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Original Article

Study of Lipid Profile and Glutamic Acid Decarboxylase (GAD) Auto-antibodies Levels in Patients With Periodontitis With and Without Diabetes Mellitus

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Abstract

Diabetes mellitus is a systemic disease with several major complications affecting both the quality and length of life. One of these complications is periodontal disease (periodontitis). Periodontitis is much more than a localized oral infection. Recent data indicate that periodontitis may cause changes in systemic physiology. The interrelationships between periodontitis and diabetes provide an example of systemic disease predisposing to oral infection, and once that infection is established, the oral infection exacerbates systemic disease.

Materials and methods: A total of 150 periodontitis patients with diabetes mellitus and 150 periodontitis patients without diabetes mellitus have selected for the study. Glutamic acid decarboxylase (GAD) auto-antibodies and lipid profile levels have compared with 100 healthy non-diabetics, non-periodontitis subjects. Fasting blood sugar, lipid profile have done by semi auto-analyzer diagnostic kit and Glutamic acid decarboxylase (GAD) auto-antibodies by ELISA Method.

Results: Analysis of lipid profile, Glutamic acid decarboxylase (GAD) have showed a significant relationship between periodontal disease in both diabetics and non-diabetics.

Conclusion: The results of this study indicate that the presence of a significant relationship between periodontitis and diabetes mellitus, hence lipid profile and glutamic acid decarboxilase- auto antibodies are useful in predicting the future risk of periodontitis and diabetes mellitus.

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Introduction

Diabetes mellitus is a systemic disease with several major complications affecting both the quality and length of life (1). One of these complications is

periodontal disease (periodontitis). Periodontitis is much more than a localized oral infection. Research in several countries indicates that 5–20% of any population will have severe periodontal disease and a majority of adults suffer from moderate forms of this disease (2). Evidence suggests that one out of two adults above the age of 35 years have periodontal disease in India, and 35% of the teeth extracted are as a consequence of periodontal disease (3). One study reported 13.3, 12.5, 29.1, and 97% prevalence of various periodontal parameters (4).

The increased prevalence and severity of periodontal disease typically seen in patients with diabetes mellitus, especially those with poor metabolic control, has led to the designation of periodontal disease as the 'Sixth Complication of Diabetes' (5). The American Diabetic Association has officially recognized that periodontal disease is common in patients with diabetes, and the Association's Standards of Care include taking a history of current or past dental infections as part of the medical examination (6, 7).

Periodontal diseases and diabetes are both common complex chronic diseases for which there is considerable evidence for a bidirectional relationship [8]. It has long been accepted that the prevalence, severity and progression of periodontal diseases are higher in diabetic patients when compared with non-diabetic subjects (9, 10), which confirm the notion of diabetes as a major risk factor for periodontitis. It is becoming increasingly difficult to ignore the importance of glycaemic control as a key determinant for the increased risk of periodontitis in diabetic individuals. On the other hand, periodontal diseases have a significant negative impact on the glycaemic control in diabetes (i.e. having periodontitis increases the risk of poor glycaemic control over time) (11). It is clear from a large volume of published studies that diabetes is a major risk factor for periodontal disease, and that the risk for periodontitis is greater if glycaemic control is poor.

This study reviews the relationship between diabetes and oral health, particularly focusing on periodontal diseases.

Materials & Methods

For this study newly diagnosed type 2 diabetic and periodontitis patients are selected. The study protocol has in keeping with the ethical guidelines of the 1975 declaration of Helsinki and all the patients have gave written informed consent to the study. Patients are taken from outpatient department of periodontics Govt. Dental College Indore. This study is conducted in the department of Biochemistry, M.G. M. Medical College Indore, M.P. BMI of all patients is calculated by using the formula weight in Kg/m^2. Brief clinical history, Blood pressure, dietary habit and information on physical activity are taken before entry of all patients.

Sample size:

1. Control group (healthy non-diabetic, non-periodontitis) – 100.

2. Study group – (a) Non diabetic adults with periodontitis – 150.

(b) Diabetic adult with periodontitis – 150.

Methodology:

– Estimation of fasting blood sugar is done by GOD-POD method.

– Estimation of serum lipid profile is done by enzymatic method.

– Serum Glutamic acid decarboxylase auto-antibodies is determined by ELISA method.

Observations & Results

Analysis of Glutamic acid decarboxylase auto-antibodies is found significant relation with the diabetics with periodontitis ($p<0.030$) when compared to control healthy groups. In this study, in diabetic patients the presence of GAD–auto-antibodies are low while in normal healthy patients is high, it means diabetes kills the GAD auto-antibodies in patients.

TABLE I : Comparison between Three Groups (Diabetes mellitus + Periodontitis, Non-Diabetes mellitus + Periodontitis and Non-Diabetes mellitus + Non-Periodontitis)

Characteristics (mean±SD)	*DM+P(150)*	*NDM+P(150)*	*NDM+NP(100)*	*F-Value*	*P-value*
Age (years)	43.33±10.08	43.87±7.30	41.53±6.37	1.880	0.155
Fasting bloodglucose (mg/ dL)	142.3±5.26	102.72±2.36	92.4±3.17	137.252	<0.0001
Total cholesterol(mg/dL)	252±10.6	253.6±9.6	210.1±13.6	269.581	<0.0001
VLDL(mg/dL)	39.5±1.35	37.25±0.29	34.18±1.81	271.723	<0.0001
Triglyceride (mg/dL)	149.2±19.3	127.4±12.6	120.36±2.3	139.152	<0.0001
LDL (mg/ dL)	123.2±3.61	126.41±3.2	126.98±4.1	117.355	<0.0001
HDL (mg/dL)	35.7±2.35	36.2±2.45	37.2±0.721	9.687	<0.0001
BMI (kg/m^2)	24.30±3.42	23.71±3.78	21.19±3.13	17.780	<0.0001

TABLE II : Comparison of Glutamic acid decarboxylase (GAD) auto-antibodies betweenThree Groups (Diabetes mellitus + Periodontitis, Non-Diabetes mellitus + Periodontitis and Non-Diabetes mellitus + Non-Periodontitis)

	GAD auto-antibodies		*Chi Sq*	*P-value*
	Present	Absent		
DM+P	69	81	7.03	0.030
NDM+P	78	72		
NDM+NP	67	33		

DM=Diabetes Mellitus, P=Periodontitis, NDM=Non-Diabetes Mellitus, NP=Non- Periodontitis

The level of total cholesterol and triglycerides is also found significant correlation with diabetics with periodontitis and non diabetics with periodontitis when compared to healthy control groups and p<0.0001. Study of low density lipoproteins (LDL) showed a significant relationship between periodontal disease and in both diabetics and non-diabetics. Serum LDL levels in diabetics with periodontitis is 123.2±3.61 mg/dL, (p<0.0001), While in non-diabetics with periodontitis is 126.41±3.2 mg/dL, (p<0.0001) when compared to control groups (non-periodontitis and non- diabetics) 126.98±4.1 mg/dL. High density lipoproteins (HDL) showed a significant relationship between periodontal disease and in both diabetics and non-diabetics. Serum HDL levels in diabetics with periodontitis is 35.7±2.35 mg/dL, (p<0.0001), While in non-diabetics with periodontitis is 36.2±2.45 mg/dL, (p<0.0001) when compared to control groups (non-periodontitis and non- diabetics) 37.2±0.72 mg/ dL. Very low density lipoproteins (VLDL) also showed a significant relationship between periodontal disease and in both diabetics and non-diabetics.

Age is not significant with the diabetics with periodontitis 43.33 ±10.08 and non-diabetics with periodontitis 43.87±7.30 when compared to healthy controls groups 41.53±6.37 and p<0.155.

BMI is significantly higher in the diabetics with periodontitis (p<0.0001).

Discussion & Conclusion

Diabetic patients are prone to elevated low density lipoprotein cholesterol and triglycerides (LDL/TRG) even when blood glucose levels are well controlled (12). This is significant, as recent studies demonstrate that hyperlipidemia may be one of the factors associated with diabetes-induced immune cell alterations. Recent human studies have established a relationship between high serum lipid levels and periodontitis. Some evidence now suggests that periodontitis itself may lead to elevated LDL/TRG. Periodontitis-induced bacteremia/endotoxemia has been shown to cause elevations of serum proinflammatory cytokines such as interleukin-1 beta (IL-1 beta) and tumor necrosis factor-alpha (TNF-alpha), which have been demonstrated to produce alterations in lipid metabolism leading to hyperlipidemia (13, 14, 15).

An abundance of evidence has demonstrated that all five single components of metabolic syndrome (BMI, blood pressure, serum HDL-cholesterol, serum triglycerides and impaired fasting glucose) predicted the occurrence of T2DM (16).

Interestingly, the present study showed significantly higher levels of triglycerides and lower levels of HDL cholesterol in diabetic subjects compared to non-

diabetic controls. This finding is in agreement with previous studies which demonstrated a high prevalence of elevated triglycerides and low HDL cholesterol in individuals with diabetes (17). Another study demonstrated a trend for increased levels of total cholesterol in subjects with T2DM compared to non-diabetic subjects (17). On the other hand, Kardesler et al. (2010) found no significant differences in total cholesterol, triglycerides and low density lipoprotein levels between patients with T2DM and non-diabetic subjects (18).

The results of present study suggest that the periodontal disease can raise blood lipid levels. Although we found higher total cholesterol and triglycerides levels in the test group, and also more frequent pathological values of serum lipids, the results are not statistically significant as those of the works of Cutler et al (19) and Lösche et al. (20).

Cutler et al. (19) showed a significant association between periodontitis and hyperlipidemia, in relation to the triglycerides and total cholesterol levels. However, the groups were not age-matched, and the mean age of the group with disease was 50.5 years, statistically higher than that of the control group, 41.6 years.

Similar methodology was used in the study of Lösche et al. (20) in which the authors observed significantly higher total cholesterol levels, LDL and triglycerides in the group with periodontal disease in comparison to the group without the disease. In our study we verified a positive correlation between tooth loss and the levels of TC, LDL and triglycerides and a negative correlation between tooth loss and the levels of HDL; there is a statistically significant correlation between tooth loss and age.

Very little has been reported in the literature regarding study of glutamic acid decarboxylase (GAD) autoantibodies in subjects with T2DM and periodontitis. One study demonstrated the study of glutamic acid decarboxylase (GAD) autoantibodies in patients with IDDM (21) reported significantly elevated GAD autoantibodies levels in combination with elevated Ig G titers to Pg before treatment is indicative of IDDM patients with refractory periodontitis. In the present study, significant difference is found in the study of glutamic acid decarboxylase (GAD) autoantibodies in patients with T2DM [p=0.030].

In our study, we concluded that the presence of a significant relationship between periodontitis and diabetes mellitus, hence lipid profile and glutamic acid decarboxilase- auto antibodies are useful in predicting the future risk of periodontitis and diabetes mellitus.

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