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The Relationship Between Diabetes Mellitus Type 2 and Periodontal Disease

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Abstract

Periodontal disease is an infection that can generate a systemic inflammatory response. Chronic inflammation as a response to periodontal disease can be the basis of the relationship between periodontal disease and type 2 diabetes. Systemic inflammation emerges as a type 2 diabetes predictor, and individuals with periodontal disease exhibit elevated levels of systemic proinflamators and poor glycemic control. Hyperglycemia in diabetes induces non enzymatic protein glycosylation, which results in the increase in advanced glycation end products (AGES.) AGES stimulate the expression of proinflammatory mediators compromised in diabetes, which are likewise associated with periodontal disease and increase risk of cardiovascular disease.

Keywords: Diabetes; Periodontal Disease; Inflammatory response; Type 2 diabetes; Hyperglycemia; Protein glycosylation; Cardiovascular disease

Abbreviations: DM: Diabetes Mellitus; T2D: Type 2 Diabetes; IL6: Interleukin 6; PGE2: Prostaglandin E2; PCR-us: Ultrasensitive Reactive C Protein

Introduction

Periodontal disease is a local infection that generates a chronic inflammatory response in tissue surrounding and supporting the teeth, producing periodontal degradation and in consequence, dental loss. If we broaden the perspective of the natural history of periodontal disease and consider periodontitis as potential factor of infectious exposition in and of itself, we then start to visualize the risk in the context of Medicine. The central hypothesis points out that periodontal infection displays an inflammatory response that goes beyond local oral levels and becomes systemic. Thus, during the immune response to periodontal infection, events that develop into the increase of local liberation of inflammation mediator (TNF alfa, IL-1b, PGE2, IL-6), which as they enter the bloodstream on account of local vasodilatation natural of the local immune event, bring as a consequence an increase of pro-inflammatory mediators on a systematic level [1].

The association between Diabetes mellitus and periodontitis has been well documented [2] and periodontal disease that had been traditionally seen as a consequence to diabetes [3] is now being seen as a two-way relationship in the sense that periodontal disease can also be a risk factor for diabetes [4,5]. Chronic inflammation as a response to periodontal infection can be the foundation of the relationship between periodontal disease and Type 2 diabetes. Systemic inflammation emerges as a type 2 diabetes predictor in individuals with periodontal disease, where it has been shown that they exhibit elevated level of systemic proinflammatories [6].

Relationship Diabetes and Periodontal Disease

The negative impact of periodontal disease on diabetes can be due to the finding that periodontal infection alters the endocrine metabolic state and increases insulin resistance. Grossi [7] has pointed out that negative gram infections and chronic endotoxemy such as the one displayed in periodontal disease results in an elevated production of Interleukin 1 (IL 1), tumoral necrosis factor alpha (TNF alpha), interleukin 6 (IL 6) and prostaglandin E2 (PGE2). These cytokines could induce insulin resistance and worsen metabolic control in diabetic patients. For example, TNF alpha inhibits insulin receptor phosphorylation, which increases resistance to it [3].

The effect on periodontal therapy on glycemic control has also been studied, having as an important point in diabetic pations to analyze if periodontal treatment improves glycemic control. Tsobgny-Tsaque et al. [8] investigated the effect of periodontal

therapy on glycemic control in pations with type 2 diabetes. They conducted a controlled clinical trial whose objective was to evaluate the effects of non-surgical periodontal treatment of chronic periodontitis on glycemic control in patients with poorly controlled type 2 diabetes (T2D). A total of 34 poorly controlled T2D patients with chronic periodontitis with known duration of diabetes of 55.5 ± 42.6 months, and HbA1c of 9.3 ± 1.3% were randomized to two groups.

The treatment group (Group 1, n = 17) received an immediate ultrasonic scaling, and escaling and a root planning together with an irrigation of povidone with 10% subgingival iodine, while the control group (Group 2, n = 17) was assigned to receive deferred periodontal treatment 3 months later. The immediate non-surgical periodontal treatment induced a reduction of HbA1c levels by 3.0 ± 2.4 points. Concluding that non-surgical periodontal treatment markedly improved glycemic control with an attributable reduction of 2.2 HbA1c points in patients with poorly controlled T2D.

Beyond the relationship between periodontal disease and diabetes, it is worth it to inquire on the impact that these two pathologies have together, and given their bidirectional relationship, the impact they could have on cardiovascular disease. Evidence is overwhelming in pointing out diabetes as a cardiovascular risk factor, and for acute myocardium heart attack. So, if periodontal disease is considered as possible risk factor for diabetes, we could also think in the same line that periodontal disease could be contributing to cardiovascular risk.

With this in mind, Latorre C, Escobar F. et al performed a study which objective was to assess the impact of diabetes on periodontal disease and on systemic inflammation markers such as ultrasensitive reactive C protein (PCR-us) in patients with and without acute cardiac arrest to myocardium. They performed a case and control study with 401 with and without heart attacks, diabetes and periodontal disease. Results showed that 43 (78.4%) of the 57 patients showed advanced chronic periodontitis, followed by moderate chronic periodontitis. (12,2%). The PCR-us in diabetic patients with advanced chronic periodontitis was 5.31mg/L. (D.R. 6.82), and in non-diabetic patients with advanced chronic periodontitis was 2.38mg/L (SD 4.42), thus finding a statistically significant difference. (p=0000).

In patients with IAM diabetes and advanced chronic periodontitis the PCR-us value was 6.16 (D.E. 0.649). Research showed that in patients with acute myocardium cardiac arrest, diabetes and periodontal disease considerably increase systemic inflammatory response. With an increase of C reactive protein. (p=0,000). In patients with IAM diabetes and advanced chronic periodontitis, the PCR-us value was 6,16 (S.D. 0,649). Research concluded that in patients with acute myocardium heart attack, diabetes and periodontal disease considerably increase systemic inflammatory response with an increase in C-reactive protein, thus together increasing acute myocardium heart attack risk [9].

Conclusion

Given this outlook on the evidence linking periodontal disease as risk factor for Diabetes Mellitus, it is worth it that medical doctors and dentists join efforts to achieve an optimal integral treatment for patients with diabetes. The doctor, visualizing the mouth as possible focus of infection that through inflammatory pathways could alter glycemic control in diabetic patients; the dentist rationalizing the impact that adequate control and treatment of periodontal disease has in systemic health of their patients. A pathology such as periodontal disease that has the possibility to increase the risk of diabetes mellitus should be important in the field of global public health.

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