Research Paper

Volume : 2 | Issue : 10 | October 2013 • ISSN No 2277 - 8179

A Study of Corelation of Glycemic Control and Periodontal Health

Medical Science

KEYWORDS : Gingivitis, Periodontitis, Glycated hemoglobin, Diabetes

Dr. Ankita Shah — Former graduate student, Govt. Dental college & Hospital, Ahmedabad

Dr. Paras Parekh — Post graduate student, Department of Physiology, B.J.Medical college, Ahmedabad

ABSTRACT

Introduction : Healthy gingiva is pink and firm which is tightly attached to teeth and do not easily bleed when you brush your teeth. Gingival disease may be either in mild form called Gingivitis or it may be in severe inflammatory form which destroys bone that hold teeth called Periodontitis. Glycated hemoglobin (HbA1c) is glycemic control over past two to three months. Methdology : A total of 158 subjects who have come to clinic with various dental complain were included in study. Gingival & periodontal examinations were done. Glycemic control of all these subjects was measured by doing Glycated hemoglobin assay (HbA1c). Result : Significant association (P<0.0001) between poor glycemic control and occurrence of gingivitis & periodontitis was found. Conclusion : Poor glycemic control can affect gingival health and cause gingivitis and Periodontitis.

Introduction :

India has more diabetics than any other country in the world, according to the International Diabetes Foundation. The disease affects more than 50 million Indians - 7.1% of the nation›s adults - and kills about 1 million Indians a year.[1] Diabetes mellitus is multi factorial and multi systemic disorder in which high blood sugar is the cause of all morbidities that affect our gingival health also. The bacteria cause inflammation of the gums that is called gingivitis. In gingivitis, the gums become red, swollen and can bleed easily. Gingivitis is a mild form of gum disease which when not treated turns into Periodontitis. In periodontitis, gums pull away from the teeth and form spaces called pockets, that become infected.[2] The association between diabetes mellitus and periodontitis has long been discussed with conflicting conclusions. Most of the connective tissue destruction-taking place in periodontal disease results from the interaction of bacteria and their products with mononuclear cells.[3] One possible mechanism to explain as to why diabetics have more severe periodontal disease is that glucose mediated AGE (advanced glycation end products) accumulation would affect migration and phagocytic activity of mononuclear and polymorphonuclear phagocytic cells resulting in establishment of more pathogenic sub-gingival flora. This triggers an infection-mediated pathway of cytokine regulation, especially with secretion of TNF-α and IL-l and a state of insulin resistance, affecting glucose utilizing pathways. Excessive local secretion of TNF-α and IL-l also mediates tissue destruction of connective tissue and alveolar bone evident in periodontal disease.[4] Glycated hemoglobin (HbA1c) is a form of hemoglobin that is measured primarily to identify average plasma glucose concentration over prolonged periods of time. When blood glucose levels are high, glucose molecules attach to the hemoglobin in red blood cells. The longer hyperglycemia occurs in blood, the more glucose binds to hemoglobin in the red blood cells and the higher the glycated hemoglobin. So this HbA1c test provides an estimate of glycemic control over a period of approximately two to three months before the test, and the normal value is less than 6 percent.[5]

Material & methods : This study was carried out on 158 subjects of 35-45 years age group who have consulted at dental clinic for various dental problems. All procedures followed were in accordance with the ethical standards of experimentation and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was taken from all subjects. Personal and family history of systemic diseases like diabetes and hypertension was taken in detailed performa. All subjects were examined for gingival & periodontal health that includes colour & texture of gingiva, bleeding on probing, presence of plaque and calculus, depth of periodontal pockets if present, gingival recession and tooth mobility. On the basis of presence or absence of these signs they were diagnosed either as normal gingival health or as case of gingivitis and periodontitis. Then glycemic control of all these subjects was measured by doing glycated haemoglobin assay (HbA1c) by high performance liquid chromatography in biochemistry laboratory. Data was analyzed using Graphpad prism (6.0.3) software.

Result :

Out of 158 subjects, 87 (55.06%) subjects have one or more signs of gingivitis and periodontitis while 71 (44.93%) subjects have normal gingival health.

Table 1 : Association of gingival & periodontal health with HbA1c value

	HbA1c >6%	HbA1c <6%	Total
Gingivitis & Periodontitis present	69	18	87
Gingivitis & Periodontitis absent	27	44	71
Total	96	62	158

P<0.0001 by chi square which is statistically significant.

Table 2 : Sex distribution of gingival & periodontal health

	Male	Female	Total
Gingivitis & Periodontitis present	38	49	87
Gingivitis & Periodontitis absent	40	31	71
Total	78	80	158

P>0.0001 which is statistically not significant.

Discussion :

Years of research have established a number of mechanisms by which diabetes can influence the periodontium. Many of these mechanisms share common characteristics with those involved in the classic complications of diabetes, such as retinopathy, nephropathy, neuropathy, macrovascular diseases and altered wound healing. Because periodontal diseases are infectious diseases, research initially focused on possible differences in the subgingival microbial flora of patients with and without diabetes. Although some early studies reported higher proportions of certain bacteria in the periodontal pockets of

patients with diabetes, later studies involving cultures generally revealed few differences in periodontally diseased sites of subjects with diabetes and those of subjects who did not have diabetes.[6] Because the pathogens associated with periodontitis do not appear to differ greatly in people with and without diabetes, researchers have focused attention on potential differences in the immunoinflammatory response to bacteria between people with diabetes and those without diabetes. The function of cells

involved in this response, including neutrophils, monocytes and macrophages, is altered in many people with diabetes. The adherence, chemotaxis and phagocytosis of neutrophils often are impaired.[7] These cells are the first line of host defense, and

inhibition of their function may prevent destruction of bacteria in the periodontal pocket, thereby increasing periodontal destruction. The accumulation of advanced glycation end products (AGEs) in patients with diabetes also increases the intensity of the immunoinflammatory response to periodontal pathogens, because inflammatory cells such as monocytes and macrophages have receptors for AGEs.[8] Interactions between AGEs and their receptors on inflammatory cells result in the increased production of proinflammatory cytokines such as IL-1β and TNF-α.[9] Our study findings showed significant association between poor glycemic control and occurrence of gingivitis & periodontitis which is same as meta analysis conducted before 1996 by papapanou.[10] Diabetes also

may increase the risk of experiencing continued periodontal destruction over time. For example, a two-year longitudinal study demonstrated a fourfold increased risk of progressive alveolar bone loss in adults with type 2 diabetes compared with that in adults who did not have diabetes.[11]

Conclusion :

Dentists should discuss with their patients the relationships between diabetes and periodontal health, using the evidence as a basis for discussion. Diabetes is associated with an increased risk of developing inflammatory periodontal diseases, and glycemic control is an important determinant in this relationship. Research reveals numerous biologically plausible mechanisms through which these interactions occur. Large, randomized, controlled intervention trials are needed to extend the evidence base.

REFERENCE

1. Gale, Jason (November 7, 2010). "India's Diabetes Epidemic Cuts Down Millions Who Escape Poverty". Bloomberg. Retrieved 8 June 2012. | 2. http://www.nidcr.nih.gov/OralHealth/Topics/GumDiseases/PeriodontalGumDisease.htm | 3. Denis FK, Bartold PM. Clinical relevance of the host response of periodontitis. Periodontology 2000; 43: 278-93. | 4. GrossiS G,G encoR J. Periodontadl disease and diabetes mellitus: a two-way relationship. Ann Periodontol1998; 3: 51-61. | 5. Mealey BL, Ocampo GL. Diabetes mellitus. Periodontal 2000 (in press) | 6. Mealey B. Diabetes and periodontal diseases J Periodontol 1999;70(8):935-49. | 7. Manoucher-Pour M, Spagnuolo PJ, Rodman HM, Bissada NF. Comparison of neutrophil chemotactic response in diabetic patients with mild and severe periodontal disease. J Periodontol 1981;52(8):410-5. | 8. Schmidt AM, Weidman E, Lalla E, et al. Advanced glycation end products (AGEs) induce oxidant stress in the gingiva: a potential mech anism underlying accelerated periodontal disease associated with dia bêtes. J Periodontal Res 1996;31(7):508-15. | 9. Schmidt AM, Hori O, Cao R, et al. RAGE: a novel cellular receptor for advanced glycation end products. Diabetes 1996;45(supplement 3):S77-S80. | 10. Papapanou PN. Periodontal diseases: epidemiology. Ann Peri odontol 1996:1(1):1-36. | 11. Taylor GW, Burt BA, Becker MP, et al. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. J Periodontol 1998;69(1):76-83. |