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ImmunologicalParameters amongPeriodontitis Patients with CoronaryHeart Disease and Diabetes

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ABSTRACT

Background: Periodontitis is a chronic inflammatory disease causesinflammation of the supporting structures of the teeth and especially the periodontal membrane, It is the most frequent cause of tooth loss in the population mostly in adult patients. Periodontitis is caused by microorganisms that adhere to and grow on the tooth's surfaces, along with an over-aggressive immune response against these microorganisms **Objective**: To examine the association of some immunological parameters such IgA, IgG antibodies and CRP among periodontitis patients with coronary heart diseaseand diabetes. **Results**:The mean age of patients was 42.21±4.89years and for healthy controls was 40.29±5.62. Revealed that the serum concentration of IgG and IgA are significantly higher than the control, whereas, levels of CRP are elevated in Periodontitis patients where the positive result appear at percent (93.3%), negative percent at (6.6%) in compares with control group at (20%), (80%) percentage for positive and negative result respectively. **Conclusion**:These findings documented that the immune response plays a serious role in the pathogenesis of periodontitis with coronary heart disease and DM. In addition, raised antibody and CRP levels may explain why CHD amplifies periodontitis. So these results concluded that the immune response plays a dynamic role in the pathogenesis of periodontitis with CHD and DM.

KEYWORDS: Chronic Periodontitis, Coronary HeartDisease,DM, CRP, IgG, IgA.

INTRODUCTION

Periodontitis is a local inflammatory process mediating destruction of periodontal tissues triggered by bacterial insult. However, this disease is also characterized by systemic inflammatory host responses that may contribute, in part, to the recently reported higher risk for coronary heart diseaseamong patients with periodontitis. Moderate elevation of C-reactive protein (CRP) has been found to be a predictor of increased risk for CHD.CRP is an acute phase reactant that is mainly produced in the liver in response to a variety of inflammatory cytokines such as IL-6. It therefore serves as a marker for systemic inflammation in a variety of conditions [1]. Elevated CRP levels in periodontal patients have been reported by several groups [23]. Clinical study revealed that serum C-reactive protein (CRP) value increased in periodontitis patients and that periodontal treatment improved the level of HbA(1C) in diabetic patients. These data indicate that periodontal pathogen influenced systemic conditions and these are partly improved by periodontal therapy. Also, periodontal pathogen possibly promotes atherosclerosis formation. Further investigation is necessary to clarify the

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relationship between diabetes and periodontal disease [22]. There is accumulating evidence that inflammation is an important risk factor in CHD. Elevated levels of the inflammatory marker high-sensitivity C-reactive protein (hs-CRP) are associated with increased risk for CHD and diabetes mellitus. Adding hs-CRP to the definition of the metabolic syndrome has been shown to improve the prediction of CHD. Elevated hs-CRP levels may also be predictive of development of the metabolic syndrome [15].It is worthy to mention that some studies indicated significant increase serum immunoglobulin's and complement factors in diabetic patients with periodontitis [2,3]. 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[12].Several antibodies that may impact pathogenic inflammatory responses in atherosclerosis have been identified. Several of these antibodies are examples of "molecular mimicry" wherein cross-reactive antibodies induced by periodontal pathogens recognize host antigens and modulate their function. In some cases, these antibodies increase the risk for or accelerate atherosclerosis by enhancing endothelial inflammation, promoting uptake of lipids into macrophages, or blocking anti-atherogenic effects of protective molecules[28].

Subjects and Methods:

Blood samples were collected from forty chronic periodontitis patients from both males and females, were clinical diagnosed by the specialized Dentists, and then serum was separated from blood to estimate the concentration of IgA, IgG, , by single radial immune diffusion kits, according to [4, 20] and performed as recommended in leaflet with kits (Immuno Diffusion Biotechnologies, France). Quantitative Determination of CRP Concentration in Serum by using latex agglutination according to the [13], which is a slide agglutination test for the detection of CRP in human serum; all samples were run in duplicate test. Also blood samples without anticoagulants in a rate of 5ml in plane tubes were collected from both patients and controls.Statistical analysis: It was assessed using P (T-test), P value less than the 0.05 was considered statistically significant.

Results:

The demographic study are presented characteristics of patients group and controls group included in table (1). The mean age of patients was 42.21±4.89years and for healthy controls was 40.29±5.62.

Table 1: ClinicalChactaristicsofStudyGroups.

Characters of study group	Chronic Periodontitis with CHD&DMn=30	Control n=10
Age Range	30-55	24-50
Age Mean ±SD	42.21±4.89	40.29±5.62
Female	14	6
Male	16	4
PD	7.1	2.2
GAL	4.2	Nill
BOP	21.3	8.1

The results in table (2) revealed that mean serum concentrations of IgG 431.97±208.950 (mg/dl)are significantly difference at (p< 0.05) in patients than healthy control (812.390±85.70 (mg/dl); the serum concentration for IgA is 243.567±187.544there is also Significantly difference in comparism with the control group at serum concentration 262.140±129.32.

Table 2:Mean serum concentration of IgG and IgA in chronic periodontitis with CHD and DM.

Patients		control	p. value
IgG			
Mean	431.97	812.390	0.0001
S.D	208.950	85.70	
IgA			
Mean	243.567	262.140	0.001
S.D	187.544	129.32	

CRP can be measured using immuno-turbidimetric or immuno-electrophoretic assays or latex slide agglutination method. CRP is an acute-phase reactant produced by the liver in response to diverse inflammatory

stimuli, in table (2) have shown that their levels of CRP are elevated in periodontal disease where the positive result appear at percent (93.3%), negative percent at (6.6%) in compares with control group which at percent (20%), (80%) for positive and negative result respectively.

Table 3:Prevalence of Concentration for CRP in Chronic Periodontitis with CHD and DM in compare with control groups

Study groups	NO.	Concentrationwith +ve result	Concentration with-ve result	Total
Chronicperiodontitis with DM and CVD	30	93.3%	6.6%	100%
Control	10	20%	80%	100%
Total	40			

Discussion:

Several reports have demonstrated a possible association of periodontalinfections with coronary heart diseaseby elevated immunoglobulin titer to periodontal bacteria in CHD andcontrols[27].The possible links between periodontitis associated with DM and CHD report evidence of a relationship between periodontal antibodies and CHD. That, particularly among diabetic subjects, those with elevated periodontal antibodies had significantly more coronary artery defect and higher blood pressure[12].

The present study showed that the mean serum levels of IgG and IgA were significantly higher in patients with periodontitis as compared with control groups as revealed in Table 2. These findings were similar to study reported by Califano*et al* [8] who reported that 60% of periodontitis patients were positives IgG and host response to bacteria in periodontitis can be detected by the serum immunoglobulin to specific Periodontalpathgenes. The results found here were almost agree to those of Engstrom*et al* [11] and Kobayashi*etal*[17]who revealed that the elevation in IgG level as a result of host response to bacterial colonization. Also, Awartani, [5]found that serum antibodies levels in type 2 diabetes patients have been considered important in preventing periodontal destruction in patients with chronic periodontitis. On the other hand, Craig*et al* [9] reported that the serum IgG antibody may be reflective of the destructive periodontal disease, and its level can be considered a risk indicator for disease progression. So serum antibodies levels are detected routinely in clinical practice because they provide key information on the humoral immune status that provide possible interaction between serum antibodies and clinical periodontal destruction in association to diabetes and CHD [14].

In the present data the both of IgG and IgA levels are significantly difference at ($p< 0.05$) in patients compare to control. Thus explain the putative relationship, and even elucidate a possible causal association among diseases. It is possible seen in diabetes and periodontitis at least associated with pathogens might increase the risk of cardiovascular disease groups are related to the existence of common risk factors and common underlying pathophysiology and serological evidences Mustapha *et al.*[21] and Lockhart *et al.*,[18].Berezow and Darveau[6] andPeter*et al* [25] revealed in the diabetes elevated glucose was more common among individuals with higher serum IgG antibody titer to periodontal pathogen (the red complex), this lead to increased severity and pathogenicity of the periodontal bacteria in the plaque biofilm, which in turn may lead to impaired arterial endothelial function in CVD, associated periodontitis and showed several mechanisms relating periodontal infections risk to stroke, similar to hypertension, and smoking.

While in regarding to the concentration of CRP the present study showed the serum level of CRP in chronic periodontitis with CHD and DM is significantly higher compare with controls. A large number of studies demonstrate that there are increased circulating levels of inflammatory mediators in patients with periodontal diseases compared to healthy controls. Elevated levels of many of these mediators are statistically associated with increase cardiovascular risk and are therefore thought to be potential mechanistic links between periodontal infection and CHD, either as disease markers or as participants in inflammatory responses in endothelial tissue and atheromatous lesions [16]. There is sample evidence that serum CRP and other acute phase reactant and inflammatory cytokine concentrations are higher in otherwise healthy individuals with chronic and aggressive periodontitis than in periodontally healthy controls [10].Furthermore, CRP levels may be elevated in sera from patients with aggressive periodontitis based on 2 studies [26, 29].Epidemiological data confirm that diabetes is a major risk factor for periodontitis; susceptibility to periodontitis is increased by approximately threefold in people with diabete. Furthermore, the risk of cardiorenal mortality (ischaemic heart disease and diabetic nephropathy combined) is three times higher in diabetic people with severe periodontitis than in diabetic people without severe periodontitis [7]. The elevated inflammatory state in diabetes contributes to both microvascular and macrovascular complications, and it is clear that hyperglycaemia can result in the activation of pathways that increase inflammation, oxidative stress and apoptosis[19]. The serum levels of IL-6 and CRP are also raised in patients with periodontitis, with IL-6 levels correlating with the extent of disease [24,

30]. The systemic inflammation that is associated with periodontal disease may therefore enhance the diabetic state.

Conclusion and Future work:

These findings documented that the immune response plays a serious role in the pathogenesis of periodontitis with coronary heart disease and DM. In addition, raised antibody and CRP levels may explain why CHD amplifies periodontitis and in future use these immunological markers as screening test for possibility of periodontitis.

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