The Peerian Journal

Open Access | Peer Reviewed

Volume 33, August, 2024
Website: www.peerianjournal.com

ISSN (E): 2788-0303
Email: editor@peerianjournal.com

The bidirectional relationship between periodontal disease and diabetes mellitus

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Abstract

This study explores the complicated relationships between gum inflammation severity, inflammatory markers (IL-6 and CRP), and fasting blood sugar levels, linking oral and systemic health. A study of mild, severe, and moderate gum inflammation groups found significant differences in IL-6, CRP, and fasting blood sugar.

IL-6 levels are much greater in severe gum inflammation, indicating inflammation. Additionally, severe gum inflammation raises CRP, suggesting a systemic inflammatory response to periodontal disease. Fasting blood sugar differs for severe and mild gum inflammation. This disparity indicates glucose metabolism issues and oral-systemic health. Chronic inflammation and reduced insulin sensitivity from severe gum disease increase fasting blood sugar and diabetes risk. These findings emphasize dental-systemic health. Monitor gum inflammation patients' IL-6, CRP, and fasting blood sugar to assess severity and systemic consequences, the study's bidirectional relationship may enhance health outcomes through focused interventions, preventive measures, and collaborative healthcare.

Key word: Periodontal disease, Diabetes mellitus, Interleukin-6, and C-reactive protein (CRP)

Introduction

Periodontal disease meaning a **c**hronic gum inflammation. Mostly plaque, bacteria's film. Poor dental hygiene can cause periodontitis and gingivitis (Sedghi, et al., 2021). Infection from gingiva. Plaque hurts gums. Brushing or flossing red, swollen gums with gingivitis bleeds. The tooth-supporting parts are salvageable (Aral, K., 2020). Periodontitis can result from untreated gingivitis. Inflammation invades teeth and bone. Pockets in teeth collect bacteria and debris. Bacteria and chronic immune system toxins harm bone and connective tissues (Liccardo, et al., 2019 and Scannapieco, F., 2020). Diabetes mellitus causes high blood sugar. Poor insulin synthesis and cell responsiveness can cause this syndrome (Cole, J. B., et al., 2020). Diabetic problems are particularly common in Types 1 and 2. Itching, weight loss, fatigue, reduced vision, poor wound healing, and frequent urination are diabetic symptoms. Diabetes is diagnosed by blood glucose testing (Himanshu, et al., 2020). Diabetes and periodontal disease interact. This means that diabetics are more likely to acquire periodontal disease, which can affect diabetes treatment. Multiple factors complicate the relationship:

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1-Diabetes as a Risk Factor for Periodontal Disease:

Diabetes can promote oral bacterial development due to elevated blood glucose levels. This may raise gum disease risk. Diabetes weakens the immune system, making gum infections harder to fight. Diabetes can increase or prolong inflammation, making gum inflammation (gingivitis) and periodontitis more likely (Balaji, et al., 2019).

2-Periodontal Disease as a Factor Affecting Diabetes:

Periodontal disease complicates diabetes. Gum inflammation can cause insulin resistance and blood sugar issues. Periodontal disease worsens renal and cardiovascular diabetes. Diabetes and periodontal disease result from smoking, poor tooth hygiene, and poor nutrition. Both diseases benefit from risk factor management (Graves, et al., 2020). Protein cytokines include Interleukin-6. Immune system cytokines help cells interact. T lymphocytes, macrophages, and others create IL-6. It controls the immune system, especially during inflammation (Rose-John, et al., 2018). Inflammation is linked to IL-6. The immune system inflames during damage or infection, but chronic inflammation can harm health (Ridker, et al, 2021). Inflammation causes the liver to create CRP. Inflammation, infection, and tissue injury raise CRP levels considerably. It is a popular blood test marker for inflammation (Wang, L. et al., 2020). The inflammatory marker CRP is widely utilized. Increased blood CRP levels indicate inflammation (Hart, P. C., et al., 2020).

Material and methods

Subjects and study design : This study includes 80 individuals suffering from gum inflammation along with diabetes. Among them, 40 individuals experience severe gum inflammation with abscess formation, while the remaining 40 individuals have mild gum inflammation. All participants are diagnosed with diabetes. The samples were collected from specialized dental centers in Iraq, specifically in Baghdad, during the period from October to December of the year 2023. The levels of inflammatory markers, such as IL-6 and CRP, were examined. Additionally, the fasting blood sugar levels were measured for all individuals included in the study.

Preparation of samples

Iraqi dentistry clinics provided all blood samples. Five milliliters of blood were drawn from 40 persons with severe gum inflammation and abscesses and 40 with mild gum inflammation. All participants have diabetes. The gel tube held blood samples at room temperature for 20 minutes. The serum was centrifuged at 2000 rpm for 15 minutes. In small serum aliquots, IL-6, CRP, and fasting blood sugar were measured.

Determination of IL-6, CRP, and fasting blood sugar level

Fasting blood sugar levels, C-reactive protein (CRP), and IL-6 were assessed in 40 patients with severe gum inflammation and abscesses and 40 patients with mild gum inflammation using a closed-system laboratory kit and the fully automated Cobas C411 and C111 devices. A chemical process that

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emits visible light upon an excited electron's return to ground state was used to test Cobas C411. Light absorption, as measured by the C111 apparatus, rises as concentration rises.

Statistical analyses

SAS (2018) analyzed patient-control effects on study parameters to meaningfully compare means, T-test was utilized. Examine estimated correlations**.**

Result

In this study, 80 individuals suffering from gum inflammation, with 40 of them experiencing mild gum inflammation, while the remaining 40 individuals had severe gum inflammation. All participants were also diagnosed with diabetes. The results, as shown in Table 1 and Figure 1, revealed significant statistical differences between the groups included in this study: the first group with mild gum inflammation and the second group with severe gum inflammation. The findings indicated highly significant differences (P≤0.01) between the mild gum inflammation group compared to the severe gum inflammation group in IL-6.

Table 1: Comparison between Mild and Severe gum inflammation in IL-6

Gum inflammation	Mean ± SE of IL-6 (pg/mL)
Mild	17.01 ±0.90
Sever	29.62 ±1.57
T-test	3.610 **
P-value	0.0001
** (P≤0.01).	

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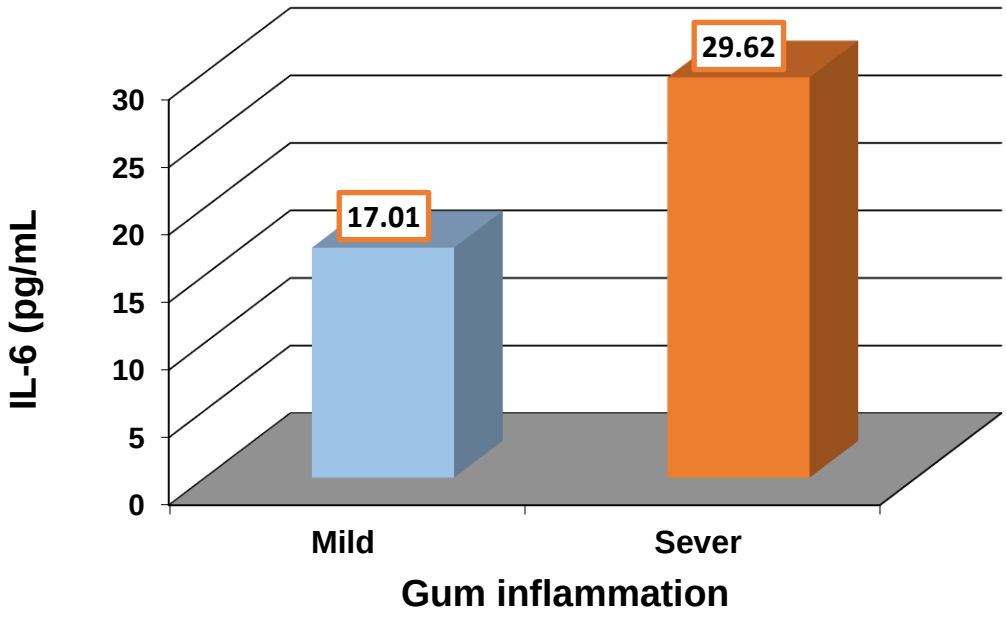

Figure 1: Comparison between Mild and Severe gum inflammation in IL-6

On the other hand, the results, as indicated in Table 2 and Figure 2, demonstrated highly significant differences (P≤0.01) between the group with severe gum inflammation compared to the group with moderate gum inflammation in CRP (C-reactive protein).

Table 2: Comparison between Mild and Severe gum inflammation in CRP

Gum inflammation	Mean ± SE of CRP (mg/dL)
Mild	13.99 ±0.42
Sever	46.62 ±1.64
T-test	3.381 **
P-value	0.0001
** (P≤0.01).	

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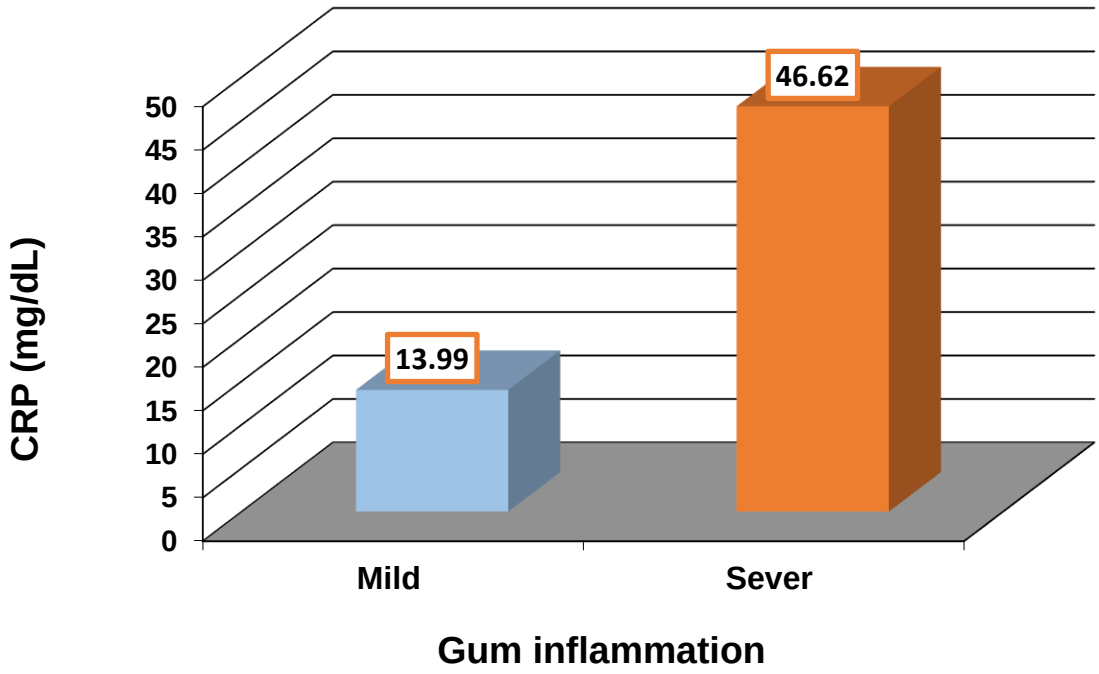

Figure 2: Comparison between Mild and Severe gum inflammation in CRP

Meanwhile, the results revealed highly significant statistical differences (P≤0.01) in fasting blood sugar levels between the group suffering from severe gum inflammation compared to the group with moderate gum inflammation, as illustrated in Table 3 and Figure 3.

Table 3: Comparison between Mild and Severe gum inflammation in Fasting blood sugar -FBS

Gum inflammation	Mean ± SE of F.B.S. (mg/dL)
Mild	138.45 ±3.61
Sever	295.67 ±13.11
T-test	27.086 **
P-value	0.0001

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** (P≤0.01).

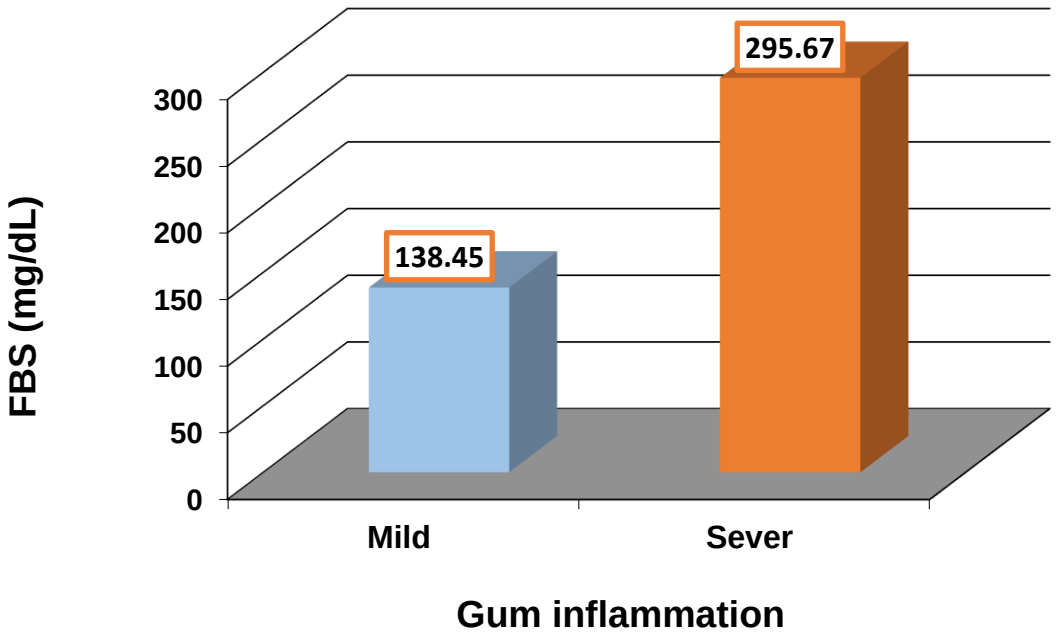

Figure 3: Comparison between Mild and Severe gum inflammation in FBS

Discussion

The results, as presented in Table 1 and Figure 1, indicate significant statistical differences between the groups involved in this study: the first group with mild gum inflammation and the second group with severe gum inflammation. The findings revealed highly significant differences (P≤0.01) in IL-6 levels between the group with mild gum inflammation compared to the group with severe gum inflammation. High IL-6 levels indicate immune response and inflammation. Research demonstrates that severe gum inflammation elevates IL-6 levels more than mild gum inflammation. IL-6, an anti-inflammatory cytokine, is associated to gum inflammation, which helps explain oral inflammation. Keles Yucel, Z. P., et al. (2020) say bacteria on teeth and gums cause gingivitis, an immunological and inflammatory reaction. Gum inflammation from pathogenic microorganisms and tissue injury increases IL-6. Gingivitis inflammation boosts IL-6. IL-6 controls inflammation and fights germs. According to Gheorghe et al. (2023), dangerously high IL-6 levels can aggravate gum and tissue inflammation.

In general, evidence links high IL-6 levels to gum inflammation. Gum health can be assessed by measuring IL-6 levels, which can indicate disease development and inflammation. IL-6

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levels rise in inflamed tissues due to inflammatory reactions. Severe gingivitis may affect IL-6 levels significantly and cause inflamed and bleeding gums. Expect IL-6 to rise with gingivitis. However, case variables may determine the magnitude of this increase. IL-6 levels may vary more in acute gingivitis, this agree with Gheorghe, et al, (2023). The levels of C-reactive protein differ considerably ($P \leq 0.01$) between the groups with moderate gum inflammation and those with severe gum inflammation, as illustrated in Table 2 and Figure 2. CRP is produced by the liver in response to inflammation and often indicates systemic inflammation. C-reactive protein measures gum inflammation, as found by Pejcic, et al, (2011). The extremely significant variations in CRP levels between severe and moderate gum inflammation groups show that severe gum inflammation causes a stronger systemic inflammatory response. One possibility is that severe gum inflammation releases more inflammatory mediators, including CRP, into the circulation. A more extensive and strong inflammatory response may be associated with severe periodontal disease, this agree with Machado, et al, (2021).

Increasing data suggests persistent gum inflammation has systemic effects. Gingivitis and periodontitis result from the immune system's response to oral bacteria. Immune response causes gum inflammation. Immune cells release IL-6 and other pro-inflammatory cytokines during gum inflammation and infection. IL-6 controls long-term and short-term immune responses. The liver produces acute-phase proteins like CRP in response to IL-6. Bloodstream CRP indicates systemic inflammation. High blood IL-6 and CRP levels indicate systemic inflammation. Systemic inflammation may cause many health issues. IL-6 and CRP levels in gum inflammation patients reveal the intensity of the inflammatory response and its systemic effects. High levels of these markers may indicate a stronger immune response and a higher risk of systemic illness. this agree with Bansal, et al, (2014). The results indicating highly significant statistical differences ($P \leq 0.01$) in fasting blood sugar levels between the group suffering from severe gum inflammation and the group with moderate gum inflammation, as presented in Table 3 and Figure 3, are noteworthy. Fasting blood sugar variations imply a link between moderate and severe gum inflammation and glucose metabolic abnormalities. This may indicate a bidirectional link between oral and systemic health. Severe gum inflammation is an immunological reaction to gum microorganisms. Local inflammation can release pro-inflammatory cytokines and other mediators into the circulation, causing systemic inflammation. Insulin sensitivity decreases with chronic inflammation. There is evidence that chronic inflammation, such as the kind seen in severe gum inflammation, can interfere with glucose metabolism. This can lead to an imbalance in blood sugar levels, potentially resulting in elevated fasting blood sugar levels over time, as shown by Chen, et al, (2015). Type 2 diabetes causes poor glucose metabolism and elevated fasting blood glucose. Gum inflammation increases the body's inflammatory load, which can aggravate or cause diabetes (Schenk, et al., 2008).

Conclusion:

Finally, gum inflammation, IL-6, CRP, and fasting blood sugar interact complexly. GUM inflammation affects CRP, IL-6, and fasting glucose.
In severe gum inflammation, IL-6 activates the immune system. Serious gum inflammation raises CRP, suggesting a systemic inflammatory milieu that may affect CHD and other diseases. Due to

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fasting blood sugar variations, severe and moderate gum inflammation alter glucose metabolism. This study found that severe gum diseases that alter insulin sensitivity may raise fasting blood sugar and diabetes risk.

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