumulation of uremic toxins, including urea and its breakdown products, could also contribute to the inflammatory process in the gums. In light of this association, chronic kidney disease (CKD) and gingival disease (GD) may feed each other and result in a spiralling inflammatory response. Research has demonstrated a link between BUN and clinical periodontal parameters such as gingival index (GI), plaque index (PI) and pocket depth. Moreover, urea levels are associated with gingival bleeding and plaque. Ioannidou and Swede (2011) showed urea plays an important role in the aetiology of periodontal inflammation in people with systemic disease via direct chemical and immunologic mechanisms.

Conclusion

- Impairment of renal function and associated blood biomarkers such as creatinine, urea and white blood cell (WBC) count are typical features of chronic kidney disease (CKD), a prevalent condition.
- The findings of this study demonstrate increased prevalence of gingivitis in chronic kidney disease (CKD) patients with elevated levels of serum creatinine and urea, which indicates a likely link between oral and kidney health.
- The increased white blood cell (WBC) count in some of the study participants, which suggests chronic inflammatory disease, may be due to dental disease or the systemic inflammatory burden of chronic kidney disease (CKD).
- Gingival inflammation may worsen the renal disease via systemic immune response, which is why regular oral exams for CKD patients are crucial, as shown in our study.
- Our findings show the need for collaboration between dentists and nephrologists in the treatment of patients with chronic kidney disease (CKD).
- To improve patient health, we need to routinely measure renal and inflammatory factors. This will facilitate early detection and treatment of issues.

Recommendations

- Make it clearer how oral bacteria can increase the risk of chronic renal disease.
- Find out how much patients with renal disease know about the importance of good oral health and how to prevent periodontal disease.
- Find out how common periodontal disease is in people with healthy kidneys and people with chronic renal disease
- Recommend ways, such as improved oral hygiene, to minimise the impact of gum disease on kidney function.
- Encourage people with renal disease to regularly see the dentist and how that may prevent disease.

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