Journal of Natural Sciences Research www.iiste.org
ISSN 2224-3186 (Paper) ISSN 2225-0921 (Online)
Vol.5, No.10, 2015

Estimation of Humoral Immunological Parameters among Chronic Periodontitis Patients with Type 2 Diabetes Mellitus

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Abstract

Background: Periodontitis is a bacterial infection of tooth-supporting tissues. The host reacts to this bacterial challenge by activating its defense mechanisms; the immune responses can be mediated either by humoral factors or by lymphocytes. Recently studies found that periodontitis might be related to several systemic diseases especially diabetes mellitus.
Objectives: The current study was established to evaluate the alteration in humoral immune response in chronic periodontitis with type 2 diabetes mellitus as compared controls.
Subjects and Methods: A total of 30 patients and 20 controls were included in the study. Blood samples were collected from each subject to determine serum concentrations of (IgA, IgG, IgM, C3 and C4) by single radial immune diffusion method. In this study, clinical examination included plaque index, gingival index, probing pocket depth, clinical attachment level and bleeding on probing.
Results: serum concentrations of IgG and IgA are significantly higher ($p< 0.05$) in patients than healthy control, and serum concentration of C3 is significantly less than corresponding value of control ($p< 0.05$), while there are no significant ($p> 0.05$) differences in serum concentrations of each IgM and C4 between the patients and controls. Furthermore there are no significant ($p> 0.05$) correlation between serum immunological parameters and periodontal parameters.
Conclusion: These findings corroborate the thought that the humoral immune response plays a crucial role in the pathogenesis of periodontitis with type 2 DM. In addition, elevated antibody levels may explain why diabetes aggravates periodontitis.

Keywords: periodontitis, diabetes mellitus, immunoglobulin, complement componenet

1. Introduction

Periodontitis is defined as inflammatory disease of supportive tissue of teeth caused by specific microorganism which lead to progressive destruction of periodontal membrane and alveolar bone, with formation of periodontal pockets and gingival recession (Savage *et al.*, 2009).The incidence and progression rate of periodontitis depends on complex interaction between periodonotopathic bacteria, cells of the host immune system and environmental factors (Page *et al.*, 1997). Evidences indicate that periodontitis may have profound effects on systemic health. Epidemiologic studies reported that greater prevalence or severity of periodontitis was seen in diabetic individuals than in non-diabetic (Taylor and Borgnakke, 2008). Diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycaemia due to the defective secretion or activity of insulin. DM present with the classical triad of symptoms such as polydypsia, polyuria and polyphagia. This is often accompanied by chronic fatigue and loss of weight. Complications of DM include retinopathy, nephropathy, neuropathy, and cardiovascular disease (Khader et al., 2008). Periodontitis is now referred to as the fifth most common complication of diabetes. However, clinical studies have demonstrated a higher prevalence of periodontitis in diabetic patients. Periodontal disease and diabetes have a number of common pathways in their pathogenesis; both diseases are polygenic disorders with some degree of immunoregulatory dysfunction (Soskolne et al., 2001).

Immune response has been shown to play a crucial role in the initiation and progression of periodontal disease. It is well known that cellular immunity is reported to play a protective or aggressive role in the pathogenesis of periodontitis. Change immune function in diabetic patients with periodontitis has been showed in several researches (Anil *et al.,* 1990a, Anil *et al.,* 1995 and Albandar *et al.,* 2001). It is worthy to mention that some

studies indicated significant increase serum immunoglobulins and complement factors in diabetic patients with periodontitis (Anil *et al.,* 1990b and Anil *et al.,* 1990c). In 1999, Fontana and colleagues also reported that a systemic factor might be responsible for promoting the local pathological alterations, which produce gingivitis and periodontitis in diabetes patients (Fontana *et al.,* 1999). Therefore the present study is undertaken to evaluate the alteration in humoral immune response in chronic periodontitis with type 2 DM as compared controls.

2. Subjects and Methods

A total of 30 periodontitis patients with type 2 DM, their ages range (30-60) years, and 20 healthy controls, their age range (30-55) years were enrolled in this study. Periodontal parameters used in this study were plaque index (PI), gingival index (GI), probing pocket depth (PPD), clinical attachment level (CAL) and bleeding on probing (BOP). Blood samples were collected from all patients and controls, and then serum was separated from blood to estimate the concentration of IgA, IgG, IgM, C3 and C4 by single radial immune diffusion kits, and performed as recommended in leaflet with kits (Immuno Diffusion Biotechnologies, France).

Statistical analysis: It was assessed using P (T-test), correlation among different parameters was calculated by the Spearman correlation coefficient test, P value less than the 0.05 was considered statistically significant.

3. Results

The demographic characteristics of patients group and controls group included in this study are presented in table (1). There are no statistical significant differences ($p>0.05$) in ages and sexes were existed between two study groups. The mean age of patients was 45.21±6.89 years and for healthy controls was 41.49±4.62 year. All clinical periodontal parameters were significantly higher in the patients group as compared to the healthy control ($p< 0.001$).

Table-1: Demographic and Clinical Parameters in Patients and healthy Control groups .

	Study groups		**P-value**
	Periodontitis with type 2DM Patients n=30	**Healthy control n=20**	
Demographic Parameters			
Age Range	(30-60)	(30-55)	
Age Mean ±SD	45.21±6.89	41.49±4.62	0.947 NS
Female	16	11	
Male	14	9	
Clinical Parameters			
PI	1.54±0.66	0.79±0.39	P<0.001
GI	1.29±0.45	0.74±0.28	P<0.001
PD	2.23±0.94	0.84±0.39	P<0.001
CAL	2.30±1.03	0.0	P<0.001
BOP	24.66±9.79	5.76±1.67	P<0.001

The current results revealed that mean serum concentrations of IgA and IgG (269.07±14.2 mg/dl; 942.37±26.04 mg/dl) are significantly higher ($p< 0.05$) in patients than healthy control (163.48±9.7 mg/dl; 737.08±17.09 mg/dl) respectively, and serum concentration of C3 is significantly less (91.20±8.90 mg/dl) than corresponding value of control (136.4±9.78 mg/dl), ($p< 0.05$), while there are no significant ($p> 0.05$) differences in serum concentrations of each IgM and C4 between the patients and controls, as clearly observed in table (2). Furthermore there are no significant ($p> 0.05$) correlation between serum immunological parameters and periodontal parameters, table (3).

Table-2: The differences in mean serum concentrations of IgA, IgG, IgM, C3 and C4 concentrations in Studied Groups

	patients	Healthy control	P (T-test)
Serum IgA			
Range	(54.1-479.1)	(82.4-241.4)	
Mean	269.07±14.2	163.48±9.7	p< 0.05
Median	273.05	168.8	
Serum IgG			
Range	(598.4-1345.2)	(349.8-1085.6)	
Mean	942.37±26.04	737.08±17.09	p< 0.05
Median	886.8	686.1	
Serum IgM			
Range	(48.3-445.1)	(73.5-375.2	
Mean	200.83±10.13	220.12±11.13	NS
Median	122.2	138.0	
Serum C3			
Range	(84.4-220.5)	(52.9-200.5)	
Mean	91.20±8.90	136.4±9.78	p< 0.05
Median	93.25	135.25	
Serum C4			
Range	(9.2-40.6)	(7.5-36.4)	
Mean	21.60±4.23	19.04±3.91	NS
Median	21.95	18.65	

Table 3: Correlation between serum concentrations (IgA, IgG & C3) and clinical periodontal parameters in patients.

	PI		GI		PD		CAL		BOP	
	r	P	r	P	r	P	r	P	r	P
IgA	0.140	0.171	0.051	0.891	0.299	0.083	0.057	0.846	0.170	0.252
IgG	0.273	0.289	0.266	0.358	0.132	0.652	0.153	0.101	0.284	0.325
C3	0.072	0.902	0.235	0.151	0.109	0.45	0.143	0.32	0.040	0.528

r: correlation

4. Discussion

Recently, there has been intensive attention in potential associations between periodontitis and various chronic systemic diseases and conditions. In particular the association between diabetes and periodontitis has been well documented (Taylor, 2001). Linden and colleagues reported that periodontal disease is associated with an increased risk of premature death from any cause; suggest the hypothesis that periodontitis may be a risk factor for other diseases (Linden *et al.,* 2012, Linden *et al.,* 2013).

The present findings are in accordance with other studies, who have demonstrated significantly higher levels of IgA and IgG in of periodontitis patients with type 2 DM than those of control groups (Anil *et al.,* 1990a, Anil *et al.,* 1990b and Awartani, 2010). Similarly Fontana and colleagues showed significant elevation of these immunoglobulins among diabetic patients with periodontitis (Fontana *et al.,* 1999). Furthermore; Anil *et al.,* observed that the concentrations of the IgG and IgA in gingival tissues of diabetic and non-diabetic patients were found to be significantly high, when compared to the healthy subjects (Anil *et al.,* 2006). The tissue alterations caused by diabetes may create an environment that is less resistant to the invasion of microorganisms (Grant, 1996). Awartani in her study indicates that poor glycemic control may be associated with the increase in IgA and IgG serum antibodies. Elevated antibody levels may explain why poorly controlled diabetes exacerbates periodontal disease. So she concluded that the importance of the immune system as well as good glycemic control, especially in patients diagnosed with periodontitis. The increased incidence of periodontitis in diabetic patients suggests that the alteration in immune response may contribute to the pathogenesis of periodontitis in patients with poorly controlled diabetes (Awartani, 2010).

Inconsistent with our results Al-Jebouri and Al-Hadeethi who showed that serum levels of IgM, C3 and C4 were significantly higher in periodontitis patients than that in control (Al-Jebouri and Al-Hadeethi, 2014). On the other hand, previous studies indicated that the serum levels of complement components (C3 and C4) were significantly higher in periodontits patients with diabetic (Anil *et al.,* 1990b and Anil *et al.,* 1990c), Anyway, these results were disagreement with presnt results in this study.

One of the possible biological explanations for the epidemiological associations between DM and periodontitis, may be related to modification of antibody production. Several host-related determinants and environmental factors have been suggested to modify antibody production differentially. Both the periodontal and medical literature indicates that DM may be important in relation to variation in antibody production (Smith *et al.,* 1996).

5. Conclusion

In conclusion these findings corroborate the thought that the humoral immune response plays a crucial role in the pathogenesis of periodontitis with type 2 DM. In addition, elevated antibody levels may explain why diabetes aggravates periodontitis.

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