Association between periodontitis and acquired coronary heart disease

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ABSTRACT

Background: The association between oral microbial infection and systemic disease is not a new concept. A major confounding issue is that oral infections often are only one of the many important factors that can influence systemic diseases .

Objective: This study was conducted to evaluate the periodontal health status of patients with acquired coronary heart disease.

Type of the study: Cross-sectional study.

Methods: The study group consisted of 200 patients with an age range (35-70) years, having coronary heart disease .This study group were compared to a control group of non-coronary heart disease (200 individuals) matching with age and gender. The oral parameters were examined including the periodontal conditions, assessment of periodontal loss of attachment as well as the missing teeth according to the World Health Organization criteria (1997)[1] were followed for diagnosis and recording of oral health status.

Results: Results of the present study show that the total sample had a highly significantly periodontitis and periodontal loss of attachment with advancing age ($P<0.01$), while no statistically significant differences were found between males and females with periodontal condition and periodontal loss of attachment among the study group ($P>0.05$), while there was significant differences among their corresponding controls ($P<0.05$). High percentage of missing teeth was recorded among the study and control group. Highly statistically significant differences were seen between study and control groups in all the degree of severity related with periodontal conditions ($P<0.001$).

Conclusion: The data showed that 50.5%, 35% of the control group were with periodontal disease and loss of attachment respectively compared with study group 68%, 58.5% . The present study indicate that periodontal disease more common among patients with CHD than among controls matched for age and gender. Thus the possibility that chronic oral infection or similar factor may he positively associated with CHD at least in form patients susceptible to CHD.

Although causality cannot be inferred from the present data, the observed association between chronic oral infections and CHD for susceptible patients in the present study can not be excluded.

Keywords: Periodontitis, Coronary heart disease.

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Although we are in the 21 century, but still coronary heart disease is the leading cause of adult mortality and morbidity throughout the world [2]. Cardiovascular disease, the number 1 cause of death every year in the last century [3]. Recent data suggest that chronic infection may play an important role in the development of atherosis, this concept arises from the fact that traditional risk factors for atherosclerosis and consequent coronary artery disease such as hypertension, hypercholesteremia, diabetes mellitus, and smoking, don't account for all of the atherosclerosis found in a large proportion of the population [4].

The association between oral microbial infection and systemic disease is not a new concept. A major confounding issue is that oral infections often are only one of the many important factors that can influence systemic diseases [5]. One of the most common and often undiagnosed diseases of human is periodontitis, this is a chronic infection of the supporting tissues of the teeth. It is estimated that about 15% of adults from 21 to 50 years and about 30% of subjects >50 years have ever periodontitis [6]. In Baghdad city using Community Periodontal Index Treatment Need (CPITN) index showed 91.7 % of the sample had signs of periodontal disease and the periodontal condition worsen with age and the majority of subjects were with poor oral hygiene [7]. The hypothesis being that periodontal pockets could release pro-inflammatory bacterial components for instance endotoxins, into the blood stream. It is known that, the oral cavity can be source of circulating bacteria, but this has never been shown for bacterial endotoxins and no evidence exists so for the risk of systemic injury is related to the severity of periodontitis [8&9]. Mastication – induced increase of endotoxemia was found in healthy individuals and periodontitis patients, the level of endotoxemia being significantly higher in this latter group. This provides evidence that the periodontal pockets themselves can be a chronic source of passage of proinflammatory bacterial components in the blood stream, thus supporting the hypothesis that periodontal disease could play a causative role in the development of systemic pathologies [9]. The unsupported and unattached soft tissues of diseased periodontium undergo high level of rubbing of pressure, or of depression that can create a (pumping effect) favoring the passage of component of the subgingival biofilm through the thin and often ulcerated epithelium of

periodontal pockets [10]. This could be the missing link explaining that abnormally high plasma levels of some inflammatory markers can be observed in periodontal disease and that periodontal disease has been found by some authors to be statistically associated with the risk of occurrence of some systemic disease such as coronary heart disease [9&10].

Importantly, based on cross-sectional an prospective epidemiological studies, periodontitis has been linked to cardiovascular diseases and cerebrovascular ischemia, although mechanisms responsible for this association are obscure. Patients with periodontal disease are an average at twice the risk for coronary vascular diseases [11]. The increased risk for systemic disease in subjects with periodontal disease may be due to increased prevalence and severity of bacteremia with oral microorganisms [9&11].

Several microorganisms including Chlamydia pneumonia and Cytomegalovirus have been implicated in the infectious etiology of atherosclerosis However, it is not possible to say whether Chlamydia Pneumonia infection is the initial factor in the early phase of atherosclerosis or whether Chlamydia Pneumonia is a common secondary infectious complication in atheroma plaques. In both cases, it may be an important factor in the development of atherosclerotic diseases. The age when the person develops the primary infection of Chlamydia Pneumonia infection and smoking are probably important factors together with genetic factors to determine whether Chlamydia Pneumonia infection remains chronic in some individuals and not in others.[12&13].

Eradication of cardiovascular disease may not be a realistic goal in the near future, but increase efforts in educating, screening and improving treatment can influence the morbidity and mortality associated with these diseases. Identifying people at high risk of developing coronary heart disease is paramount to preventing initial coronary events. Secondary prevention, defined as preventing recurrent coronary events in patients with coronary heart disease. Oral health care providers are in a position to aid with both screening for and monitoring of risk factors associated with cardiovascular disease, as well as to facilitate and provide patient education.

Mmethods: A total of 400 individual, 200 with acquired coronary heart disease and 200 without, were included in this study with an age range 30-70 years attending to the department of cardiology (Ibn Al Betar Hospital) / Baghdad, complaining from chest pain.

Clinical examination: All patients were clinically examined and completed a medical questionnaire in Ibn Al Betar Hospital. The oral examination of the study groups done after 24-48 hour after Cardiac Catheterization, while that of the control groups done when the patients attending the Non – Invasive department of Ibn Al Betar Hospital. In this department are routinely perform all the clinical examinations for the patients to exclude any coronary heart diseases. These include:

Treadmill exercises, ECG, Echo, Holter, Sonar, Pulmonary test

The complete oral examination and questionnaires forms were done for control groups in non- invasive department, and study groups in Cardiac Department of hospital, after the approval of the management of hospital was obtained.

The oral examination included:

Periodontal health status:-

By using community periodontal index (CPI). Three indicators of periodontal status were used for this assessment, gingival bleeding, dental calculus, and periodontal pockets (WHO, 1997).[1]

Loss of attachment:-

Was examined and recording immediately after recording (CPI) according to WHO, 1997.[1]

Missing teeth:

Teeth missing due to caries, periodontal disease or others were recorded as missing. The retained root, badly caries non restorable teeth which were indicated for extraction were not considered as missing teeth, only the extracted and missing were recorded [1].

Results

The distribution of the study and control groups stratified by age and gender shows in the Table 1 & 2

Table 1: Distribution of the Study and Control Groups Stratified by Age and Gender

Age (year)	Gender	Study group		Control group	
		No.	%	No.	%
35-49	M	77	38.5	78	39.0
	F	23	11.5	27	13.5
	T	100	50.0	105	52.5
50-70	M	75	37.5	67	33.5
	F	25	12.5	28	14.0
	T	100	50.0	95	47.5

Table 2:- Mean and Standard Deviasion of Total Sample Stratified by Age and Gender

Study group				
Age (year)	**Gender**	**No.**	**Mean**	**SD**
35-49	M	77	41.75	4.94
	F	23	39.69	4.44
50-70	M	75	56.76	6.49
	F	25	57.08	5.64
Control group				
35-49	M	78	41.06	4.36
	F	27	41.92	3.33
50-70	M	67	58.59	6.07
	F	28	54.60	5.12
Total	Total M&F	400	49.00	9.57

Oral parameter index among the study and control groups:

Periodontal condition:

Table 3 illustrates the periodontal condition of the total sample according to the categories of CPI. This table clearly shows that highly significant difference was noticed between the values of study and control groups, in score 0 (healthy periodontal condition), 32.0% and 49.5% respectively. Similar findings were observed when compared with the total males and total females in the study group to their corresponding controls.

In both CHD patients and non CHD subjects pathological periodontal pockets was found to increase with the increase of age to reach the highest value at 50-70 years. This difference was found to be statistically highly significant ($p<0.01$). Assessment of periodontal condition showed that dental calculus to be the predominant disorder in CHD patients 40.5% while only 15.0% in their corresponding controls.

Followed by gingival bleeding to be 11.0% among the study group while 6.0% in their control. The pathological periodontal pockets ≥ 6 mm showed no difference between the study and control groups. Only the periodontal pocket 4-5 mm 8.5% among the control group which more than study group 19.5%.

Table 3:- Distribution of the Total Sample According to the Periodontal Conditions Stratified by Age and Gender

Periodontal Conditions											
Study group											
		Healthy		Bleeding		Calculus		pcket 4 -5mm		ket ≥ 6 mm	
Age		No.	%	o.	%	No.	%	No.	%	No.	%
35-49	M	34	20.9	9	26.5	25	22.5	7	8.5	2	20
	F	10	6.1	3	8.8	10	9	0	0.0	0	0.0
	T	44	27	2	35.3	35	31.5	7	8.5	2	20
50-70	M	15	9.2	6	17.6	33	29.7	18	22	3	30
	F	5	3.1	4	11.8	13	11.7	3	3.7	0	0.0
	T	20	12.3	0	29.4	46	41.4	21	25.7	3	30
Control group											
35-49	M	42	25.8	9	26.5	11	9.9	13	15.9	3	30
	F	21	12.9	3	8.8	3	2.7	0	0.0	0	0.0
	T	63	38.7	2	35.3	14	12.6	13	15.9	3	30
50-70	M	23	14.1	0	0.0	8	7.2	34	41.5	2	20
	F	13	8	0	0.0	8	7.2	7	8.5	0	0.0
	T	36	22	0	0.0	16	14.4	4	50	2	20

P< 0.01 x^2 = 116.547 df=28

Loss of attachment:-

In both CHD and non CHD subjects, loss of attachment increased with age. CHD subjects showed a significantly higher loss of attachment over non CHD group at age 35-49 and 50-70. At age group 35-49 years, the percentage of sites showed 0-3 mm loss of attachment in CHD and non CHD group were 27.2% and 38.5% respectively, while percentage of sites showed 4-6 mm loss of attachment were 25.7% and 12.4%.

CHD group showed 6-8 mm loss of attachment were 20.4% also higher than non CHD individuals 11.1%, while loss of attachment 9-11 mm, CHD group show 10.5%, non CHD group 15.8%, loss of attachment ≥ 12 mm observed only on non CHD group and for one subject. At age group 50-70 years only 11.8% of total sites showed 0-3 mm loss of attachment among the CHD group, while non CHD group showed more sites with 0-3 mm loss of attachment 22.6%. CHD patients had 38.9% of their sites with loss of attachment 4-5 mm , while non CHD group had 23.0% CHD patient showed more sites with loss of attachment 6-8 mm and 9-11 mm than corresponding their control. For the total groups, high statistically significant difference in loss of attachment between study and control groups in relation to the age and gender (p<0.01). The pattern of loss of attachment in CHD and non CHD subjects in all age groups was shown in Table 4.

Table 4:- The Pattern of Loss of Attachment Within Total Sample Stratified by Age and Gender.

Loss of attachment (LA)											
Study group											
Age		0-3 mm (LA)		4-5 mm (LA)		6-8 mm (LA)		9-11 mm (LA)		≥12 (LA)	
		N	%	N	%	N.	%	N	%	N	%
35-49	M	41	19.2	23	20.4	11	20.4	2	10.5	0	0.0
	F	17	8	6	5.3	0	0.0	0	0.0	0	0.0
	T	58	27.2	29	25.7	11	20.4	2	10.5	0	0.0
50-70	M	17	8	34	30.1	17	31.5	7	36.8	0	0.0
	F	8	3.8	10	8.8	5	9.3	2	10.5	0	0.0
	T	25	11.8	44	39.9	22	40.8	9	46.3	0	0.0
Control group											
35-49	M	55	25.8	14	12.4	6	11.1	3	15.8	0	0.0
	F	27	12.7	0	0.0	0	0.0	0	0.0	0	0.0
	T	82	38.5	14	12.4	6	11.1	3	15.8	0	0.0
50-70	M	27	12.7	19	16.8	15	27.8	5	26.3	1	100.
	F	21	9.9	7	6.2	0	0.0	0	0.0	0	0.0
	T	48	22.6	26	23	15	27.8	5	26.3	1	100.

P< 0.01 x^2 = 93.281 df=28

Missing Teeth (MT):-

For the missing teeth (MT) mean value, among the study and control group , the lowest values were found to increase with the increase of age to reach the highest at 50-70 years. Concerning sex variation, differences were found between the two sexes in the mean of MT value (p>0.05) within the study group. While differences were found between the two sexes in the mean of MT value within the control group (p<0.05).

Dentate subjects with CHD with score 0 (no MT) and 1-3 MT 17.0% and 31.5% was differ from corresponding controls (15.5% and 26.0% respectively). A statistically highly significant difference were recorded between mean values of MT between different groups. In score 4-7, 8-11 and > 12 MT, dentate subjects without CHD had more percentage of MT than CHD patients. The distribution of TM among total sample in age and gender was shown in Table 5 &6.

Table 5:- Numbers and Percentages of Subjects with Missing Teeth (MT) Stratified by Age and Gender

Missing Teeth (age)								
Study group								
	0-3 MT		4-7 MT		8-11 MT		≥12	
Age	N	%	N	%	N	%	N	%
35-49	63	63	31	31	5	5	1	1
50-70	34	34	42	42	21	21	3	3
Total	97	48.	*73*	36	26	13.	4	2
Control group								
35-49	59	56.	36	34.	6	5.7	4	3.8
50-70	24	25.	46	48.	22	23.	3	3.2
Total	83	41.	82	41	28	14	7	3.5
Missing Teeth (gender)								
Study group								
	0-3 MT		4-7 MT		8-11 MT		≥12	
SEX	No	%	No	%	No	%	N	%
M	74	48.	56	36.	18	11.	4	2.6
F	23	47.	17	35.	8	16.	0	0.0
Total	97	48.	73	36.	26	13.	4	2.0
Control group								
M	49	33.	61	42.	28	19.	7	4.8
F	34	61.	21	38.	0	0.0	0	0.0
Total	83	41.	82	41	28	14	7	3.5

Table 6:- Missing Teeth (Mean ± SD) Among the Total Sample Stratified by Age and Gender

Missing Teeth (MT)					
Study group					
Age	Sex	No.	%	Mean	SD
35-49	M	77	38.5	3.05	2.72
	F	23	11.5	3.26	2.84
50-70	M	75	37.5	5.14	3.43
	F	25	12.5	5.24	3.00
Control group					
35-49	M	78	39	3.55	3.52
	F	27	13.5	2.25	1.87
50-70	M	67	33.5	6.92	3.13
	F	28	14	3.28	2.44
Total		400	100	4.30	3.37

H.S. (P<0.001) F value = 13.210 df=7

Total sample in relation to common risk factors for acquired coronary heart disease

The distribution of the sample according to the type of systemic diseases shows in Table 7. Blood hypertension affected 37% of the total study group, and 12.5% had diabetes mellitus. No significant difference showed between age and systemic diseases within study group (p>0.05). Among the control group, blood hypertension and diabetes mellitus affected 39.5% and 9% respectively. The findings showed high significant difference between age and blood hypertension (p<0.001) in control and no statistically significant in study group, while no significant difference with diabetes mellitus in control and study group. Also the findings were observed when comparing the total males and total females in relation to both systemic diseases, no significant differences in study group while high significant in control group. Among the study group 7% had both diseases while only 3.5% in their corresponding controls.

Table 7:- Distribution of the Total Sample According to the Systemic Diseases.

Systemic Diseases								
	Study group				Control group			
	No	%	Sig	X^2	No	%	Sig	X^2
BP	74	37	NS	0.590	79	39.5	HS	5.554
M	54	73	NS		50	63.3	HS	
F	20	27	NS		29	36.7	HS	
DM	25	12	NS	1.003	18	9	NS	7.503
M	21	84	NS		18	100	HS	
F	4	16	NS		0	0	HS	
BP& DM	14	7.	S		7	3.5	HS	

HS= highly sig NS= non sig S=sig

Discussion

The present study is the first Iraqi endeavor to deal with the oral health problems as a risk factor among the Iraqi acquired coronary heart diseases patients, compared with non- coronary heart diseases individuals. The basic method of oral health surveys of WHO (1997) was utilized in the present study as a guide in the diagnosis and quantification of oral conditions, also it can give a standard measurement of oral disease, for planning and evaluating of oral health programs.

Oral health status

The present study have shown that patients with acquired CHD had worse oral health status than controls matched for age and gender with agree with Humphrey et al, (2008) [14]. Despite the variations existing between the present study and other studies in the CHD under study, age group and methodologies used to measure oral diseases, in addition to different environmental factors which makes it difficult to cross match the results,

comparisons were made with closest figures in these studies that could be used to draw conclusions. Several possible explanations for this should be considered. First, dental disease and CHD share several common etiological factors for example, low socioeconomic state, smoking, diabetes [15]. Second, subjects who take care of their dentition may also be concerned about other aspects of their health, in lauding a lifestyle conductive to CHD. The relation between oral health and CHD, however, remain significant even after adjustment for age social class, hypertension, serum lipid and lipoprotein concentration, smoking and presence of diabetes [16]. During the review of literatures on the relationship between acquired CHD and periodontal condition it becomes clear that many studies demonstrated obvious deficiencies as to experimental design and performance. The periodontal conditions of the total sample according to the categories of CPI in the present study shows high significant difference between CHD patients and non- CHD subjects in relation to age and gender. High prevalence of periodontal disease (gingival bleeding, calculus and pathological pockets) in the present study among CHD patients compared with corresponding control group. It has been observed in the current study among the elderly CHD patients were similar to that reported by other studies [17&18]. Concluding that the more severe the periodontitis, the higher the risk of heart attack. Following decades of intensive research it is now unequivocally acknowledged that poor oral hygiene has profound negative effects on systemic health [19].

The moderate and severe periodontitis of CHD patients in the present study may be related to plaque and calculus accumulation, these findings explain that periodontitis has been traditionally regarded as a chronic inflammatory oral infection and release of pro-inflammatory factors [20]. The possibility that elevated serum factors in these patients in the present study may be due to many factors, on the other hand, it is conceivable that systemic exposure to low dose endotoxin, with elevated antibody against Parphyromonans Gingivalis LPS (lipopolysuccaride), or other gram negative periodontal pathogen, could be responsible, in part, for the hyperlipidemia, even low level chronic exposure to gram- negative microorganisms and / or their LPS can manifest a similar response [21]. These organisms can invade deep connective tissues vascular endothelium associated with the periodontium, can be found within vascular pathological plaques, and can elicit a circulating antibody response [20].The potential exists for chronic low - level systemic exposure to microorganisms / LPS leading to generalized alterations in lipid metabolism [20&21].

Although causality cannot be inferred from the present data, the observed association between chronic oral infections and CHD for susceptible patients in the present study can not be excluded. In most cases, early recognition and intervention may have prevented or significantly delayed the onset of the disease, which may have resulted in decreased mortality and morbidity.

References

1. World Health Organization (1997): Oral Health Surveys, basic methods. 4th ed World Health Organization, Geneva, Switzerland.
2. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics–2015 update: a report from the American Heart Association. *Circulation*. 2015;131:e29-322
3. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. *Circulation*. 2011;123:933-44. Epub 2011 Jan 24.
4. CDC, NCHS. Underlying Cause of Death 1999-2013, released 2015. Data are from the Multiple Cause of Death Files, 1999-2013, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program. Accessed Feb. 3, 2015.
5. Seymour GJ, Ford PJ, Cullinan MP, Leishman S and Yamazaki K. Relationship between periodontal infections and systemic disease. *Clin Microbiol Infect.* 2007; 13 Suppl 4:3-10.
6. Pizzo G, Guiglia R, Lo Russo L and Campisi G. Dentistry and internal medicine: from the focal infection theory to the periodontal medicine concept. *Eur J Intern Med.* 2010; 21:496-502
7. Al-Waheb, AM (1998): Tooth loss in the permanent dentition of adult and elderly in Baghdad City. Jordan Dental, J, 13:23-27.
8. Friedewald VE, Kornman KS, Beck JD, Genco R, Goldfine A, Libby P, Offenbacher S, Ridker PM, Van Dyke TE and Roberts WC. The American Journal of Cardiology and Journal of Periodontology Editors' Consensus: periodontitis and atherosclerotic cardiovascular disease. *Am J Cardiol.* 2009; 104:59-68.
9. Nakano K, Nemoto H, Nomura R, Inaba H, Yoshioka H, Taniguchi K, Amano A and Ooshima T. Detection of oral bacteria in cardiovascular specimens. *Oral Microbiol Immunol.* 2009; 24:64-8.
10. Chen YW, Nagasawa T, Wara-Aswapati N, Ushida Y, Wang D, Takeuchi Y, Kobayashi H, Umeda M, Inoue Y, Iwai T, Ishikawa I and Izumi Y. Association between periodontitis and anti-cardiolipin antibodies in Buerger disease. *J Clin Periodontol.* 2009; 36:830-5.
11. Seymour GJ, Ford PJ, Cullinan MP, Leishman S, West MJ and Yamazaki K. Infection or inflammation: the link between periodontal and cardiovascular diseases. *Future Cardiol.* 2009; 5:5-9.
12. Iwai T. Periodontal bacteremia and various vascular diseases. *J Periodontal Res.* 2009; 44:689-94.
13. Toyofuku T, Inoue Y, Kurihara N, Kudo T, Jibiki M, Sugano N, Umeda M and Izumi Y. Differential detection rate of periodontopathic bacteria in atherosclerosis. *Surg Today.* 2011; 41:1395-400.
14. Humphrey LL, Fu R, Buckley DI, Freeman M and Helfand M. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. *J Gen Intern Med.* 2008; 23:2079-86.
15. Jones JA, Miller DR, Wehler CJ, Rich SE, Krall-Kaye EA, McCoy LC, Christiansen CL, Rothendler JA and Garcia RI. Does periodontal care improve glycemic control? The Department of Veterans Affairs Dental Diabetes Study. *J Clin Periodontol.* 2007; 34:46-52.
16. Linden GJ, Lyons A and Scannapieco FA. Periodontal systemic associations: review of the evidence. *J Periodontol.* 2013; 84:S8-S19.
17. Paraskevas S, Huizinga JD and Loos BG. A systematic review and meta-analyses on C-reactive protein in relation to periodontitis. *J Clin Periodontol.* 2008; 35:277-90.

18. Tonetti MS and Van Dyke TE. Periodontitis and atherosclerotic cardiovascular disease: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. *J Periodontol.* 2013;84:S24-9.
19. Seymour GJ, Ford PJ, Cullinan MP, Leishman S and Yamazaki K. Relationship between periodontal infections and systemic disease. *Clin Microbiol Infect.* 2007; 13 Suppl 4:3-10.
20. Loos BG. Systemic markers of inflammation in periodontitis. *J Periodontol.* 2005; 76:2106-15.
21. Li X, Iwai T, Nakamura H, Inoue Y, Chen Y, Umeda M and Suzuki H. An ultrastructural study of Porphyromonas gingivalis-induced platelet aggregation. *Thromb Res.* 2008; 122:810-9.