DIABETES AND TOOTH LOSS

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CONTEXT: Diabetes is one of the most common diseases of the modern times and the most frequent systemic disorder connected with the development of periodontal disease. Diabetes mellitus is a metabolic disorder characterized by hyperglycemia due to insulin deficiency or secretion. Periodontal disease is an inflammatory disease caused by infection of supporting tissues of teeth and may subsequently lead to tooth loss if left untreated. The pathogenic collagenic mechanisms of diabetes stimulate the more intense progression of periodontal disease. These mechanisms include metabolic impairment, vascular changes, and changes of microbiotic flora in periodontal pocket and functional disorders of leukocytes. Tooth loss is an inevitable result of periodontal disease and destructive features of periodontal disease are more common in diabetics. Also, the prevalence of periodontal disease and the need of dental treatment are significantly higher in diabetes. AIMS AND OBJECTIVES: To compare & correlate the number of missing tooth in a group of 100 diabetic patients with that of 100 non-diabetic individuals (30-60 years of age). MATERIALS AND METHODS: 200 individuals (100 diabetic & 100 control) were examined for dental status. Dental status was recorded for the teeth that were missing including third molars. Diabetic patients with other systemic manifestations like hypertension, cardiac diseases were excluded from the study. Data were collected on the subject's age, sex, occupation and location. All data were recorded in WHO Oral Health assessment forms. Appropriate statistical tests were employed for analysis. **STATISTICAL ANALYSIS:** Statistical analysis was done using SPSS software. Kruskal Wallis Chi-square test was employed for statistical analysis. **RESULTS & CONCLUSION:** The mean number of missing teeth in diabetic group was found to be 6.45 which significantly exceeded that of control group (1.43; p<0.001). The mean number of teeth present was found to be higher in control group, the mean number of missing teeth was found to be higher in diabetic group. KEYWORDS: Diabetes, Hyperglycemia, Tooth loss, Advanced glycation end products, Vascular changes, Periodontitis.

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INTRODUCTION: Diabetes is one of the most common disease of the modern times and one of the most frequent systemic disorders that can be connected with the development of periodontal disease.¹ Diabetes mellitus and periodontal disease have long been considered to be biologically linked.² Diabetes mellitus comprises a group of metabolic diseases characterized by hyperglycemia resulting from deficiency in insulin secretion, insulin action or both. Periodontal disease is an inflammatory disease caused by infection of the supporting tissue around the teeth which may result in tooth loss if left untreated.³ It is assumed that, there are pathogenic collagenic mechanisms of diabetes that stimulate the more intense progression of periodontal disease. These mechanisms include metabolic impairment, vascular changes in periodontium, changes of microbiotic flora in periodontal pocket and functional disorders of leukocytes.¹ According to many researchers, there is a statistically non significant difference in caries frequency between diabetic and healthy persons. Tooth loss is an inevitable result of periodontal disease and destructive features of periodontal disease are more common in diabetic patients. Also, the prevalence of periodontal disease and the need of dental treatment are significantly higher in diabetes.² Hyperglycemia progressively glycates body proteins, forming advanced glycation end products (AGE), which stimulate phagocytes to release inflammatory cytokines such as TNF-α and IL-6.4

Current evidence regarding the biologic link between diabetes and periodontal disease supports diabetes and persisting hyperglycemia leading to an exaggerated immuno-inflammatory response to the periodontal pathogenic bacterial challenge, resulting in more rapid and severe periodontal tissue destruction. In the metabolic dysregulation of diabetes, persisting hyperglycemia causes non-enzymatic glycation and oxidation of proteins and lipids, and the subsequent formation of advanced glycation end products (AGEs), which accumulate in the plasma and tissues. Hyperglycemia and resultant AGE formation are considered to be a major causal factor in the pathogenesis of diabetes complications.²

AIMS AND OBJECTIVES: To compare the correlation between the number of missing tooth and diabetes in a group of 100 diabetic patients with that of 100 non-diabetic individuals of the same age group.

MATERIALS AND METHODS: Patients belonging to the age group of 30 to 60 years, who visited the OPD and the in-patients of a tertiary care hospital, were randomly selected for the study. The oral cavity of 200 individuals was examined. A case control study was undertaken comprising of 100 diabetic patients including 46 males and 54 females. The control group included 100 samples with 38 males and 62 females. Dental status was only recorded for the teeth that were missing including third molars. Diabetic patients with other systemic manifestations like hypertension, cardiac diseases were excluded from the study. Data were also collected on the subject age, sex, occupation and location. All data were recorded in WHO Oral Health assessment forms. Appropriate statistical tests were employed to analyse the data.

INCLUSION CRITERIA:

Known diabetic patients Patients belonging to the age group of 30-60 years **EXCLUSION CRITERIA:** Patients with history of other systemic disorders

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RESULTS: The mean number of missing teeth in diabetic group was found to be 6.45 which significantly exceeded that of control group (1.43; p<0.001). Although the number of missing teeth does not give a direct insight into the periodontal status, it is an important factor in estimating oral health. It is known that by the age of 34, teeth are mostly lost due to caries, and after the age of 34 the cause is in most cases periodontal disease. In this study, the difference between the two groups was found to be statistically significant for age (P<0.01), number of teeth present (P<0.001), number of missing teeth (P<0.001) and the number of tooth lost (P<0.001). The mean number of teeth present was found to be higher in control group, the mean number of missing teeth was found to be higher in diabetic group and the mean number of tooth lost was found to be higher in diabetic group.

The mean tooth loss between the different age groups was found to be statistically significant (P<0.05). Higher mean loss was recorded in 46-50 yrs age group followed by >55 yrs age group and 51-55 yrs age group respectively. The difference in mean tooth loss was found to be statistically significant between \leq 40yrs & >55yrs age group (P<0.05) and 41-45 yrs & >55 yrs (P<0.01). The mean tooth loss (as it was loose) between the different age groups was found to be statistically significant (P<0.01). Higher mean loss was recorded in 46-50 yrs age group followed by >55 yrs age group and 51-55 yrs age group respectively. In both the groups, age was found to be a significant factor influencing the number of tooth lost (P<0.05). In control group age could explain 4.7% of the variation in tooth loss and in diabetic group it could explain 4.3% of the variation in tooth loss.

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DISCUSSION: Severe periodontal disease often coexists with severe diabetes mellitus. Diabetes is a risk factor for severe periodontal disease. An infection-mediated up regulation cycle of cytokine synthesis and secretion by chronic stimulus from lipopolysaccharide (LPS) and products of periodontopathic organisms may amplify the magnitude of the advanced glycation end product (AGE)-mediated cytokine response operative in diabetes mellitus.⁵ Control of chronic periodontal infection is essential for achieving long-term control of diabetes mellitus. ⁵ The association between diabetes mellitus and periodontitis has long been discussed with conflicting conclusions. Both of these diseases have a relatively high incidence in the general population (diabetes 1% to 6% and periodontitis 14%) as well as a number of common pathways in their pathogenesis (both diseases are polygenic disorders with some degree of immunoregulatory dysfunction). On the one hand, numerous reports indicate a higher incidence of periodontitis in diabetics compared to healthy controls, while other reports fail to show such a relationship.⁶ In hyperglycemic states found in diabetics, a non-enzymatic glycation and oxidation of proteins and lipids occurs. As a result, advanced glycation end products (AGEs), particularly N-(carboxymethyl) lysine, accumulate in the plasma and tissues of diabetic subjects. This accumulation has been linked to the development of pathogenic complications of diabetes. Many of the effects of AGEs are receptor-dependent and involve a multi-ligand member of the immunoglobulin superfamily of cell surface molecules. The best characterized of these is the receptor for advanced glycation end products (RAGE), which is expressed by multiple cell types including endothelium and mononuclear phagocytes. RAGE plays a

central role in oral infection, exaggerated inflammatory host responses, and destruction of alveolar bone in diabetes. It is possible that antagonists of RAGE might have a valuable adjunctive therapeutic role for the management of periodontal disease found in diabetics.⁷

Diabetes-induced changes in immune cell function produce an inflammatory immune cell phenotype (upregulation of proinflammatory cytokines from monocytes/polymorphonuclear leukocytes and downregulation of growth factors from macrophages). This predisposes to chronic inflammation, progressive tissue breakdown, and diminished tissue repair capacity. Periodontal tissues frequently manifest these changes because they are constantly wounded by substances emanating from bacterial biofilms. Diabetic patients are prone to elevated low density lipoprotein cholesterol and triglycerides (LDL/TRG) even when blood glucose levels are well controlled.⁸

Glavind et al found that diabetes had 9.5% teeth less at age of 20-40 yrs, whereas Bacic et al found the mean tooth loss of 12.3 was noted. Mackenzie and Millard also found greater number of extracted because of alveolar bone loss. On the other hand, the studies by Ainan et al and Oliver et al showed that well controlled diabetes do not loose more teeth than healthy individuals and the risk of periodontitis in diabetes can be reduced by minimising plaque and calculus under professional dental care.¹ A study by Ivana Cukovic Bagic, Zeljiko Verzak, Nikica Car, Antoinette Car concluded that the mean number of missing teeth per person in diabetic group was 16.4, which significantly exceeded the mean number of extracted teeth in control subjects (13.6, p<0.001)which is correlating with this study. The etiopathogenesis of periodontal disease is complex. Several factors are probably responsible for the increased risk of periodontal disease in diabetic subjects. Systemic inflammation and hyperglycaemia are thought to play an important role in the pathogenesis of periodontal disease in diabetic subjects.³ The mechanism of diabetes correlation with periodontitis primarily involves vascular changes, neutrophilic dysfunction, impaired collagen synthesis and genetic predisposition. It is known that diabetes induces vascular changes in all tissues, including capillaries of periodontal structures. Gingival capillaries undergo basal membrane thickening, however, other pathologic changes such as membrane disruption, intramembranous presence of collagen and edematous endothelium may also be observed. These changes have been postulated to impair leukocyte migration, immune factor activities and thus contributing to progression of periodontitis and tooth loss by disordered microcirculation in diabetes.¹

In addition to the substantial evidence demonstrating diabetes as a risk factor for poor periodontal health, there is a growing body of evidence supporting periodontal infection adversely affecting glycemic control in diabetes and contributing to increased risk for the pathogenesis of diabetes complications. Because of the high vascularity of the inflamed periodontium, this inflamed tissue may serve as an endocrine-like source for TNF- α and other inflammatory mediators. Because of the predominance of Gram-negative anaerobic bacteria in periodontal infection, the ulcerated pocket epithelium is thought to constitute a chronic source of systemic challenge from bacteria, bacterial products and locally produced inflammatory mediators.

TNF- α , IL-6, and IL-1, all mediators important in periodontal inflammation, have been shown to have important effects on glucose and lipid metabolism, particularly following an acute infectious challenge or trauma. TNF- α has been reported to interfere with lipid metabolism and to be an insulin antagonist. IL-6 and IL-1 have also been reported to antagonize insulin action.²

A study conducted on "Periodontal disease: associations with diabetes, glycemic control and complications" supports that diabetes have an adverse effect on periodontal health and periodontal infection have an adverse effect on glycemic control and incidence of diabetes complications. ²

Another study done on "Association between type 1 and type 2 diabetes with periodontal disease and tooth loss" confirmed an association between both type 1 and type 2 Diabetes Mellitus with periodontitis and tooth loss.³

A study on "Tooth Loss among Diabetic Patients" showed that the number of extracted teeth per subject to be significantly greater in the group of diabetes than in the control group. Furthermore, patients with non insulin dependent diabetes had significantly more extracted teeth than those with insulin dependent diabetes. The number of teeth was found to increase with the disease duration.¹

Graves and colleagues described that diabetes has been reported to adversely affect bone repair by decreasing expression of genes that induce osteoblast differentiation, and diminishing growth factor and extracellular matrix production.²

CONCLUSION: It can be concluded that inadequate metabolic control, dental calculus and long standing diabetes can increase the risk of periodontitis as well as of tooth loss. On the other hand, diabetics who regularly control their disease and oral health through selfcare and regular oral professional care, have a lower risk of tooth loss. This study results pointed that the number of missing teeth were significantly higher in diabetic patients than in the control group. The mean tooth loss was highest in the age group of 46-50 years in diabetic patients (7.65) while it was much less in the control group (3.25) for the same age group. The higher percentage of tooth loss may suggest that diabetic patients are not aware of oral health and the necessity of oral hygiene control by which the risk of tooth loss can be reduced irrespective of the type and duration of the disease.

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Graph 1:







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Parameter	Group	Mean	Std dev	SE of Mean	Mean difference	Z	P-Value
Ago	Control Group	46.76	9.11	0.91	4.040	-	0.002*
Age	Diabetic Group	50.80	6.57	0.66	-4.040	3.117	
Number of	Control Group	30.55	3.45	0.35	5 000	-	<0.001*
teeth present	Diabetic Group	25.55	7.94	0.79	5.000	7.411	<0.001
Number of	Control Group	1.43	3.45	0.35	5 020	-	<0.001*
missing teeth	Diabetic Group	6.45	7.94	0.79	-3.020	7.460	
Tooth lost	Control Group	1.17	3.47	0.35			
(as it was mobile)	Diabetic Group	5.68	8.16	0.82	-4.510	6.300	<0.001*

Table 1: Comparison of different parameters between the two groups:

*denotes significant difference

Table 2: Comparison of tooth loss within diabetic group according to age:

Age	N	Mea n	Std dev	SE of Mea	95% Confidence Interval for Mean		Mi	Ma x	Kruska l Wallis Chi-	P- Value
				n	Bound	Bound			square	
≤40 yrs	8	2.50	3.42	1.21	-0.36	5.36	0	9		
41-45	1	2.50	3.44	0.86	0.66	4.34	0	10		
yrs	6						-			
46-50	2	7 65	11.7	2.45	2 56	12 74	0	32		0.042
yrs	3	/.00	7	2.10	2.00	12.7 1	Ŭ	02	9.914	*
51-55	2	5 76	8.61	1 7 2	2 20	0.32	0	32		
yrs	5	5.70	0.01	1.72	2.20	9.32	U	52		
>55yrs	2	6.71	6.50	1.23	4.19	9.23	0	32		
	0									

*denotes significant difference

Table 3: Correlation (Spearman's rank correlation) between age and number of tooth lost:

Group	Spearman's ρ	P-Value
Control group	0.439	< 0.001*
Diabetic group	0.207	0.039*

*denotes significant correlation

The correlation between age and number of tooth lost was found to be moderate (ρ =0.439) and significant (P<0.001) in control group. The correlation was found to be weak (ρ =0.207) and significant (P<0.05) in diabetic group.