IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 9 Ser. 11 (September. 2024), PP 45-50
www.iosrjournals.org

Peri-Implantitis And Diabetes: A Critical Review Of GlycemicInfluence On Implant Health

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Abstract:
Type 2 diabetes mellitus (T2DM) is a prevalent metabolic disorder characterized by insulin resistance and chronic hyperglycemia, adversely affecting numerous physiological processes such as immune response and wound healing. The heightened risk of periodontal diseases among diabetic patients frequently leads to edentulism, with dental implants serving as a common treatment. However, poorly controlled diabetes increases the likelihood of complications such as peri-implantitis, an inflammatory condition that impairs bone healing and osseointegration. Elevated blood glucose levels in diabetic individuals contribute to the formation of advanced glycation end-products (AGEs), which exacerbate tissue damage around implants through the upregulation of inflammatory pathways. The role of systemic inflammation in T2DM further complicates the pathophysiology of peri-implantitis. Toll-like receptor (TLR) signaling, particularly TLR2 and TLR4, is implicated in inflammatory responses, leading to elevated pro-inflammatory cytokines in diabetic tissues. These mechanisms, combined with the detrimental effects of hyperglycemia on bone remodeling, result in an increased risk of bone resorption and implant failure. Additionally, bone loss in peri-implantitis is closely linked to glycemic control. Studies indicate that both sustained hyperglycemia and glycemic fluctuations aggravate peri-implant bone loss, underscoring the importance of systemic glycemic management in peri-implant health. Moreover, local treatments alone may not sufficiently mitigate the systemic effects of diabetes on peri-implantitis progression. Finally, there is a growing recognition of the bidirectional relationship between peri-implantitis and systemic inflammation. Chronic inflammatory conditions like apical periodontitis can worsen glycemic control in diabetic patients, while elevated levels of inflammatory cytokines, such as interleukin-1β (IL-1β) and interleukin-17 (IL-17), contribute to insulin resistance and exacerbate the inflammatory response around dental implants. Future research should focus on the combined impact of glycemic control and local peri-implant treatments to improve outcomes for diabetic patients.

Keyword: *Blood glucose; Inflammation; Peri-implantitis; Systemic disorders; Type 2 diabetes mellitus.*

Date of Submission: 20-09-2024 Date of Acceptance: 30-09-2024

I. Critical Review

Peri-Implantitis and Diabetes: A Critical Review of Glycemic Influence on Implant Health

Introduction

Type 2 diabetes mellitus (T2DM) has become an increasingly prevalent metabolic disorder in recent decades, posing significant health challenges worldwide (Gomes et al., 2019). T2DM is characterized by insulin resistance and chronic hyperglycemia, which can adversely affect various physiological processes, including wound healing, immune response, synthesis of collagen and others (Shang et al., 2021). In addition, substantial evidence links T2DM with a heightened risk of periodontal diseases, which in turn can lead to tooth loss and the need for dental implants as a common treatment option for edentulism (Khandelwa et al., 2013, Enteghad et al., 2024). Despite the advancements in dental implant technology and the generally high success rates, patients with poorly controlled diabetes face an increased likelihood of complications such as peri- implantitis which could affect the soft and bone tissues surrounding the implant and can promote, in some cases, implant/prosthetic loss (Dioguardi et al., 2023).

Peri-implantitis, a chronic, inflammatory, and infectious disease (Tessarin et al., 2024), is particularly concerning for diabetic patients, as hyperglycemia is known to impair bone healing and osseointegration (Gomez-Moreno et al., 2015). The impaired ability to integrate the implant with the surrounding bone is compounded by a higher susceptibility to infections, often leading to inflammatory responses and bone resorption around the implant site (Cosyn et al., 2012). Poorly regulated blood glucose levels have been shown to negatively impact the peri-implant environment, contributing to an increased risk of implant failure (Li et al., 2021). This correlation is supported by both animal models and clinical data, which indicate that hyperglycemia elevatesthe risk of peri-implant inflammation and bone loss (Enteghad et al., 2024).

Moreover, the role of systemic inflammation in T2DM adds complexity to the pathophysiology of peri-implantitis (Dioguardi et al., 2023). The Toll-like receptor (TLR) signaling pathway, particularly TLR2 and TLR4, is implicated in the initiation of inflammatory responses in diabetic tissues (Li et al., 2021). Upregulation of these pathways in diabetic patients has been associated with increased levels of pro-inflammatory cytokines, which play a crucial role in the progression of peri-implantitis (Yu et al., 2018). Additionally, the regulation of bone remodeling processes, such as osteoplastic matrix synthesis, appears to be altered under varying glycemic conditions, further complicating the osseointegration process (Dioguardi et al., 2023).

Peri-implantitis and periodontitis are similar to each other (Tessarin et al., 2024) and given the high global prevalence of both diabetes and periodontal diseases, understanding the relationship between glycemic control and peri-implant health is of paramount importance (Enteghad et al., 2024). Although dental implant treatment can be successfully performed in well-controlled diabetic patients, poorly managed diabetes remains a significant risk factor for peri- implantitis (Li et al., 2021). Therefore, this article aims to explore the potential mechanistic links between diabetes, peri-implantitis, and glycemic control, emphasizing the need for further researchto clarify the underlying biological processes and improve treatment outcomes for diabeticpatients.

Impact of Advanced Glycation End-Products on Peri-Implantitis

Elevated blood glucose levels in chronic hyperglycemia enhance the formation and accumulation of advanced glycation end products (AGEs) in oral tissues, including gingival tissues and periodontal fibroblasts (Chiu et al., 2017). Increased interactions between AGEs and their receptors (receptors for AGEs [RAGE]) have been linked to impaired fibroblastic growth in periodontal tissues. Similar mechanisms might be involved in peri-implant breakdown in diabetic individuals. Hyperglycemia could play a crucial role in the pathogenesis of tissue destruction around dental implants in type 2 diabetes mellitus (Al-Sowygh et al., 2017).

Poor glycemic control has been extensively studied as a modifying risk factor for peri- implant tissue destruction, with a well-documented association between elevated glycated hemoglobin (HbA1c) levels and poor peri-implant parameters (Eskow et al., 2017; Lee et al., 2016). A recent 3-year longitudinal study by Gómez-Moreno et al. (2015) reported that dental implant treatment in diabetic patients showed improved outcomes in terms of peri-implant probing depth (PD) and crestal bone loss (CBL), provided that the HbA1c levels of these patients were controlled. While data exist on AGEs and their correlation with chronic periodontitis in diabetes mellitus, the correlation between clinical and radiographic peri-implant parameters and AGEs levels across different glycemic levels remains unexplored. Therefore, it is possible to speculate that peri-implant inflammatory parameters deteriorate with increasing glycemic levels (HbA1c), and that AGEs levels in peri-implant sulcular fluid (PISF) are higher in diabetic patients comparedto non-diabetic individuals.

Al-Sowygh et al. (207) investigated the influence of advanced glycation end-products (AGEs) on peri-implantitis, hypothesizing that diabetic patients exhibit more severe peri-implant inflammation and increased levels of AGEs in peri-implant sulcus fluid (PISF) compared to non- diabetic individuals. Consistent with this hypothesis, the study revealed that diabetic patients had significantly worse peri-implant parameters, such as probing depth (PD) and bleeding on probing (BOP), and those with HbA1c levels exceeding 10% showed elevated AGE levels in PISF. Additionally, a positive correlation was observed between AGE levels and both BOP and PD in patients with poorly controlled diabetes.

Chronic hyperglycemia appears to play a crucial role in exacerbating peri-implant damage (Ghiraldini et al., 2016). The persistent elevated glucose levels disrupt collagen synthesis and the maintenance of the extracellular matrix in periodontal and peri-implant tissues (Lalla et al., 2000; Ghiraldini et al., 2016). This disruption is largely due to non-enzymatic glycosylation of proteins, leading to the accumulation of AGEs. These AGEs alter collagen structure, making it less soluble and more prone to deterioration, which impairs the normal repair processes of peri- implant tissues (Goldin et al., 2006). Moreover, AGEs are associated with increased production of pro-inflammatory cytokines, such as interleukins and matrix metalloproteinases, which further contribute to inflammatory responses and tissue damage around dental implants (Al-Sowygh, et a., 2017).

The impact of chronic hyperglycemia extends to altering host tissue responses and immune functions (Turina et al., 2005). Elevated glucose levels impair neutrophil chemotaxis and phagocytosis, which compromises the body's defense mechanisms against periodontal pathogens (Turina et al., 2005). This weakened

immune response can exacerbate peri-implant tissue damage,particularly in patients with high levels of AGEs.

Bone Loss and Glycemic Fluctuations in Peri-Implantitis

Bone loss around dental implants in diabetic patients has been consistently linked to poor glycemic control (Li et al., 2021). Systematic reviews indicate that diabetes, particularly when not well-controlled, increases the risk of peri-implantitis and contributes to a higher failure rate of implant therapy (Li et al., 2021). However, there is still debate surrounding diabetes as a conclusive risk factor for peri-implantitis, with some studies failing to find a significant association betweenthe two when controlling for confounding factors.

The impact of glycemic fluctuations on peri-implant bone loss has emerged as a critical area of focus. While sustained hyperglycemia is well-known to impair alveolar bone healing, studies suggest that fluctuating blood glucose levels may exacerbate this condition even further (Enteghad et al., 2024). Studies has shown that glycemic fluctuations, as opposed to consistent hyperglycemia, result in more pronounced bone loss in peri-implant tissues (Wang et al., 2020). This suggests that erratic blood glucose control may have a greater influence on peri-implant osteolysis than previously thought.

Additionally, managing blood glucose levels effectively has been shown to reduce bone loss around implants, highlighting the importance of systemic glycemic control in peri- implantitis management (Alberti et al., 2020). Local treatments, such as anti-inflammatory or anti- osteolytic interventions, may not be sufficient to mitigate the impact of systemic hyperglycemia on peri-implant health (Li et al., 2021). The interaction between poor glycemic control and increased inflammatory responses, particularly through the activation of the TLR2/4 signaling pathway, further compounds the risk of peri-implant bone loss in diabetic patients (Andrews et al., 2015; Sun et al., 2016).

Therefore, it is plausible to infer that the effective management of blood glucose levels plays a vital role in preserving peri-implant bone integrity in diabetic individuals. Future research should aim to explore the combined effects of glycemic control and local treatments in reducing the progression of peri-implantitis, with particular emphasis on the role of glycemic fluctuations in exacerbating bone loss.

Peri-implantitis, Systemic Inflammation, and Alterations in Insulin Signaling

While peri-implantitis is primarily initiated by biofilm accumulation, which underscores the importance of biofilm control for maintaining peri-implant health, systemic factors should also be considered in treatment planning (Tessarin et al., 2024). There is evidence suggesting that, much like periodontal pockets, the peri-implant sulcus may allow bacterial infiltration into the systemic circulation, given that the tissue-implant interface is structurally weaker than the gum around natural teeth. This infiltration triggers an immune response, characterized by the recruitment of polymorphonuclear neutrophils (PMNs) and monocytes/macrophages (MMs), which may lead to heightened production of proinflammatory cytokines and potentially contribute to systemic inflammation (Nasser et al., 2023).

Although established risk factors for peri-implantitis, such as smoking and poor hygiene, a history of periodontitis, and diabetes, have been well-studied, the relationship between systemic inflammation and peri-implant diseases remains less clear. Some studies have shown similar inflammatory biomarkers in both peri-implantitis, periodontitis and apical periodontitis (AP) within the same individual, suggesting shared inflammatory pathways (Jansson et a., 2021). The regulation of periapical inflammation is a highly intricate process that involves a wide array of cells, signaling molecules, and immune responses working together to manage the infection. When the body's defense mechanisms fail to completely eradicate the infection, chronic periapical lesions can develop, acting as a barrier to limit the spread of microbes (Graunait et al., 2012; Colić et al., 2009).

Studies indicate that diabetes can worsen the effects of AP. For instance, Kohsaka et al. (1996) found that diabetic rats with AP exhibited more severe alveolar bone resorption compared to non-diabetic rats. Similarly, Garber et al. (2009) demonstrated that hyperglycemia impaired pulp healing in diabetic rats. On the other hand, AP can also aggravate diabetes. Cintra et al. (2014) demonstrated that diabetic rats with AP had elevated blood glucose and glycated hemoglobin levels, suggesting that AP contributes to impaired glucose regulation. A clinical study further showed that poor glycemic control in type 2 diabetes patients correlated with worse periapical health, as reflected by elevated glycated hemoglobin (HbA1c) levels. However, the mechanisms are not fully understood. Some studies suggest that cytokines produced locally during AP may enter systemic circulation and promote insulin resistance (Astolphi et al., 2014; Pereira et al., 2015). Pro-inflammatory mediators like interleukin-1 (IL-1) and interleukin-6 (IL-6), which are elevated in AP models, are linked to insulin resistance. IL-6, for instance, has been shown to reduce insulin-stimulated glycogen synthesis in the liver and impair glucose uptake in adipose tissue, though it can also enhance glucose uptake in skeletal muscle during exercise, where it acts as an anti-inflammatory agent by inhibiting TNF-α (Rotter et al., 2003; Hotamisligil et al., 1996).

IL-1β, another cytokine, has been implicated in the progression of type 2 diabetes by damaging

pancreatic beta cells and interfering with insulin signaling (Maedler et al., 2002). Inhibiting IL-1β through antibodies or interleukin-1 receptor antagonists (IL-1ra) has been shown to reverse some of these harmful effects (Gao et al., 2014). Additionally, elevated levels of interleukin-17 (IL-17) have been linked to insulin resistance, possibly due to its interaction with the renin-angiotensin system, which increases oxidative stress and disrupts insulin signaling (Cintra et al., 2014).

Astolphi et al. (2014) studied the effects of AP on insulin sensitivity and signaling in insulin-responsive tissues, finding that AP induced insulin resistance, particularly in muscle and adipose tissue, likely due to increased plasma TNF-α levels. Their findings highlight the importance of treating AP, as procedures like apicoectomy and dental extraction have been associated with reduced systemic inflammation. Due to the similarity of the local and systemic inflammatory processes observed in apical periodontitis and peri-implantitis, it is possible to inferthat the immunomodulatory effects of these conditions on insulin signaling are similar.

Acknowledgments

We thank the North University Center of São Paulo, UNORTE, Union of Colleges of the Great Lakes (UNILAGO) and School of Dentistry of Araçatuba, São Paulo State University (UNESP), Araçatuba, Brazil for supporting this study.

Funding: None.

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Title: Peri-Implantitis and Diabetes: A Critical Review of Glycemic Influence on Implant Health

Type of Manuscript: Critical Review

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