Journal of Research in Medical and Dental Science
2022, Volume 10, Issue 9, Page No: 194-197
Copyright CC BY-NC 4.0
Available Online at: www.jrmds.in
eISSN No.2347-2367: pISSN No.2347-2545

Periodontitis and Diabetes Mellitus–A Two Way Relationship

Nandini Sharma[1*], Khushboo Jeevan Durge[1], Pavan Bajaj[1], Bhairavi Kale[1], Anjali Borle[2]

[1]Department of Periodontics, Sharad Pawar Dental College and Hospital, Datta Meghe Medical Sciences, Sawangi (Meghe), Wardha, Maharashtra, India

[2]Department of Prosthodontics, Crown and Bridge Sharad Pawar Dental College and Hospital, Datta Meghe Medical Sciences, Sawangi (Meghe), Wardha, Maharashtra, India

ABSTRACT

We have long recognised that the need for diagnosis of diabetes in our patients, which is linked to a variety of oral disorders, like most commonly occurring periodontitis and various others like xerostomia, candidal infections.

There exists a two way relationship of diabetes and periodontitis diabetes a complex disorder which affects the metabolisms of carbohydrate, lipid and protein in which hyperglycaemia is the main clinical sign.

Periodontitis is caused mainly due to plaque associated bacteria as well as defect in the host defence mechanism. These two way relationship can be taken under control by controlling the normal levels of blood glucose or by maintaining the proper oral hygiene to prevent the accumulation of plaque and calculus as they are the main retentive factors for the development of periodontitis.

Weather the diabetes mellitus is present or not, as well as the controlled degree of the disease in the patients, affects the occurrence and severity of periodontitis. Furthermore, various oral consequences of undiagnosed or poorly treated diabetes mellitus have been documented and must be considered during diabetic patients' periodontal disease therapy.

"Periodontal diseases" can have a huge effect on a diabetic's metabolic status. The increase the risk of "glycemic deterioration" over time is because of the presence of periodontitis.

Key words: Periodontal disease therapy, Metabolic status, Periodontitis, Glycemic deterioration

HOW TO CITE THIS ARTICLE: Nandini Sharma, Khushboo Jeevan Durge, Pavan Bajaj, Bhairavi Kale, Anjali Borle, Periodontitis and Diabetes Mellitus-A Two Way Relationship, J Res Med Dent Sci, 2022, 10 (9): 194-197.

Corresponding author: Dr. Nandini Sharma
E-mail: nandinisharma035@gmail.com
Received: 05-Jul-2022, Manuscript No. JRMDS-22-47380;
Editor assigned: 07-Jul-2022, PreQC No. JRMDS-22-47380 (PQ);
Reviewed: 21-Jul-2022, QC No. JRMDS-22-47380;
Revised: 06-Sep-2022, Manuscript No. JRMDS-22-47380 (R);
Published: 14-Sep-2022

INTRODUCTION

"Periodontitis is a chronic inflammatory disease triggered by microbial plaque", Clarke and Hirsch, et al. [1] proposed a multi factorial model for the development of periodontitis which involves the effect of environmental, systemic and personal areas, with bacteria to explain individuals variable risk of periodontitis. It's a slow growing disease, but the tissue damage it causes is largely permanent. Periodontitis causes the "periodontal ligament's collagen fibres" to deteriorate, which eventually "periodontal pocket" is formed in between gingiva and the tooth. The deepen pocket causes attachment loss occurs as they destroy periodontal ligament fibres more and reabsorb the alveolar bone. More of research have given attention on "type 2 diabetes" as a periodontitis risk factor, which has history to develop in the patients with the age of their "40's and 50's".

Periodontitis can be further classified as aggressive and chronic periodontitis. Aggressive periodontitis is marked by a rapid pace of disease progression; as a result, by the time the patient is recognised, he or she has lost periodontal attachment to many permanent teeth. At the initial stage, radiographic views of the periodontal lesions at the proximal surfaces of posterior teeth undergo vertical bone loss, and the bone loss is typically bilateral. Advanced cases shows horizontal bone loss in aggressive periodontitis [2,3].

Aggressive periodontitis can be generalised and localised. Tissue loss normally begins at the permanent first molars and incisors in localised severe periodontitis, and as the patient becomes older, the condition may also spread to the surrounding teeth.

Later diagnostic methods which allow for the recognition of main causative factors it allow for the early visibility in patient with "aggressive periodontitis", hence improve the

prevention and control of the disease and by increasing the existing diagnostic methods.

Diabetic patients are more susceptible to periodontitis due to altered microbial flora, inflammatory, and immune processes, on the one hand, and periodontitis has been proposed as a risk factor for diabetes mellitus on the other. The susceptibility to aggressive periodontitis is mediated by the host's reaction to microbial infection [4-7].

LITERATURE REVIEW

Microbiologic etiologic factors

The microbial causative agents have taken much time to be discovered. Recent advances in the microbiological detection of sub-gingival biofilm causing organisms in health and periodontal disease. Recent findings from studies using more sensitive molecular microbiologic procedures may not confirm previous findings that "Aggregatibacter action mycetemcomitans" is the main causative organisms for the periodontitis

The "A. actinomycetemycomitans-JP2" clone has been linked to the development of aggressive periodontitis in some people.

Even though both diseases have typically progressed in people in their 40's and 50's, the main focus is on type 2 diabetes as a periodontitis risk factor.

Type 1 diabetes, on the other hand has caused increased problem of "periodontitis" and all of the "diabetic people" which include kids and teenagers should be assessed for periodontal risk. Despite similar plaque levels, roughly 10% of children (aged 18) with type 1 diabetes mellitus had greater attachment loss and bone loss compared to controls, according to one early study [8].

The 'two-way' relationship between diabetes and periodontitis has recently received much interest [9-14].

Furthermore, multiple studies have conducted that the unrelated diabetic disorders which are problem in the retina of the eye knows as "Retinopathy", "Diabetic Neuropathy" which is nervous system related problem, Proteinuria which is increased levels of protein and various cardiovascular condition, these are associated with the increased severity of periodontitis [15-18].

For example, diabetic participants with "aggressive periodontitis" at border had a "6 times greater risk of decreasing glycaemic control" in time as compared to the other diabetic subjects which do not have "periodontitis" in a two-year longitudinal research. Periodontal disease-related immunologic pathways have been shown in studies to influence systemic diseases like diabetes and cardiovascular disease, in addition to breaking down local tissues.

When compared to diabetic individuals without periodontitis, "diabetic subjects which have severe periodontitis had a six-time greater risk of poor glycemic control over time in a two-year longitudinal experiment".

Periodontal disorders can cause or exacerbate a "systemic chronic inflammatory response". Acute bacterial and viral infections were shown to increase insulin resistance in persons without diabetes, a condition which can last for weeks or months after the sickness is gone. Glycemic management is greatly aggravated by such conditions and the resulting increases in insulin resistance in patients with diabetes. "A chronic gram-negative bacterium which causes the periodontal infections as a result of increased resistance to insulin and has decreased glycemic control". A proper treatment plan which decreases the "periodontal inflammation" by improving the metabolic control we may restore insulin sensitivity. Such a hypothesis is supported by the earlier mentioned intervention studies that showed improved glycaemic control after periodontal care. A diabetic individual with periodontitis has even more badly "systemic inflammatory condition", with elevated serum levels of "Interleukin-6, Tumour Necrotic Factor (TNF), and C-Reactive Protein" (CRP), which might affect "insulin resistance" and "glycaemic management". This could justify why periodontitis raises the likelihood of poor glycaemic control in patients with type 2 diabetes. It could also justify why periodontal therapy improves glycaemic level control in diabetic people in some trials.

"Williams and Mahan", demonstrated that periodontal therapy has been shown to enhance metabolic management, as seen by decreased insulin needs and blood glucose levels, for several decades. Several studies addressed the effect of periodontal treatment on glycemic control of diabetic patients. These trials found that periodontal treatment improved diabetes patients' periodontal health, but that improvements in metabolic control could only be achieved by combining mechanical periodontal treatment and systemic antibiotics.

"Williams and Mahan" and "Miller, et al." found that periodontal therapy and systemic antibiotics reduced glycated haemoglobin in type 1 diabetic patients. Grossi, et al. found similar results with systemic doxycycline administration in type 2 diabetic patients.

HbA1c, or glycated haemoglobin, is formed when haemoglobin is glycosylated. Its value represents a person's glycemic status over the last two to three months [3].

Trivelli, et al. observed a two-times raised in HbA1c in diabetic patients compared with the non-subjects in a bigger investigation. At the time of mid-1970's, it was obvious that "HbA1"and "HbA1c" levels are higher in people with diabetes, although even the cause of this remained unclear.

DISCUSSION

Pathogenic mechanisms that link diabetes mellitus and periodontitis

The disease "Periodontal disease" is a "complex and extremely chronic inflammatory condition" recognises the presence of a sub gingival biofilm for an extended

period of time induces inflammation in the periodontal tissues (dental plaque).

Dysregulated release of host derived inflammatory mediators and tissue degradation characterise the inflammatory response.

"TNF, nuclear factor activated by receptor B ligand (RANKL)", "matrix metallo-proteinases primarily which involve MMP-8, MMP-9, and MMP-13", as well as "T cell regulatory cytokines such as interleukin-12, interleukin-18" and chemokines, are among the most commonly studied [10].

The double relationship of the inflammatory mediator of cytokine has network in "periodontal pathophysiology" is now becoming clearer, and it is well known that the inflammatory response differs significantly between people. This variability is controlled by genetic, epigenetic, and environmental variables and exists not just across individuals, but also within individuals over time. Diabetes and periodontitis are both caused by inflammation.

High levels of inflammatory mediators in the blood are linked to diabetes mellitus, both type 1 and type 2 [11]. Diabetes' exaggerated inflammatory state causes both micro vascular and macro vascular problems, and it is well known that hyperglycaemia can activate the easy path which increase inflammation, oxidative stress, and apoptosis [12].

In diabetes, "serum levels of IL-6 and TNF" are elevated, and "IL-6 and C-reactive protein levels" have been shown to estimate the onset of type 2 diabetes mellitus in the future. As a result, the systemic inflammation linked to periodontal disease may exacerbate the diabetic condition.

Diabetes which increases inflammation in the periodontal tissues. For example, patients with diabetes type 1 gingivitis or periodontitis have "higher levels of prostaglain-2 and interleukin-1" in gingival cerevicular fluid (a watery exudate that flows from the gingival margin) than non-diabetic patients with the same severity of periodontal disease.

In diabetic patients, researches have frequently shown abnormalities in polymorph nuclear leucocyte (PMN) activity, including impaired chemo taxis, phagocytosis, and microbicidal activities [19-20].

When compared to diabetic patients with less severe periodontitis, diabetic patients with severe periodontitis have depressed polymorph neutrophils chemo taxis and defective polymorph neutrophil apoptosis this lead to increased accumulation of polymorph neutrophil in periodontal tissue, resulting in more tissue demolition due to more sever and continued MMP and "Reactive Oxygen Species" release (ROS) [13,14].

CONCLUSION

"Rapid bone loss" and "clinical attachment loss" appears in the sever form of aggressive periodontitis.

Diabetes mellitus is a common medical condition that every dentist will come across in their dental practice. The doctor must be conscious of the clinical and oral signs and indications of poorly controlled diabetes mellitus or which remain undetected, and patients who exhibit various different signs or symptoms should be referred to a medical physician. Patients with diabetes mellitus who are suspected or which are undiagnosed with the undetected diabetes should only receive emergency care until their health is properly assessed. Proper antibiotic therapy should be advised to the patient in association with any necessary surgical procedure

The occurrence of oral infection with the uncontrolled diabetes has the degree of known diabetic with the unknown patient prognosis. The doctor must be well oriented to manage diabetic emergencies as and when they should occur in the dental clinic, most commonly occurring incidence in the clinic is hypoglycaemia.

Although the recent advances in the advance therapies which are available to manage of diabetes mellitus told that this condition must be under controlled in the future, proper periodontal patient management requires medical consultation. Advanced periodontal disease can make it difficult to control diabetes in later stages, so the general practitioner should be informed of the patient's condition. Most cases, the patient with has the sever un-controlled diabetes mellitus must receive harmless and active periodontal therapy with some dental hospital etiquette modifications.

REFERENCES

1. Rajan P, Nera M, Pavalura AK, et al. Comparison of glycosylated haemoglobin (HbA1C) levels in patients with chronic periodontitis and healthy controls. Dent Res J 2013; 10:389-393.
2. Clarke NG, Hirsch RS. Personal risk factors for generalized periodontitis. J Clin Periodontol 1995; 22:136-145.
3. Tervonen T, Karjalainen K. Periodontal disease related to diabetic status: a pilot study of the response to periodontal therapy in type 1 diabetes. J Clin Periodontol 1997; 24:505-510.
4. Nibali L, D'Aiuto F, Griffiths G, et al. Severe periodontitis is associated with systemic inflammation and a dysmetabolic status: A case-control study. J Clin Periodontol 2007; 34:931–937.
5. Loos BG. Systemic markers of inflammation in periodontitis. J Periodontol 2005; 76:2106-2115.
6. Rohlfing CL, Little RR, Wiedmeyer HM, et al. Use of GHb (HbA1c) in screening for undiagnosed diabetes in the U.S population. Diabetes Care 2000; 23:187–191.
7. Wankhede A, Dhadse P, Jaiswal P, et al. Comparative evaluation of interleukin-17 in gingival crevicular fluid of patients with aggressive periodontitis and healthy gingival sites. J Datta Meghe Inst Med Sci Uni 2021; 16:90–93.

8. Cianciola LJ, Park PH, Bruck E, et al. Prevalence of periodontal disease in insulin-dependent mellitus (juvenile diabetes). J Am Dent Assoc 1982; 104:653–660.
9. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Ann Periodontol 2001; 6:99–112.
10. Preshaw PM, Taylor JJ. How has research into cytokine interactions and their role in driving immune responses impacted our understanding of periodontitis? J Clin Periodontol 2011; 38:60–84.
11. Dandona P, Aljada A, Bandyopadhyay A. Inflammation: the link between insulin resistance, obesity and diabetes. Trends Immunol 2004; 25:4–7.
12. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. Diabetes 2005; 54:1615–1625.
13. Manouchehr-Pour M, Spagnuolo PJ, Rodman HM, et al. Impaired neutrophil chemotaxis in diabetic patients with severe periodontitis. J Dent Res 1981; 60:729–730.
14. Graves DT, Liu R, Alikhani M, et al. Diabetes-enhanced inflammation and apoptosis—impact on periodontal pathology. J Dent Res 2006; 85:15–21.
15. Karjalainen KM, Knuuttila ML, von Dickhoff KJ. Association of the severity of periodontal disease with organ complications in type 1 diabetic patients. J Periodontol 1994; 65:1067–1072.
16. Moore PA, Weyant RJ, Mongelluzzo MB, et al. Type 1 diabetes mellitus and oral health: assessment of tooth loss and edentulism. J Public Health Dent 1998; 58:135–142.
17. Moore PA, Weyant RJ, Mongelluzzo MB et al. Type 1 diabetes mellitus and oral health: assessment of periodontal disease. J Periodontol 1999; 70:409–417.
18. Thorstensson H, Kuylenstierna J, Hugoson A. Medical status and complications in relation to periodontal disease experience in insulin-dependent diabetics. J Clin Periodontol 1996; 23:194–202.
19. Ambad RS, Gaikwad SB, Anshula G, et al. Polyherbal antidiabetic drug: An approach to cure diabetes. Int J Res Pharm 2020; 11:2679–2683.
20. Ambad R, Jha RK, Chandi DH, et al. Association of leptin in diabetes mellitus and obesity. Res J Pharm Technol 2020; 13:6295–6299.