A RETROSPECTIVE STUDY TO EVALUATE THE EFFECT OF TYPE 2 DIABETES MELLITUS ON THE SEVERITY OF PERIODONTAL DISEASE. A PRELIMINARY INVESTIGATION

D. Swaminathan, N. Ismail, S. S. Mohd Ali. A Retrospective Study to Evaluate the Effect of Type 2 Diabetes Mellitus on the Severity of Periodontal Disease. A Preliminary Investigation. Annal Dent Univ Malaya 2013; 20(1): 8-12

Original Article

D. Swaminathan[1], N. Ismail[1], S. S. Mohd Ali[1]

[1]Department of Oral Medicine and Periodontology,
Faculty of Dentistry,
University of Malaya
50603, Kuala Lumpur

Corresponding Author:
Prof Dr. Dasan Swaminathan
Email: dasans@um.edu.my

ABSTRACT

Aim: A preliminary investigation to assess the relationship in the severity of periodontal disease in diabetics when compared with non-diabetic subjects. **Materials and Methods:** A retrospective, comparative study using periodontal case notes of 40 subjects (20 Type 2 diabetics, 20 non-diabetics) who were selected based on the inclusion and exclusion criteria. Severity of periodontal disease was assessed through number of periodontal pocket ≥5mm. The results were compared between subjects whose age, gender and plaque scores are matched with the test group. Data obtained was then analyzed by SPSS Version 12. **Results:** When comparisons were made between test (Type 2 diabetic) and control (non-diabetic) groups, there were no significant difference ($p>0.05$) in the severity of periodontal disease. However, there was a clinically mean difference between the two groups. **Conclusions:** This preliminary investigation indicated that the severity of chronic periodontitis, as indicated in periodontal pocketing, increased in diabetic patients when compared to non-diabetics clinically, although it was not statistically significant. The finding of this investigation was thus not conclusive as it was only a retrospective study using patients' case notes. However, the results are now being further investigated with a proper clinical trial which examines periodontal parameters and diabetic status (HbA1c) of the subjects to determine the association between periodontal disease and diabetes mellitus.

Keywords
Periodontal disease, diabetes mellitus, periodontal pocketing, glycemic control.

INTRODUCTION

Diabetes mellitus is one of the most common endocrine disorders which are characterized by hyperglycaemia that leads to complications such as retinopathy, nephropathy, macrovascular and microvascular disease, delayed wound healing (1) and periodontal disease (2). Diabetes mellitus can be classified into several subtypes, based upon pathophysiology of each of the disease. There are two main classes of diabetes-; insulin-dependent Type 1 diabetes and non-dependent Type 2 diabetes. Type 1 diabetes is characterized by deficiency of insulin due to destruction of the pancreatic β cells in the islet of Langerhans and usually progresses to the stage of absolute insulin deficiency (3). Type 2 diabetes is characterized by increase blood glucose in the context of insulin resistance and decreased insulin secretion (4). Gestational diabetes can be defined as glucose intolerance that affects pregnant women who have no history of diabetes (5).

Periodontal disease is a chronic infectious disease that causes chronic inflammation of supporting tissues of the tooth that leads to connective tissue attachment and alveolar bone loss. Periodontal disease involves host inflammatory response in the periodontium to bacterial antigens found in bacterial plaque (6). The early stage of periodontal disease is called gingivitis. Gingivitis is a reversible condition where it will not progress to attachment loss when bacterial plaque is removed (7). Signs of gingivitis are erythema, oedema and also bleeding on brushing and probing. Gingivitis may progress to periodontitis if left untreated (8).

Periodontitis is an inflammatory disease affecting the periodontium. It may lead to pocket formation between the tooth and gingiva that predicts breakdown of periodontal apparatus and alveolar bone. Clinical features of periodontitis include oedema, erythema with bleeding on gentle probing and it may be associated with halitosis and foul taste.

Diabetics have a greater prevalence and severity of periodontal disease compared with subjects without diabetes (9). According to Loe (1993), periodontal disease has been identified as the sixth complication of diabetes

mellitus. Many studies have showed that one of the risk factor of periodontal disease is diabetes mellitus and the level of glycemic control appears to be an important determinant in this relationship. One of the reasons suggested for the increased prevalence and severity of periodontal disease in diabetics is the impaired host defence systems towards microbial challenges (10). Some diabetes-induced metabolic alterations serve to reduce host resistance to periodontal breakdown that results in rapid progression of periodontitis. Hyperglycaemia causes the release of the electrons that react with oxygen, producing superoxide and increasing oxidative stress inside the cell (11). Expression of pro-inflammatory cytokines can be stimulated by the formation of the superoxide and other reactive oxygen species (ROS). This in turn, contributes to dysregulation of innate immunity which is characterized by an increased production of TNF-α and IL-1β by macrophages and the release of superoxide and nitric oxide (NO). Hyperglycaemia can lead to the activation of polyol pathway. The polyol pathway may enhance inflammation by increasing the formation of advanced glycated end product (AGEs), ROS and NO. AGEs inhibit collagen production in the connective tissue and enhanced matrix metalloproteinase activity that could lead to greater connective tissues breakdown and causes prolonged inflammation (12). AGEs interfere with osteoblast differentiation and reduce bone formation in mineralized tissues.

In diabetic patients, the function of cells which include the neutrophils, monocytes, and macrophages are altered (13). Increased response of the involved cells can lead to significantly increased production of proinflammatory cytokines and mediators such as tumour necrosis factors α (TNF-α) in response to periodontal pathogens (13). This can results in increasing the host tissue destruction.

Basically, patients with diabetes mellitus are immuno-compromised. Thus, they tend to be more susceptible to infections like periodontal disease (10). Wound healing is also compromised due to primary reparative cell in periodontium and the fibroblast is not functioning well in hyperglycemic state. Collagen that is produced by these fibroblasts is susceptible to rapid degradation by matrix metalloproteinase enzymes. The production of these enzymes is increased in diabetics (14). Thus, increase of alveolar bone loss and attachment loss can occur due to delayed periodontal wound healing which is altered in subjects with sustained hyperglycaemia (13).

Periodontal diseases may influence diabetes by inducing an elevated systemic chronic inflammatory state. The highly vascular, inflamed and ulcerated periodontal pocket epithelium provides a ready portal of entry for periodontal pathogen from dental biofilm which mainly consist of gram negative and obligate anaerobic bacteria. In addition, pro inflammatory mediators such as IL-1β, TNF-α, IL-6, PGE2 and thromboxane B2 that produced locally at periodontal lesion could also enter the systemic circulation (15). Hence inflamed periodontal tissue serves as a potential endocrine –like source for chronic systemic inflammatory challenge. TNF-α has been reported to interfere with lipid metabolism and cause insulin resistant. IL-6 and IL-1 have also been reported to antagonize insulin action.

Recent studies demonstrate that some diabetic complications, especially hyperglycemia may be improved with effective treatment of periodontitis. In a study done by Grossi (1997), successful periodontal treatment and reduction of periodontal inflammation can lead to reduction in the levels of glycated haemoglobin (16).

The aim of this preliminary investigation was to access the severity of periodontal disease in diabetics when compared with non-diabetics by looking at the data of patients' case notes who had sought periodontal treatment with undergraduates at the Faculty of Dentistry, University of Malaya.

MATERIALS AND METHODS

Subject Selection

Patients' case notes from undergraduates of Faculty of Dentistry, University of Malaya were retrieved. A total of 40 subjects' that were found to have fulfilled the inclusion and exclusion criteria were utilized in this study. As this was a retrospective study, no sampling method was applied and the subjects were categorized into test (20 diabetic subjects) and control groups (20 non-diabetic subjects).

Inclusion Criteria

A) Test Group

• Subjects with type 2 diabetes mellitus whose diagnosis had been established (WHO diagnostic criteria) and were on regular follow-ups for a minimum of 2 years. Most of them were under follow up in University of Malaya Medical Centre (UMMC).
• Subjects should have at least 12 teeth present.
• Subjects with their age ranged from 40-60 years.
• Subjects should have been diagnosed with periodontal diseases (moderate to advanced chronic periodontitis).
• All the subjects included should have their case notes duly checked and signed by the supervisors on duty during the session.

B) Control Group

• Medically healthy patients especially with no history of diabetes mellitus and age, gender and oral hygiene status matched with the test group.
• Subjects should have at least 12 teeth present.
• Subjects with their age ranging from 40-60 years.

• All the subjects should have had their case notes duly checked and signed by the clinical supervisors on duty during the session.

Exclusion criteria:

• Subjects who were pregnant.
• Subjects who had received periodontal treatment else where within the past 3 months and during the duration from the patient were examined.
• Subjects who were smok ers.
• Subjects who were immune compromised; for example, subjects on steroid therapy or radiation therapy and other systemic modulating factors other than diabetes mellitus such as cardiovascular disease, respiratory problems, kidney disease, haematological disorder and other chronic diseases.
• Subjects who were on medication that can cause gingival enlargement such as calcium channel blockers and antidepressants.
• Subjects with incomplete case notes or with case notes that were not duly checked and signed by supervisors on duty.

Study parameters

Plaque score and periodontal pocket ≥ 5mm were the periodontal parameters that were recorded from the case notes of the patients who came within the inclusion criteria. The methodology used by the undergraduates to obtain these periodontal parameters were done by measuring at 6 sites per tooth (mesiobuccal, buccal, distobuccal, distolingual, lingual, and mesiolingual) for plaque and periodontal pocketing. Charting of all teeth present was included except the third molars.

Plaque score using Visible Plaque Index (17) in the case notes of patients was tabulated as percentage. We matched the subjects according to their oral hygiene status using this plaque score percentages.

We then assessed the severity of periodontal disease by calculating the percentage of numbers of periodontal pocket ≥ 5mm per number of tooth surfaces that were found in the subjects using the probing pocket depth data available in the patients' periodontal charting. To achieve the objectives of this study, this periodontal parameter of the test and control subjects were compared.

Statistical Analysis

Data entry and analysis was done using Statistical Program for Social Science (SPSS) Version 12.0. To evaluate the severity and progression of periodontal disease in test and control group Mann-Whitney Test was employed.

RESULTS

By comparing line graph for control and test group in the case notes, it was apparent that test subjects have higher percentage of number of pocket depths ≥5mm per tooth surfaces that present in the oral cavity which indicates severity of periodontal disease.

As both test and control subjects displayed skewed distribution for percentage of severity of periodontal disease, this parameter was analyzed by using Mann-Whitney Test. The non-parametric test exploited the mean values for both groups of subjects and obtained a statistically insignificant p-value of 0.099. A difference of 6.1 was observed from the means values between test and control subjects after the data was evaluated using Mann-

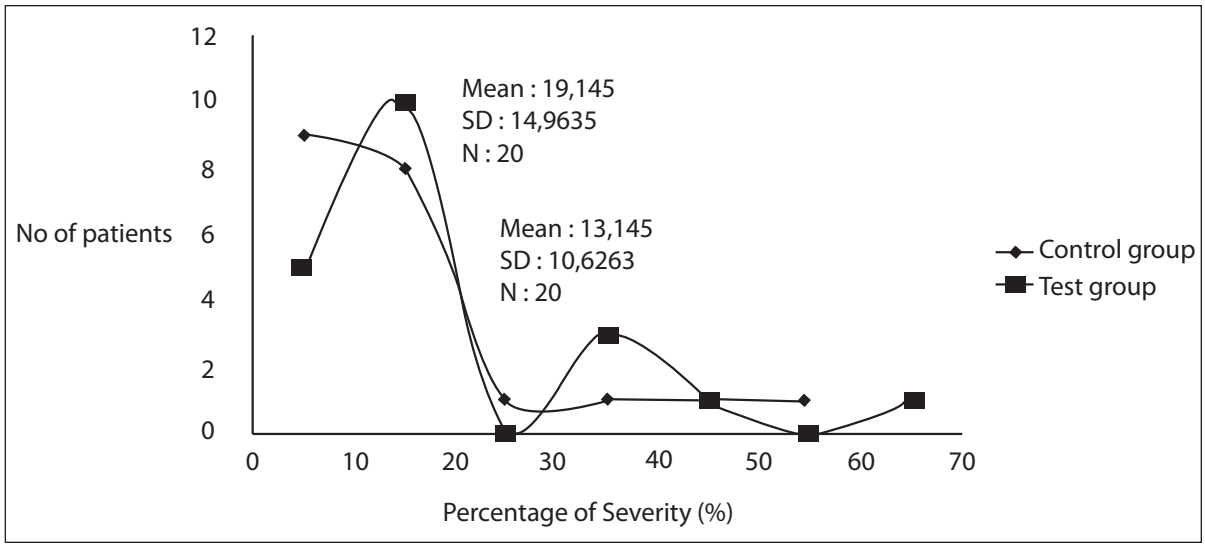

Figure 1. Percentage of Severity of Periodontal Disease (Periodontal pocket depth > 5mm) among Control and Test Subjects

Whitney Test. This showed that there was clinically mean difference between diabetics and non-diabetics. However, it did not suggest any statistical significance because p-value was more than 0.05.

DISCUSSION

Several studies have been carried out on the association between diabetes mellitus and periodontal disease and most of this research was carried out in western countries. Thus we have now embarked on a study to assess the association between these two diseases in the Malaysian population starting with this preliminary investigation.

From the case notes of patients attending the Faculty of Dentistry for periodontal treatment by undergraduates, statistically there was no significance difference in severity of periodontal disease between the type 2 diabetic and non-diabetic patients as the p-value is >0.05. However, the mean difference of 6.1 (severity of periodontal pocketing) between control and test groups showed that clinically, the severity of periodontal disease in diabetic patient is more compared to non-diabetic patients.

Based on the findings of this study, proper conclusions could not be achieved as the study was based only on the findings reported in the case notes of the patients and the actual diabetic status of the subjects could not be evaluated.

Limitations of the study

Background of the diabetic status was not recorded in detail in the case notes that were selected, such as the reading of blood glucose level on the day of examination and HbA1c. Moreover, the diabetic status may not have been diagnosed in some subjects and these subjects placed into the control group. However, we tried to include only case notes of subjects whose medical history was clearly stated.

Subjects could not be matched in terms of ethnicity and socio-economic status. Matching pairs would not have been sufficient for us to carry out this study if these criteria were applied.

Standardization of the data obtained could not be achieved in this retrospective study as it was performed by several dental students during their clinical sessions. However, all the data obtained had been approved by experienced supervisors.

CONCLUSION

This study was a preliminary investigation and presently a study is being conducted in the Faculty of Dentistry, University of Malaya where we are working with our medical colleagues to evaluate the association between periodontal disease and diabetes mellitus in Malaysian patients before and after non-surgical periodontal treatment.

The periodontal parameters and HbA1c of the patients are measured before and after periodontal treatment and we hope to reach more conclusive results from this study.

ACKNOWLEDGEMENT

This work was fully funded by a University of Malaya Research Grant.

We would also like to thank Dr Marhazlinda Jamaluddin for her assistance in statistical analysis.

REFERENCES

1. Kinane DF, Chestnutt IG. Relationship of diabetes to periodontitis. Curr Opin Periodontology 1997; 4:29-34.

2. Loe H. The sixth complication of diabetes mellitus. Diabetes Care 1993; 16(1): 329-334.

3. Atkinson MA, Maclaren NK. The pathogenesis of insulin-dependent diabetes. N Engl J Med 1994; 331: 1428-1436.

4. Janice F. Diabetes mellitus and oral care. Dental Update 2004; 31 (4): 190-198.

5. Kidambi S & Patel SB. Diabetes mellitus: Considerations for dentistry. JADA 2008; 139: 8S-11S.

6. Lingen MW, Kumar V, Abbas AK, Fausto N. Robbins & Cotran Head and Neck, Pathologic Basis of Disease. 7th ed. Philadephia, Pa:Elsevier 2004:pp 773.

7. Marsh PD. Dental plaque: Biological significance of a biofilm and community life style. J Clin Periodontal 2005; 32(6): 7-15.

8. Ireland R. Clinical textbook of dental hygiene and therapy. Blackwell Munksgaard 2006.

9. Hugoson A, Thorstensson H, Falk H, Kuylenstierna J. Periodontal conditions in insulin-dependent diabetics. J Clin Periodontol 1989; 16: 215-223.

10. Bartold PM, Marshall RI, Georgiou T, Mercado FB. What is the significance between systemic diseases and periodontitis? Asia Pacific Society of Periodontology (APSP), 2002.

11. Brownlee M. The pathobiology of diabetic compilations: a unifying mechanism. Diabetes 2005; 54: 1615-1625.

12. Goova M.T. Li J. Kislinger, T. Blockade of receptor for advanced glycation end-products restores effectives would healing in diabetics mice. A J Path 2001; 159 (2):513-525.

13. Mealey BL. Periodontal disease and diabetes. A two-way street. JADA 2006; 137 (supp1.2): 26s-31s.

14. Golub LM, Ryan ME, Williams RC. Modulation of the host response in the treatment of periodontitis. Dent Today 1998; 17(10): 102-6, 108-9.

15. Loos BG. Systemic marker of inflammation in periodontitis. J periodontal 2005; 76: 2106-2115.

16. Grossi SG, Skrepcinki FB, Decaro T, et al. Treatment of periodontal disease in diabetics reduces glycated heamoglobilin. J Periodontol 1997; 68: 713-719.

17. Ainamo D. J, Bay I. Problems and proposals for recording gingivitis and plaque. Int J Dent 1975; 25(4): 229-235