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The Relationship between Periodontitis and Systemic rheumatoid arthritis

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Abstract: *There is historical evidence of link between periodontitis and rheumatoid arthritis. This associationhas been studied in 50 patients with severe periodontitis and 20 normal subjects as controls whom aged between (20-80) years at the period from (5-2013)to(2-2014) in specialized center of dental health of Babylon. The result was gave the prevalence of severe periodontitis in old patients were aged (60-80 years) 46% and female were moreeffected 60% than male 40%. According to tobacco smoking the result reveled 54% smoking patients with periodontitis compare with non- smoking 46%, while depending on another systemic disease result was gave 38% of patients with periodontitis have diabetic mellitus, 30% with hypertension and 48% with both diseases ago. The rheumatoid arthritis result of Rheumatoid factor (RF) reveled 24% of periodontal patients were suffer from RA compare with healthy control. Hematological study erythrocyte sedimentation rate mean count show significant increasing $p<0.0001$ for both sex of periodontal and RA patients compare with healthy control, While immunological study for the last patients reveled high significant level $P<0.0001$ for IL-1β and TNF-α compare with healthy control.*

I. Introduction

Growth of scientific evidence suggests an exquisite association between oral infection and systemic diseases. Thought etiologies of periodontitis and rheumatoid arthritis (RA) are separate, their underlying pathological processes are sufficient to warrant consideration of hypothesis that individual at risk of developing RA may also be at the risk of developing periodontitis andvice versa (Ranade and Doiphode, 2012). Oral infection can either cause or aggravate many diseases. The diseases frequently mentioned are arthritis chiefly of rheumatoid type, valvular heart disease, and skin, ocular and renal disease. (Shafer et al., 1993).

Periodontitis disease is an all- encompassing term relating to the destruction inflammatory disorders of the hard and soft tissues surrounding the teeth (Mercado et al., 2000). Periodontitis is initiated by pathogenic biofilm that accumulates around the gingival (gum) margin (Socranskyet al.,1998).

RA is a chronic, immune- mediated inflammatory disease that is characterized by synovial inflammation and progressivedestruction of cartilage and bone that often results in structural damage disability and loss of function. RA is associated with significant morbidity and an increased risk of mortality (premature death) (Gabriel etal., 1999).

Numerous infectious agents, including periodontal bacteria have been implicated as contributory to the etiology of RA (Ogrendiket al., 2005). Antibodies against oral anaerobic bacteria have been detected in serum (Mikulset al., 2008) and synovial tissue (Moen et al.,2003) ,and DNA from oral bacteria has been reported in serum and synovial fluid of individuals with RA (Moen et al .,2006).

The rheumatoid factor (RF) has been found in RA and in other chronic inflammation diseases, including PD (Rosenstein ED et al., 2004). The RF could be verified in the gingiva, in the subgingival plaque, and in the serum of patients with PD (McGraw WT et al., 1999). Seropositive patients with PD showed increased titers of IgG and IgM antibodies against oral microorganisms when compared with seronegative patients with PD(McGraw WT et al., 1999).

A high Erythrocyte Sedimentation Rate has been shown to be associated with poor prognosis in systemic inflammatory disease, but has no strong predictive value as a single marker (Ustunet al., 2002).

Several interleukins (ILs) (i.e IL-1, IL-6, IL-8, IL-15, IL-17) can be associated with RA similar proinflammatory cytokine, tumor necrosis factor (TNF)-α, have also been associated with inflammation in periodontitis (Harnandezet al., 2011).

The IL-1β is responsible for most of the systemic activity attributed to IL-1 including fever, activation of phagocytes, and production of acute phase proteins.(Stevens, 2010).

TNF-α is strong inflammatory mediator, modulating the destruction of cartilage and bon, and also elevated plasma levels of TNF-α are considered as part of systemic of inflammatory response, and is elevated plasma and synovial fluid of patient with RA (Nordahlet al., 2000).

II. Methods

Hematological study:

Bloodsamples were collected from patients with periodontitisand from healthy controls according to (Lewiset al.,2006).

Immunological study:-

Collection of serum: serum samples were collected from patients with periodontitis and from healthy individual as a control samples according to (Forbes etal., 2007). The RF was analysis with routine procedure by (RF-Latex). It is aslide agglutination test for the detection of RF in human serum (Frederick Wolf et al., 1991).Erythrocyte Sedimentation Rate is measured according to (Fischbach and Dunning,2009).Human IL- analysis by a commercially available enzyme immunoassay kit (Kupper and Graves, 1995). TNF- α analysis by a commercialy available enzyme immunoassaykit (Rudy, 1997).

Statistical Analysis:-

Statistical analysis (Mean± Standard deviation) and probability was done depending on (Daniel, 1999).

III. Results:

Distribution of periodontal patient disease depending on age can be divided in to groups for both sex starting from youngest patient who was (20) years to oldest who was (80) years. They classified in to patient and control groups. The majority of the patients with periodontitis aged from (60- 80) years old is about (46%). Table (1).

Table (1): Distribution of periodontal patient disease compared with healthy controls according to the age.

Variable	Age	No.	Total
Patients with chronic periodontitis(50)	(20-40) (40-60) (60-80)	9 18 23	(50)87.1%
Health control (20)	(20-40) (40-60) (60-80)	5 8 7	(20)28.9%
Total		70	(70)100%

The distribution of periodontal patient disease depending on the sex, results in female to male ratio 4/1 affected with periodontitis. Table (2).

Table(2):Distribution of periodontal patients and healthy control according to the sex.

Sex	No. of variable
Male	Periodontal patients.(13)26%
	Healthy control.(10)
Female	Periodontal patients.(37)74%
	Healthy control.(10)
Total	(70)

The classification of periodontal patient according to tobacco smoking for both sex results in ratio 54% (27 from 50) smoking compare with 46%(23 from 50) non- smoking. Table(3).

Table(3): Distribution of periodontal patients and healthy control according to smoking and non-smoking history.

No.	Smoking	Non smoking
Periodontal patients(50)	27(54%)	23(46%)
Control(20)	_	20

The association of periodontitis with other disease such as diabetic mellitus and hypertension disease for both sex results that is 38% (19(10male+9female)) of periodontal disease have diabetic mellitus, 30%(15(9female+6male)) have hypertension and 48%(24(16female+8male)) have both diseases with periodontitis. Table(4).

Table(4):The percentage of systemic disease in periodontal patients (Diabetic mellitus and hypertension).

Number of periodontitis patient.	Number of periodontitis and diabetic mellitus patients.	Number of periodontitis andhypertension patient.	No. of D.M, HTN and periodontitis patient.
50 (46%)	19(10male+9female) 38%	15(6male+9female)30%	24(8male+16female)48%

Both female and male with periodontitis have highly significant increase in serum rheumatoid factor when comparison with healthy control table (5). Rheumatoid factor classification in to positive (24%) and negative (76%) for patients with or without rheumatoid arthritis.

Table(5): Relationship between periodontal rheumatoid arthritis patients by (RF).

Patient with chronic periodontitis(50)	Patient with positive result for RF factor (12) 24%
	Patient with negative result for RF factor (38). 76%
Total	(50)100%

The value of Erythrocyte Sedimentation Rate for periodontal and rheumatoid arthritis patient for both sex when compare with healthy control give high significant elevation result (p>0.0001) table (6).

Table (6): The value of Erythrocyte Sedimentation Rate(mm/hr) in periodontal and Rheumatoid arthritis patients compared with healthy control.

Groups		No.	Mean± S.D	P value
Patient ESR(mm/hr)	control	12 12	47.0660±0.9520 12±1.5370	<0.0001 <0.0001

***high significance level (P<01000)**

The serum tumor necrosis factor-α in periodontal and RA patient show significant decrement (p<o0.0001) when compare with healthy control for both sex table (8).

Table(7): Differences between periodontal and rheumatoid arthritis patients with healthy control in Tumor necrosis factor-α(pg/ml) in both sex.

Variable	Groups	No.	Mean± S.D	P value
TNF-α(pg/ml)	Patient	12	21.800±1.773	<0.0001
	Control	12	11.225±0.660	<0.0001

***High significance level (P<0.0001)**

The measurement value of serum interlukine-1β for periodontal and RA patient give high significantly increase (p<0.0001) when compare with healthy control for both sex. Table(8).

Table(8): Differences between periodontal and rheumatoid arthritis patients with healthy control in Interlukine-1β (pg/ml) in both sex.

Variable	Groups	No.	Mean±S.D	P value
IL-1β(pg/ml)	Patient	12	8.283±0.867	<0.0001
	Control	12	2.433±0.342	<0.0001

***High significance level (p<0.0001)**

IV. Discussions

The majority of clinical and epidemiological studies indicate that patients with RA have increased prevalence of periodontitis and tooth loss. Several causal and non- causal pathways could explain the observed association (de Pablo et al., 2009). In present study, the age, gender (range) and tobacco smoking of individual in rheumatoid arthritis and periodontal group reflected the epidemiology of disease, i. e, more female(74%) which is consistent with findings by(Mercado et al., 2001) which (74.6%) female were effected table(2). The age range of the present study (20- 80 years) is also consistent with the study by Mercado et al., 2001) (50- 70 years), table(1).

Several host and environmental factors, such as tobacco smoking (Klareskoget al., 2007) might cause increased susceptibility to both periodontitis and RA, and thus determine a non- causal association between the two conditions. The result of table(3) In this study demonstrated the distribution of periodontal disease among smoking patients(54%) compare with non- smoking (46%), this agree with (de Pablo et al., 2009) who reported

that there are several important shared features in the pathogenesis of periodontitis and RA that make the existence of other common susceptibility factors likely.

Table(4) clarify the association between periodontitis and other systemic disease. The result of table (4) was gave about (38%) of periodontal patients have diabetic mellitus, (30%) have hypertension and (48%) have both with periodontitis this agree with (de Pablo et al., 2009) who reported that many periodontitis disease is associated with other systemic conditions such as diabetic mellitus and cardiovascular disease. While result of table (5) was gave (24%)of periodontal disease have rheumatoid arthritis this is in accordance with that reported by (Mercado et al., 2001) (62.5%). The diagnosis of RA is based on clinical history, blood count (ESR) and rheumatoid factor(RF). Periodontal patients carries a unique risk for development of autoimmune antibodies associated with RA Patients with RA have either lost many teeth or usually have severe periodontitis .(Persson, 2012).

The Erythrocyte sedimentation rate result in Periodontal and RA patients was statistically and significantly higher ($P<0.0001$) compare with NRA group table (6). This indicated that periodontitis could contribute to severity of arthritis by lowering hemoglobin and increasing ESR and this is agree in conformity with (Ranade and Doiphon, 2012).

The proinflammatory cytokine interlukine- 1β (IL-1β) andtumor necrosis factor- α(TNF- α) is highly increased in periodontal RA patients when compared with healthy control once (table 7, 8) this result is accepted with (Persson, 2012) who reported that TNF-α and IL- 1β regulate a cascade of inflammatory events in both RA and periodontitis.

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