Austin Journal of Dentistry

Open Access

Austin Publishing Group

Short Communication

Current Evidence Relating Oral Health and Diabetes Mellitus

Pai PP[1], Ramachandra Pai RP[2]* and Kangath RV[3]

[1]Dr Basil's Dental Clinic, Cochin, Kerala, India

[2]Endocrinology and Metabolism, Department of Internal Medicine, San Francisco VA, CA, USA

[3]Infectious Diseases, Department of Internal Medicine, San Francisco VA, CA, USA

***Corresponding author:** Rajasree Pai Ramachandra Pai, Endocrinology and Metabolism, Department of Internal Medicine, San Francisco VA, CA, USA

Received: October 28, 2021; **Accepted:** November 18, 2021; **Published:** November 25, 2021

Abstract

Diabetes mellitus is a systemic disease characterized by increased blood glucose levels and abnormalities of lipid metabolism due to deficiency of insulin or insulin resistance. Being a systemic disease, diabetes affects most body parts, which in case of oral health can lead to periodontitis.

Keywords: Diabetes Mellitus; Periodontitis

Introduction

Diabetes Mellitus (DM) is chacterised by altered glucose and lipid metabolism. Diabetes can either be type 1 which occurs mostly in children and young adults from insulin deficiency or type II diabetes which occurs due to insulin resistance among adults. Among the two, type II is more common form and is related to sedentary lifestyle and consumption of simple carbohydrates and processed foods [1]. Global prevalence of type II diabetes is on the rise and in some countries upto 30% population is affected by diabetes mellitus.

The common symptoms of diabetes such as polyuria, polydypsia and polyphagia are a direct result of hyperglycemia. Diagnosis is made by blood test such as fasting glucose or glycated A1c and is treated by oral hypoglcyemics, incretin therapy or insulin.

Periodontal disease, is an inflammatory condition that affects tissues surrounding the teeth. In its early stage, called gingivitis, the gums become swollen, red, and may bleed. In its more advanced stage, called periodontitis, the gums can pull away from the tooth, bone can be lost, and the teeth may loosen or fall out. Bad breath or halitosis may also occur. Periodontal disease is generally due to oral bacteria infecting the tissue around the teeth [2].

Discussion

Periodontitis is the sixth common complication of diabetes in order of occurrence [3]. Prevalence of severe periodontitis in diabetics is 59.36% as compared to non-diabetics which is 6.39%. Most of studies show a higher prevalence and severity of periodontal disease in diabetics than in non-diabetics with similar local irritation including greater loss of attachment, greater alveolar bone loss, increased bleeding on probing, and increased tooth mobility resulting in tooth loss [4].

Ray and Orban observed that basic structural changes in the diabetic periodontium are degeneration of tissues and the presence of calcified bodies in and around small blood vessels of the gingiva [5].

Severe gingival inflammation, deep periodontal pockets, rapid bone loss, and frequent periodontal abscess often occur in diabetic patients with poor oral hygiene. Various studies have shown increased prevalence and severity of periodontal disease in Type I diabetics. The increased susceptibility to infection and reduced healing capacity with altered collagen metabolism leads to increased periodontal destruction [5].

Insulin-dependent diabetic patients with periodontitis have shown subgingival flora such as, Prevotella intermedia, and actinomycetemcomitans. Increased glucose level in crevicular fluid may favor the growth of some microbial species.

Oliver et al. showed that large number of Poly morpho nuclear cells were present in inflamed gingival crevices of poorly controlled diabetics [6]. Defects in polymorphoneutrophil function along with underlying vascular insufficiency is another contributing factor of increased infections in diabetics [7].

Shapira et al. observed a prostaglandin E2 hypersecretory response to lipopolysaccharides in monocytes from patients with early onset form of the disease [8].

Golub et al. found in experimentally induced diabetes, impaired production of bone matrix component by osteoblasts, decreased collagen synthesis by gingiva and periodontal ligament fibroblasts, and increased collagenase activity. Administration of insulin has been shown to prevent the onset and corrects the defective collagen production [9].

Vascular changes can occur from long standing hyperglycemia. This can lead to increased basement membrane thickness in the gingival capillaries causing increased bleeding.

Poor wound healing, likely from non-enzymatic glycosylation of collagen and other proteins due to hyperglycemia, altered monocyte function and altered growth factor secretion may be a key mechanism for impaired wound healing in diabetics.

Prevention and Management

Prevention of diabetes mellitus and good glycemic control are essential to prevent periodontitis and related complications. Chlorhexidine rinse has been shown in some studies to reduce glycated hemoglobin levels in addition to reducing procedure related complications for periodontal therapy [10]. Diabetics need annual

Citation: Pai PP, Ramachandra Pai RP and Kangath RV. Current Evidence Relating Oral Health and Diabetes Mellitus. Austin J Dent. 2021; 8(3): 1162.

oral examinations and have to maintain proper oral hygiene to prevent complications related to periodontitis.

Conclusion

Diabetes is a systemic disease which affects the whole body and particular attention needs to be given to oral health due to increased risk of gingivitis and periodontitis. Diabetes mellitus causes increased risk and complications to periodontium, the calcified tissue, and the oral mucosa. In summary, poor glycemic control increases the risk of periodontal disease.

References

1. Loe H. Periodontal disease: Sixth complication of diabetes Mellitus. Diabetes Care. 1993; 16: 324-334.
2. Pucher J, Stewart J. Periodontal disease and diabetes mellitus. Curr Diab Rep. 2004; 4: 46-50.
3. Albander JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in United States. J Periodontol. 1999; 70: 13-29.
4. Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in US adult population. Community Dent Oral Epidemiol. 2002; 30: 182-192.
5. Ray HG, Orban B. The Gingival structure in diabetes mellitus. J Periodontol. 1950; 28: 85-95.
6. Oliver RC. Enzyme activity in crevicular fluid in relation to metabolic control of diabetes and other periodontal risk factors. J Periodontol. 1993; 64: 358-362.
7. Hawley HP. The effect of long chain free fatty acids on human neutrophils, function and structure. Lab Invest. 1976; 34: 216-222.
8. Shapira L. Involvement of protein tyrosinkinase in lipopolysaccharide induced. TNFA and ILIB production by human monocytes. J Immunol. 1994; 153: 1818-1824.
9. Golub LM, Nicoll GA, Iacono VJ, Ramamurthy NS. *In vivo* crevicular leucocytes response to be a chemotactic. Infect Immun. 1982; 37: 1013-1020.
10. Miller LS. Relationship between reduction in periodontal inflammation and diabetes control. J Periodontol. 1992; 63: 843-848.