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A Case Control Study to Investigate the Association between Periodontal Disease and Coronary Artery Disease

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Abstract:-

➢ ***Background***

Periodontitis and cardiovascular disease share complex etiology involving common risk factors like age, smoking, diabetes etc. recent evidence suggested that dental infections could play a role in the initiation and development of cad and has led the world to focus on the fact that, periodontitis may act as an independent risk factor for initiation and progression of cad.

➢ ***Material and Methods***

In a Case Control study a total of 120 subjects the volunteers were divided in two groups, the first composed by individuals CAD as cases (n = 60) and the second by individuals without CAD as Controls (n = 60). Variables recorded include age, gender, status on hypertension, diabetes, smoking status, alcohol consumption, body mass index, lipid profile, total leukocyte count and socioeconomic data. presence of Periodontal diseases was assessed using Russel's periodontal index. Clinical pocket depth, clinical attachment levels. Percentage sites with Bleeding on Probing (BOP) and Periodontal Pocket Bleeding Index (PPBI) were used to calculate the percentage sites with bleeding on probing. Blood samples in both groups were analyzed for Lipid Profile, Complete blood count, High sensitivity C - Reactive Protein and plasma fibrinogen levels.

➢ ***Results***

Diabetes, Hypertension, Smoking and Alcohol consumption status was statistically significant in individuals with CAD. (P= 0.000), Lipid Profile, Hs-CRP and Serum Fibrinogen Levels were statistically significant in cases. The Periodontal status iindicated mean number of teeth present in controls was higher and the mean CAL levels in cases was 5.83 ± 1.2 mm as compared to 2.45 ± 0.7 mm in controls. The mean number of teeth with CAL > 3mm in cases was 12.6 ± 4.3 and in controls were 5.1 ± 2.5. The mean percentage of the sites with bleeding on probing was assessed and the number in cases were 40.17± 9.06 as compared to 22.25±14.77 in controls. Means of Russel's periodontal index scores of cases were high 3.1 ± 0.4 Vs 1.9 ± 0.9 in controls. The number of teeth with CAL >3mm showed a significant result. ('p'value =0.012), the number of missing teeth showed a statistical significant result ('p' value 0.021). The biochemical variables showed statistical significant results. 'p' value for CRP, Fibrinogen and TLC were 0.019, 0.023, and 0.001 respectively. The odds ratio was elevated with 2 times for the TLC Count, 2 times for CRP and less than two for number of missing teeth and number of teeth with CAL >3mm.

➢ ***Conclusion***

There is increasing evidence that dental infection, especially periodontal pathogens have capability of aggregation of platelets, intitation and maturation of ateromatous plaques are the most important causes. Dental infection may be an independent risk factor for CAD. The outcome of the present study strengths the association between poor periodontal health and CAD.

Keywords:- *CAD - Coronary Artery Disease, PD - Periodontal Disease, BOP- Bleeding on Probing, Atherosclerosis, Inflammation.*

I. INTRODUCTION

Periodontal disease(PD) is an infectious disease resulting in inflammation of the supporting periodontal tissues resulting in attachment loss, bone destruction and is characterized by the formation of a pocket or recession of the gingiva. The disease is usually linked to presence of local factors, plaque and calculus. The loss of attachment occurs slowly, but rapid progression can be seen with variable microbial pattern [1]. In past, much attention had gained to assess the possibility of oral infections namely PD may influence the onset or progression of systemic diseases including CAD and studies have already demonstrated that periodontal disease casuse changes in systemic health which alters blood serum inflammatory Markers, proteins and lipids levels. [2] The world's leading cause of adult mortality and morbidity is CHD. There are environmental and Genetic risk factors for Development of CHD. These factors may alone or in combination are involved in initiation and progression. Studies relate

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elevated levels of CRP, IL-6, leukocytosis and dyslipidemia that occurs in patients with cardiovascular diseases may be due to chronic infectious diseases and inflammatory processes that may result from periodontal infections.[3, 4]

Periodontal infections persists in a majority of population without being diagnosed. Research indicates that PD have elevated levels of lipids, serum CRP, fibrinogen, IL-1 and IL-6, indicating that systemic inflammatory response is seen with PD in otherwise healthy patients. [4] Evidence suggest the microbiota of periodontal disease (PD) invade the walls of blood vessels and initiates the plaques, and their DNA isolated in coronary plaques. [5] studies suggest that the risk of developing CAD is almost twice in patients with PD and some studies demonstrate a beneficial effect of periodontal therapy on endothelial function. Since periodontal diseases and heart diseases are common, quantifying their association is of significant public health importance. The interpretation of the reported associations is difficult. On one hand, the associations could be interpreted as causal. On the other hand, these data could be interpreted as being artifacts, that is, the result of bias caused by confounding factors. PD and CHD share common risk factors, such as increasing age, smoking, stress, socioeconomic status, and body mass index.[6] The potential for confounding is substantial. Incomplete adjustment of these factors may be responsible for the observed weak association. However, both PD and atherosclerosis are multifunctional and share common established risk factors like smoking and diabetes. Thus, the nature of true association is a matter of concern. Keeping in mind the nature of association, we desired to investigate the possible association between periodontal health and CAD by evaluating periodontal status, serum CRP, fibrinogen levels, lipid profile and leukocyte counts in subjects with established CAD as cases and subjects without CAD as Controls.

II. MATERIALS AND METHODS

A case-control design was chosen to include a total of 120 patients, 60 patients with Acute Myocardial Infarction (AMI) as cases and 60 patients without Coronary Heart Disease as controls as matched by age and sex.

Cases were those patients diagnosed with acute coronary syndrome (ACS) in the age group of 30-60 Yrs with changes in the electrocardiogram and elevation of cardiac biomarkers with at least 50% of teeth in the mouth and had not received any dental therapy in the previous 6 months. The excusion included history of Infective endocarditis, CHD, Valve replacement, prosthetic Joints, IDDM, patients on Immunosuppressants and chemotherapy. Controls were subjects without evidence of coronary heart disease, as verified by history and examination. Potential subjects were screened for inclusion and exclusion criteria. Subjects who consented were included.

Variables recorded include age, gender, status on hypertension, diabetes, smoking status, alcohol consumption, body mass index, lipid profile, total leukocyte count and socioeconomic data. These variables were obtained from medical records and interview.

Independent variable for presence of Periodontal diseases was assessed using Russel's periodontal index. Clinical assessment of PD were based on pocket depth, clinical attachment levels. Percentage sites with Bleeding on Probing (BOP) and Periodontal Pocket Bleeding Index (PPBI) were used to calculate the percentage sites with bleeding on probing. Collected Blood samples in cases and controls were analyzed for Lipid Profile, Complete blood count, Hs-CRP and plasma fibrinogen.

III. STATISTICAL ANALYSIS

The data collected was tabulated and was subjected to statistical analysis. Statistical analysis was performed by using the SPSS software (Version 16) and the statistical significance was defined as $P < 0.05$

1. Mean
2. Standard Deviation:
3. Student's 't – test'
4. Pearson Chi-Square Test
5. Univariate and Multivariate analysis
5. Logistic regression analysis to draw the association.

IV. RESULTS

Table 1 and 2 shows the means and proportions of the background characteristics including clinical parameters and major risk factors of both groups. There was no significant difference in Age, gender distribution, Educational status and Socioeconomic status in the two groups. The mean body mass index and showed that Body mass index was higher in cases when performed which showed statistical significant difference ('p' value = 0.000) Diabetes, Hypertension, Smoking and Alcohol consumption status were found to be statistically significant in cases when compared with controls.

The cases constituted of a mean serum CRP levels of 3.53 ± 1.47 mg/L as compared to 2.70 ± 0.77mg/L. The mean CRP levels were higher in cases. The cases constituted of a mean serum plasma fibrinogen levels of 392.23 ± 35.34 mg/dl as compared to 293.07 ± 29.87 mg/dl in controls. The mean Fibrinogen levels were higher in cases.

The cases constituted of a mean TLC counts of 8972.90 ± 23.39 mm3 of blood as compared to 5484 ± 583.7 in controls. The mean number of teeth present in cases was 24.8 ±3.5 as compared to 27.3 ± 1.5. The mean number of teeth present in controls was higher than that compared to cases. The mean CAL levels in cases was 5.83 ± 1.2 mm as compared to 2.45 ± 0.7 mm in controls. The mean CAL in cases was higher than that compared to controls. The mean number of teeth with CAL > 3mm in cases was 12.6 ± 4.3 and in controls were 5.1 ± 2.5. The

mean percentage of the sites with bleeding on probing was assessed and the number in cases were 40.17± 9.066 as compared to 22.25±14.77 in controls. On assessing the Russel's periodontal index scores the means of cases had a score of 3.1 ± 0.4 as compared to that of 1.9 ± 0.9 in controls. The severity of PD in terms of mild moderate and severe were established in both the groups and when tested with marginal homogeneity test, there was a statistical significant association between the severity of PD in cases as compared to controls. ('p'value = 0.001 in cases and 0.063 in controls) Correlating the number of teeth with CAL >3mm and other parameters by statistical analysis with levels of Fibrinogen, CRP, TLC Cholesterol, HDL and LDL levels. The statistical significant results were seen in for the levels of Fibrinogen ('p' value = 0.000), CRP ('p' value = 0.001), TLC ('p' value = 0.048) and LDL levels ('p' value = 0.001). The levels of the Fibrinogen, CRP, TLC and LDL were showing an linear elevation when compared in respect to the number of teeth with CAL > 3 mm.

The common risk factors were evaluated Univariate and Multivariate analysis [Table -3] and odds of gingivitis, smoking, PD and Diabetes showed a significant result in both analysis.

When analyzed by the logistic regression model by dependable and independent variables, (Table - 4) the results showed that the percentage of bleeding sites showed no significant results ('p' value = 0.16). The number of teeth with CAL >3mm showed a significant result. ('p'value =0.012), the number of missing teeth showed a statistical significant result ('p' value 0.021). The biochemical variables showed statistical significant results. 'p' value for CRP, Fibrinogen and TLC were 0.019, 0.023, and 0.001 respectively. The odds ratio was elevated with 2 times for the TLC Count, 2 times for CRP and less than two for number of missing teeth and number of teeth with CAL >3mm. the logistic regression model was calculated without adjusting for the confounding factors.

V. DISCUSSION

Periodontal diseases are bacterial infections which results in development of the inflammatory process. Studies have shown that infection and inflammation caused by periodontal disease increases the risk of CHD [7]. Chronic bacterial and viral infections have been hypothesized to induce the initiation and progression of inflammation in the vessel wall [8]. Periodontal pathogens have shown to increase platelet aggregation and have been identified on atheromas, which supports the etiological role.

In this study, the personal characters considered were age, sex, marital status, educational status and employment status. All of these can independently serve as potential risk factors for CHD [9] however; we found no significant difference in these characters between the cases and controls. In the present study, the prevalence of diabetes was significantly higher in the cases [58.2%] than the controls [17%] however, that of hypertension was found to be similar between the two groups. Moreover, both diabetes and hypertension were found to be significantly associated with CAD. Smoking has been estimated to cause dose dependent cardiac damage and lead to CHD and with its potential pathogenic properties, smoking is another major risk factor for both cardiovascular and periodontal disease. [10, 11]

In our study, CAD patients who smoked were more (41.7%) as compared to of the controls patients (26.7 %). This difference between the groups was statistically significant. This observation was consistent with the study performed by Matilla et al [12]. The mean body index was calculated and found that the patients had a mean BMI of 26.2 as compared to 22.6 in controls. The marginal elevation of BMI in cases is noted and was statistically significant.

The numbers of teeth present were more in controls when compared to cases indicating that the patients with CHD had more number of missing teeth. Results of study corroborate with that of Loesche [13] and Gulnur [14] who calculated the dentate state and found significant association between missing teeth and CHD in cross sectional studies. Joshipura et al [15] considers the above association was due to bias and confounding factors. In the present study, an association between poor periodontal health and AMI was found. The numbers of teeth with probing depth > 3mm were found to be significantly higher in the CHD group. Multivariate logistic regression analysis confirmed that this parameter was significantly correlated with AMI when other risk factors were adjusted. A similar observation was noted by many Arbes et al [16] and Beck et al [17]. We noted an observation that number of teeth with CAL>3mm recorded the highest correlation with AMI with the odds ratio (OR) of 1.5 as recorded in the analysis.

PD creates an ulceration in pocket epithelium by which oral microorganisms and toxins gain systemic access. The area of ulcers measures an average of 72 cm2 in severe PD [18]. The mean number of teeth with CAL >3 mm were higher in cases as compared to controls. Mean clinical attachment loss and the number of teeth with CAL >3 mm were higher and significant in cases as compared to controls.

Clinical Attachment Loss (CAL) and Bleeding on probing (BOP) were similar to that in the studies by Persson et al [19]. PD and CAL were recorded in order to quantify extent and severity of periodontal sites, as done by Beck and his associates considered BOP as likely to be the best clinical indicator of current active inflammation [20,21].

The mean percentages of sites exhibiting bleeding on probing were 40.1% in cases as compared to 22.5% in controls. The difference was statistically significant and indicates a positive association between inflammation and atherosclerosis. We found that gingival bleeding was significantly higher in the cases group when compared with

the control group. This was possibly due to poor oral hygiene and gingival inflammation in the AMI group. It was further found that even after adjusting for other risk factors in multivariate logistic regression analysis, gingival bleeding was found to be associated with CHD. Gingival bleeding is one of the definite risk indicators for periodontal disease progression, hence we believe that this is one of the most important positive finding noted.

The observed mean total leukocyte count in severe disease was 8322/mm3 which is comparable with that observed by Kweider et al, [22] of (8700/mm3). The observed mean values in moderate disease (6137 / mm3) and controls (5176 /mm3) were similarly comparable to that of Loos [23]. Leukocyte counts of 8400/ mm3 have a risk ratio of 1.4 as compared to 5600/ mm3. Moderate leukocytosis has also been observed in PD [23]. Serum lipids are traditional risk markers for cardiovascular disease and are determined by a number of factors. Bruno Loos et al, stated that chronic endotoxinemia in periodontal disease alters the lipid metabolism by cytokine induced changes [23].

In the present study, cholesterol levels were found to be elevated in cases. Chronic exposure even of a low level to gram negative organisms as in PD causes endotoxinemia, leading to profound changes in plasma concentrations of cytokines; altering lipid metabolism. Mean CRP levels were highest in CHD cases (3.53 mg/L); and lowest levels were in controls (2.7mg/L). This demonstrates that CRP behaves in a dose dependent manner and, on an average, CRP levels increase with increasing disease extent. This is in accordance with observations of various investigators like Craig et al [24], Noack et al [25] who observed higher CRP levels in severely diseased subjects as compared to controls. D'Auito et al [26] observed mean CRP value of 2.9mg/L in Italian subjects, whereas in a Japanese population, Yamazaki [27] et al reported much a lower mean CRP value of 0.317mg/L. The mean CRP levels observed in our study (3.53 mg/L) are somewhat higher than that reported by these studies. These differences can be attributed to both differences in quantification of periodontal disease severity as well as racial difference in the populations studied. The role of Plasma fibrinogen as a non specific marker of inflammation in many diseases has been postulated as one of the acute phase reactant produced as a result of increase cytokine action on liver. In the present study a linear elevation of plasma fibrinogen levels were seen in cases and controls when compared to the number of teeth with CAL > 3mm suggesting a co-relation. Using molecular methods, Brian et al [28] identified a high prevalence (60%) of periodontal pathogens' DNA in coronary artery atherosclerotic samples but not in mammary arteries' vessel wall, and suggested a possible direct role of those germs in atherosclerosis Moreover, PD was also reported to modulate HDL structure and to promote a pro-atherogenic lipid profile [29]. PD might create an auto-immune reaction: bacterial species lying in the PD lesions can induce general immune reaction with IgA antibodies production that can cross-react with some epitopes of the host cells.

All those data suggest strongly that intensive treatment of PD might be beneficial on cardiovascular risk factors and improve cardiovascular outcomes. Indeed, several randomized studies recently emphasized the favorable effect of PD treatment on endothelial function [30]. These observations raise the issue of new trends in PD in the context of prevention of systemic inflammation and possibly atherosclerosis. The potential cardio-protective benefits of periodontal therapy may provide an efficacious adjunct to standard therapies of vascular diseases [31, 32].

An attempt was made to determine the relationship between the clinical parameters and severity of CHD. It was found that CAL, number of teeth with CAL > 3mm, percentage of sites which bleed on probing, CRP levels, Plasma fibrinogen, TLC count, and cholesterol levels showed a positive co-relation and statistical significant association with CHD to strengthen these facts further studies with larger sample size are required.

With the amount of literature that has accumulated, it can be presumed that an association between periodontal disease and CHD is relatively common in different populations. The present study although being a case-control design provides further evidence of the role of periodontal disease in cardiovascular disease. However, we agree with others, [21,33] that such studies cannot be generalized to an entire population. Larger and better-controlled studies involving socially homogeneous populations and measuring specific periodontal pathogens are required to identify a definite association between periodontal disease and the risk of CHD.

VI. CONCLUSION

The theory of focal infection, particularly oral infections and their implications were revived in the mid 1980s and has lead to a new speciality called Periodontal Medicine. This paradigm has widened the scope of understanding systemic diseases and assessing its nature of association which could open new insights in terms of its prevention and management.

The outcome of the present study strengths the association between poor periodontal health and CAD. Case-control design although provides a higher strength of evidence as compared to other cross-sectional studies, is still laden with biases. As has already been pointed out elsewhere, our results cannot be generalized to the entire population. Understanding the role of periodontal pathogens in disease initiation and progression needs further research. In order to determine whether periodontal disease is indeed a true risk factor for the association in terms of casual relationship or a true association, further prospective randomized control studies with larger sample sizes and different populations are needed.

Current understanding of epidemiologic, in vitro, clinical and animal studies suggests that periodontal infections may be a contributing risk factor. However, legitimate concerns have arisen about the nature of this relationship. Even in cases where periodontal diseases poses moderate risk in development of CAD which causes significant mortality and morbidity, it is prudent to undertake studies to assess the true nature of relationship. The investigation of a possible clinically meaningful reduction in coronary heart disease resulting from the prevention or treatment of periodontal disease is a imperative in the current scenario.

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Demographic Data	Parameter	Total N= 120	Cases N=60	Control N=60	P value
Sex distribution	Male	79	**66.7%**	**65.0%**	0.847
	Female	41	**33.3%**	**35.0%**	
Age Distribuition	Age	120	51.42	49.32	0.624
Marital status	Married	117	99.4%	98.8%	1.000
	Unmarried	03	0.6%	1.2%	
Educational status	Educated	64	55%	51.6%	0.714
	Uneducated	56	45%	48.4%	
Socio-Economic status	Low	54	46.6%	53.3%	0.129
	Middle/ High	66	43%	57%	
BMI	Mean	120	26.242	22.681	**0.000***
Diabetes	Diabetic	46	58.2%	17%	**0.001***
	Non Diabetic	74	41.8%	83%	
Hypertension	Hypertensive	48	67%	33%	**0.000***
	Non-Hypertensive	72	13%	87%	
Alcohol status	Alcoholic	40	46.7%	20%	**0.002***
	Non- Alcoholic	80	53.3%	80%	
Smoking Status	Smokers	41	41.6%	26.7%	**0.023***
	Non Smokers	79	58.4%	73.3%	

Table 1:- Demographic Data

Mean of Clinical parameters	Cases N=60	Control N=60	P value
Total Leukocyte counts	8972.0	5485.0	**0.000***
Total Cholesterol	154.73	129.76	0.017
Serum Triglycerides	120.13	109.29	0.148
Serum Fibrinogen Levels	393.0	268.2	**0.000***
Serum Hs- C Reactive Protein	3.5	1.9	**0.003***
Clinical Attachment Loss	5.83	2.65	**0.000***
No. Teeth with CAL > 3mm	8.32	3.32	**0.000***
% of Sites with Bleeding of Probing	42.6%	19.3%	**0.001***
Mean of Russel's Periodontal Index	3.8	2.1	**0.012***

Table 2:- Comparison of clinical Parameters

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Variables	Univariates analysis			Multivariate analysis		
	OR	95%CI	P	OR	95%CI	P
Smoking	5.12	2.92-9.46	0.000*	2.86	1.27-6.47	0.01*
Diabetes	4.32	2.86-6.54	0.000*	1.99	2.21-3.82	0.03*
Gingivitis	6.45	3.26-12.77	0.000*	4.30	1.25-9.63	0.001*
PD	3.46	1.99-9.45	0.001*	2.47	1.54-7.53	0.03*

Table 3:- Analysis of Risk factors

VARIABLE	**β**	**SD**	**'P' VALUE**	**ODDS RATIO**
% Of Sites with BOP	0.0051	0.021	0.16	0.2
Number Of Teeth> 3 mm CAL	0.0953	0.028	0.001	1.5
Number of Missing Teeth	0.0932	0.083	0.021	1.3
C-Reactive Protein	0.1244	0.034	0.019	2.0
Serum Fibrinogen	0.0036	0.009	0.028	1.6
TLC Counts	0.0012	0.007	0.001	2.0
Constant	-1.543	0.0323	0.000	-

Table 4:- Logistic regression analysis (n=120) with clinical parameters as independent variable and CAD as dependable Variable