

COMPARISON OF GLUCOSE LEVELS IN PATIENTS WITH DIABETES MELLITUS TYPE II AFTER A PHASE I PERIODONTAL TREATMENT

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ABSTRACT

In dental practice it is increasingly common to find patients with systemic disorders that require comprehensive treatment, due to the mutual interaction of diseases. Such is the case of periodontitis and Diabetes Mellitus.

Purpose: The purpose of this research was to observe the effects of periodontal phase I treatment on plasma glucose of both diabetic and non-diabetic patients presenting periodontal disease.

Materials and methods: Patients who agreed to collaborate were classified into two groups of 10 people each: the first group was composed of diabetic patients, while non-diabetics represented the control group. Glucose samples were taken from a drop of capillary blood, with the help of a puncture device, sterile lancets, test strips and a glucometer, before periodontal treatment and during the first and second control appointment.

Results: In the results obtained, it was observed that non-surgical periodontal treatment achieved a statistically significant decrease in glucose levels in diabetic patients ($p = 0.001$), while in non-diabetic patients it helped maintain and regulate normal levels, so no statistically significant difference was found ($p = 0.283$).

Conclusion: Periodontal disease is closely linked to Diabetes Mellitus, and that, therefore, periodontal treatment contributes to lowering glucose levels in diabetic patients, while in non-diabetic patients it helps to stabilize these levels to an adequate range.

Keywords: *Diabetes Mellitus, Periodontal disease, Plasma glucose.*

INTRODUCTION

Diabetes Mellitus (DM) and periodontal disease (PD) are two common chronic diseases that are biologically linked (1). Various epidemiological studies relate poor oral health and poor glycemic control in diabetic patients. (2-4) According to the World Health Organization, DM is a public health problem that has increased significantly in recent decades. In 2012, diabetes caused 1.5 million deaths worldwide. In 2014, according to the World Health Organization, 422 million adults worldwide suffered from diabetes, an extremely high figure compared to 108 million people detected with this disease in 1980. The worldwide prevalence of diabetes has almost doubled since that year, as it has gone from 4.7% to 8.5% in the adult population. In studies conducted by Saran et al., Grossi & Skrepcinski, Carrillo & Barnabé and Lalla et al. (5-8) it is shown that DM shows an increase due to risk factors such as overweight and obesity, as well as cardiac complications, strokes, kidney failure, leg amputation, vision loss, neurological damage and various systemic alterations. Taylor & Borgnakke, Batchelor and Benza & Pareja (9-11) describe that gingivitis and periodontitis are located as the most common periodontal diseases in the field of dentistry. Moderate or severe periodontitis, with destruction of periodontal junction tissues is less common than gingivitis, yet it is a common chronic disease that affects approximately 5 to 15% of the world's population. (12-14)

Löe (14) notes that periodontitis is considered the sixth complication of Diabetes Mellitus, while Rajkumar et al., Emrich et al., Genco & Bornakke and Tonetti et al. (15-18) they report a higher prevalence and severity of the periodontal disease of severe periodontitis in diabetics, presenting a greater loss of insertion and alveolar bone, as well as more profuse bleeding at probing, greater dental mobility and tooth loss. In these cases of PD. Periodontal treatment can contribute to better control of blood glucose levels. Each population of individuals offers different aspects of genetic, nutritional, environmental, sedentary type that can lead to different levels of glycemic control. Therefore, the objective of the study was to determine glucose levels in healthy patients and patients with type II DM after non-surgical periodontal phase I treatment.

MATERIALS AND METHODS

In a longitudinal comparative clinical experiment, patients aged between 27 and 84 were included for the first time in consultation with the Master in Periodontics of the Autonomous University of Tamaulipas with diagnosis of PD and indication of scraping and root smoothing, and instruction on care of your oral hygiene, brushing techniques and correction of habits (periodontal treatment phase I). The population was composed of two groups of 10 patients each: a group of type II diabetic patients and another group of non-diabetic patients (control group).

Both groups were composed of 5 women and 5 men. Blood samples were obtained from patients who fulfilled a 3-hour fast before periodontal treatment. To obtain these samples, the pulp of the index finger of the right hand was first cleaned with a cotton swab soaked in alcohol to remove any dirt and then dried with another dry swab to remove excess.

A sterile lancet was placed on the puncture device and operated on the uppermost surface of the patient's finger by lightly pressing the device. Subsequently, a light massage was performed on the finger until a drop of blood was observed on its surface. To measure the patient's glucose level, an ACCU-CHEK® Instant glucometer was used along with a new test strip for each test. The glucometer was placed at a 90° angle to the sample and the lower part of the test strip was carefully approached until the drop of blood was touched to obtain the measurements. The information shown by the glucometer was transcribed on the data collection sheet for further analysis. When the patients went to their control appointments, the blood sample was taken again before performing the review.

STATISTICAL ANALYSIS

An exploratory data analysis was performed obtaining the centrality statistics and variation (mean, I. C. at 95% for the mean, median, standard deviation, minimum and maximum value) of the glucose indices reported in the patients of each study group.

Subsequently, the assumptions of normality and homogeneity of variances were verified using the Kolmogorov-Smirnov tests, and the Levene test. Failing to comply with the parametric assumptions, the Mann-Whitney U test was used for independent samples and Friedman for paired samples.

All statistical tests have been handled at an alpha value of 0.05 in the package Statistical IBM SPSS STATISTICS 23.

RESULTS

To carry out the study, 20 voluntary patients were selected and divided into two groups: patients with type II diabetes mellitus and non-diabetic patients (control group), with the aim of comparing glucose levels after periodontal phase I treatment. Surgical in both study groups.

When first assessing the glucose values of systemically healthy patients, no statistically significant difference ($p > 0.05$) was found when contrasting the pretreatment rates observed, at the first and second control appointment. (Table 1) (Figure 1).

Table 1: Descriptions of the glucose variable in systemically healthy patients pre-treatment at the 1st and 2nd appointment control

Values	Systematically healthy	
	Mean	Standar deviation
Glucose		
Pre-Treatment	100.00	9.10
1° Control appointment	103.60	9.79
2° Control appointment	101.10	7.40
<i>P value</i>	0.283	

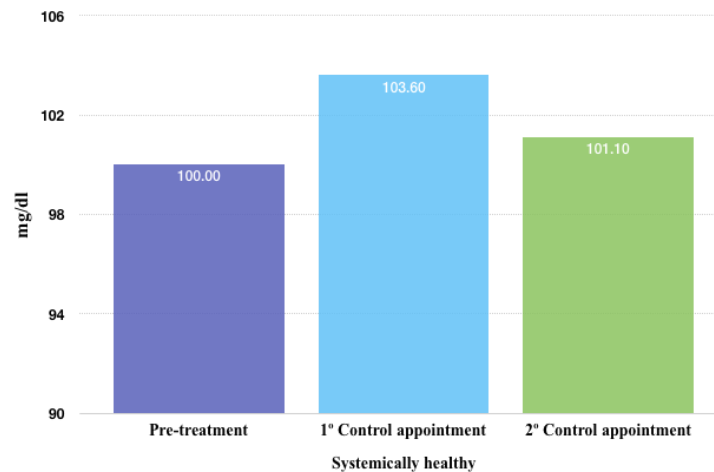


Figure 1: Average glucose in systemically healthy patients pretreatment at the 1st and 2nd control appointment.

Likewise, glucose indices were compared in the three evaluation moments in patients with diabetes mellitus, with a significant difference ($P < 0.05$) in glucose indices taken prior to treatment with an average of 210.50 mg / dl with respect to the indices of the first and second control appointment, where an average of 167.70 and 154.50 mg/dl were reported, respectively. (Table 2) (Figure 2).

Table 2: Descriptions of the glucose variable in diabetes mellitus type II patients pre-treatment at the 1st and 2nd appointment control

Values	Diabetes Mellitus Type II	
	Mean	Standar deviation
Glucose		
Pre-Treatment	210.50	77.99
1° Control appoinment	167.70	31.57
2° Control appoinment	154.50	21.31
<i>P value</i>	0.001	

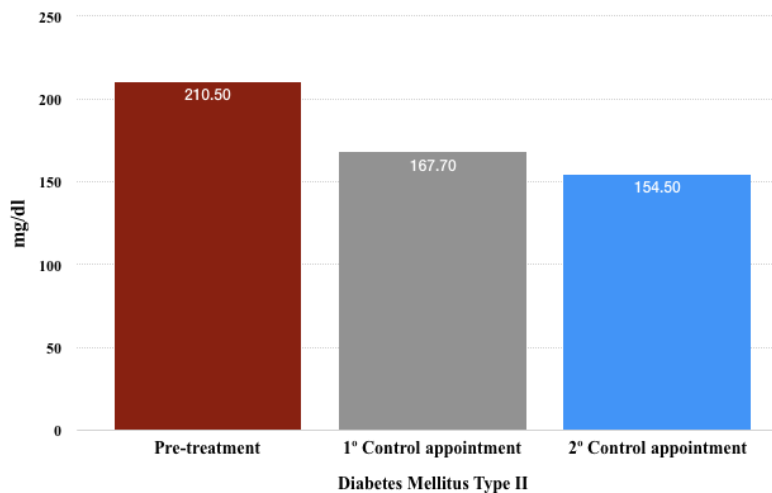


Figure 2: Average glucose in diabetes mellitus type II patients pretreatment at the 1st and 2nd control appointment.

DISCUSSION

For the reasons mentioned above we carry out this research demonstrating in our results that a satisfactory periodontal treatment leads to a better control of glucose levels in patients with Type II Diabetes Mellitus. In table 2 we can see a statistically significant difference (with a P value <0.05) of the glycemetic control of the patients before starting the periodontal treatment and during the first and second control appointment. However, in the average of the total of diabetic patients a greater decrease in glucose levels was observed when attending their first control appointment, compared to their second appointment, although the difference between both measurements was statistically significant, as evidenced by the Figure 2.

Similarly, the results obtained from the control group, as shown in Table 1, show that periodontal treatment helps maintain adequate glucose levels in healthy patients, although there was no statistically significant difference in blood levels. glucose of healthy patients throughout the three periods in which the samples were made.

The aforementioned coincides with the conclusions obtained from the investigations carried out by Rajan et al., Bukleta & Krasniqi, Aubrey and Miller et al. (19-22) to mention a few, about the effect of reducing systemic inflammatory status and improving glucose metabolism in response to non-surgical periodontal therapy.

On the other hand, Salvi et al. (23) they conducted an investigation in which they observed that the diabetic state results in a significant regulation of monocyte secretion, both of prostaglandin E2 (PG2), IL-1 and tumor necrosis factor (TNF) when compared to controls non-diabetic All this is related to a dose-response to the LPS (Lipopolysaccharide) present in many processes of Gram-negative bacteria, mainly anaerobic, involved in this type of diabetic patients, thus demonstrating that within diabetics individuals with moderate to severe Periodontitis have significantly elevated monocytic secretion of PGE 2, and FNT at the challenge of LPS.

It has been reported that patients with DM 2 clinically exhibit a low degree of systemic inflammation, including increased acute phase proteins and cytokines.

Taking into account the oral microbiome and the large number of bacterial infections that may occur, we also find a series of local or systemic reactions in the body that are known as the "acute phase response." The local reaction includes blood vessel dilation, impaired blood flow and coagulation, neutrophil and macrophage migration, lysosomal protein release and inflammation mediator formation. These mediators cause a systemic reaction characterized by fever, pain, leukocytosis, decreased plasma levels of zinc and iron and increasing the synthesis of acute phase proteins, among which are: Complement (C1 - C3 - C4 - C9), Fibrinogen, C-Reactive Protein, Ceruloplasmin, Haptoglobin, α 1-Glycoprotein-acid, Ferritin, Angiotensinogen, Hemopexin, α 2 Macroglobulin, Alpha 1 antitrypsin, Protein-linked lipopolysaccharide (LBP), Albumin, Transferrin, Insulin Growth Factor 1 (IGF one).

In recent years, evidence has accumulated that supports the hypothesis that Diabetes Mellitus is a pathology that interferes with regulatory proteins of the Complement System and, therefore, precipitates its activation, which in turn has direct implications on the pathogenesis of the disease, especially at the vascular level.

Some of the mechanisms that are affected in this response are: adhesion, phagocytosis, bacterial activity, chemotaxis and cytokine release and the main cells that have been studied in this regard are neutrophils, monocytes and lymphocytes.

Among the main complications of Diabetes Mellitus, periodontal disease occupies the sixth position. The association between both diseases has been explored in many studies during the last decades. It has been shown that the risk of periodontitis increases approximately three times in diabetic individuals and that it greatly affects the glycemic control of patients, as demonstrated by Grossi et al. (6) who observed that after adequate periodontal treatment in diabetic patients they were reduced the complications of diabetes and the amount of insulin required, and concluded that the decrease in glycosylated hemoglobin is related to the reduction of gingival inflammation.

It can also be understood that periodontal disease triggers the response of acute phase proteins that have as a consequence systemic insulin resistance, alteration of phagocyte activity and lysosomal protein release, while tumor necrosis factor (TNF- α) It promotes the suppression of lipid synthesis in adipose tissues, alters the function of phagocytes and is an endogenous pyrogenic agent, as is IL-1 and IL-6.

For which this study opens a potential use to prevent the development of diabetic complications by regulation in the host of CD 4 CD25, T reg Th17, which can have a significant impact on the treatment of type 2 diabetic patients and their complications in the clinic. Finally, it should be noted that the Th17 population has emerged as a crucial pivot in infections, inflammations, autoimmune diseases or cancer.

CONCLUSIONS

Based on the study we can conclude the following: no statistically significant changes were observed in the glucose levels of the patients in the control group, although it is worth mentioning that periodontal treatment helped regulate and maintain glucose levels; on the other hand, in diabetic patients, changes in glucose levels showed a statistically significant decrease; it should be noted that this decrease was more evident in the results found between the first and second control appointments.

REFERENCES

1. Mealey, B. & Ocampo, G. Diabetes mellitus and periodontal disease. *Periodontol* 2000, 2007;44: 127-153.
2. Preshaw, A., Alba, A., Herrera, D., Jepsen, S., Konstantinidis, A. et al. Periodontitis and diabetes: a two –way relationship. *Diabetologia* 2012; 55: 21-31.
3. Reinauer, H., Home, P., Kanagasabapathy, A., Heuck, C. Diagnóstico y monitorización de la diabetes mellitus desde el laboratorio. WHO, 1-68

4. Zambon, J., Reynolds, H., Fisher, J., Shlossman, M., Dunford, R., Genc, R. Microbiological and immunological studies of adult periodontitis in patients with noninsulin-dependent diabetes mellitus. *J Periodontol* 1988; 59(1): 23-31.
5. Saran, R., Li, Y., Robinson, B., Ayanian, J., Balkrishnan, R. et al. US Renal Data System 2014 Annual Data Report: Epidemiology of Kidney Disease in the United States. *Am J Kidney Dis* 2015; 66(1): 7-305
6. Grossi, S., Skrepcinski, F., DeCaro, T., Robertson, D., Ho, A. et al. Treatment of periodontal disease in diabetics reduces glycated hemoglobin, *J Periodontol* 1997; 68(8): 713-719.
7. Carrillo Larco, R. & Bernabé Ortiz, A. Diabetes Mellitus tipo II en Perú: una revisión sistemática sobre la prevalencia e incidencia en población general. *Rev Perú Med* 2019; 36(1): 26-36.
8. Lalla, E., Lamster, I., Schmidt, A. Enhanced Interaction of Advanced Glycation End Products with their cellular receptor RAGE: Implications for the Pathogenesis of accelerated periodontal disease in Diabetes. *Ann Periodontol* 1998; 3(1):13-19.
9. Taylor, G. & Borgnakke, W. Periodontal disease: association with diabetes, glycemic control and complications. *J Oral Dis* 2008; 14(19): 1-203.
10. Batchelor, P. Is periodontal disease a public health problem? *Br Dent J* 2014; 217(8): 405-409.
11. Benza Bedoya, R. & Pareja Vásquez, M. Diagnóstico y tratamiento de la periodontitis agresiva. *Odontoestomatología* 2017;19(30): 29-39.
12. Ray, C. Periodontal disease. *N Engl J Med* 1990; 322(6): 373-382.
13. Matic, S., Radunovic, M., Barac, M., Kuzmanovic, J., Pavlica, D. et al. Subgingival areas as potential reservoirs of different *Candida* spp in type 2 diabetes patients and healthy subjects. *PloS One* 2019; 14(1): 1-14.
14. Löe, H. Periodontal Disease, the sixth complication of diabetes mellitus. *Diabetes Care* 1993; 16(1): 329-334.
15. Rajkumar, D., Gokulanathan, S., Shanmugasundaram, N., Lakshmigandhan, N., Kavin, T. Diabetes and Periodontal Diseases. *J Pharm Bioallied Sci* 2012; 4(2): 280-284.

16. Emrich, L., Shlossman, M., Genco, R. Periodontal disease in non-insulin-dependent Diabetes Mellitus, *J Periodontol* 1991; 62(2): 123-131.
17. Genco, R. & Borgnakke, W. Risk factors for periodontal disease. *Periodontol 2000* 2000; 62(2013): 59-94.
18. Tonetti, M., D'Aiuto, F., Nibali, L., Donald, A., Storry C. et al. Treatment of Periodontitis and endothelial function. *N Engl J Med* 2007; 356(9): 911-920.
19. Rajan, P., Nera, M., Kumar, A., Medandrao, N., Kumar, S. Comparison of glycosylated hemoglobin (HbA1C) levels in patients with chronic periodontitis and healthy controls. *Dent Res J* 2013;10(3): 389-393.
20. Bukleta, D. & Krasniqi, S. Impact of combined non-surgical and surgical periodontal treatment in patients with type 2 diabetes mellitus-a preliminary report randomized clinical study. *Biomed. Res* 2018; 29(3): 633-639.
21. Aubrey, W. Epidemiological and clinical aspects of periodontal diseases in diabetics. *Ann Periodontol* 1998; 3(1): 3-12.
22. Miller, L., Manwell, M., Newbold, D., Reding, M., Rasheed, A. et al. The relationship between reduction in periodontal inflammation and diabetes control: a report of 9 cases. *J Periodontol* 1992; 63(10): 843-848.
23. Salvi, G., Beck, J., Offenbacher, S. PGE₂, IL-1 β and TNF- α Response in Diabetics as modifiers of Periodontal Disease Expression. *Ann Periodontol* 1998; 3(1): 40-50.