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Comparative Evaluation of Lipid Profile and C-Reactive Protein in Chronic Periodontitis and Coronary Heart Disease

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ABSTRACT

Epidemiological studies suggest local infections may elevate systemic inflammatory mediators and lipid levels, potentially promoting atherosclerosis. **Objective:** To investigate the correlation between Chronic Periodontitis (CP) and Coronary Heart Disease (CHD), assessing C-Reactive Protein (CRP) and lipid profile alterations in affected patients. **Methods:** This case-control study included 88 participants, divided into four groups: 22 with Chronic Periodontitis (CP), 22 with Coronary Heart Disease (CHD), 22 with both CP and CHD and 22 systemically healthy controls, aged 30-60 years, selected through consecutive sampling. Conducted at Bahria University Health Sciences and PNS Shifa Hospital from December 2022 to May 2023, the study assessed clinical and periodontal parameters, including probing depth and clinical attachment level. Fasting blood samples were analyzed for lipid profiles and C-Reactive Protein Levels. Statistical analysis included the Pearson Chi-square test for baseline demographics, the Kruskal-Wallis test for comparing biochemical and periodontal parameters, and the Spearman Rank Correlation. **Results:** Serum HsCRP levels were twice higher in participants with CP and CHD than in healthy individuals and three times higher in subjects with combined disorder (CP + CHD). Patients with both CP and CHD (CP + CHD) have the highest median CRP levels. C-reactive protein was negatively correlated with TC, LDL-C, HDL-C, and number of teeth, while positive correlations were demonstrated with PD, CAL, BoP%, and PI scores. **Conclusion:** Coronary heart disease and other inflammation driven atherosclerotic processes may be exacerbated by chronic infections, such as periodontitis, which can alter systemic levels of TC, HDL, LDL, and CRP.

INTRODUCTION

Periodontitis is a persistent inflammatory condition caused by complex interactions among the oral dysregulated microbes, the host's immunological-inflammatory mechanisms, and a wide range of genetic, behavioural and environmental risk indicators [1]. These interactions lead to permanent damage to the structures of periodontium and loss of tooth. The incidence of periodontitis, particularly its moderate and mild variants, is far higher in adult populations worldwide, with rates of occurrence about 50% [2]. According to the outcomes of the Global Burden of Disease Study carried out in 2019, the worldwide count of periodontal disease cases reached more than one billion, with roughly 91.5 million new occurrences and 7.1 million DALYs recorded in 2019 [3]. Nationally, periodontal disease is estimated to affect approximately 58.4% of Pakistan's population [4]. Coronary Heart Disease (CHD), commonly referred to as Coronary Artery Disease (CAD), is a condition characterized by obstruction of blood vessels (coronary arteries), leading to decreased blood circulation to the heart. It is a gradual disorder that can develop over many years and frequently results from atherosclerosis, a process whereby fatty deposits (plaque) are formed in the coronary arteries [5]. According to the Global Burden of Diseases study, there were 523 million cases of CVDs around the world in 2019. CVD fatalities have risen steadily from 12.1 million individuals in 1990 to an estimated 18.6 million in 2019 [6]. Pakistan has a higher prevalence rate of Coronary Artery

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Disease (CAD) which represents more than 30% of the national population (above 45 years old) being affected by the disease [7]. Epidemiological studies have investigated the correlation between periodontal health and cardiovascular disease. Numerous theories exist about the impact of periodontitis on atherosclerotic plaque development. The influence of pathogenic microbes from biofilm pockets can be categorized into three distinct pathways: bacteremia, dispersion of locally generated inflammatory mediators, and the beginning of an autoimmune response. Periodontitis is a persistent, chronic source of inflammatory mediators, including cytokines and lipopolysaccharides, which might give rise to atherosclerosis. Moreover, the pathogens responsible for periodontitis can breach the periodontal epithelium and infiltrate the bloodstream, resulting in localized atherogenic consequences [8, 9]. There is growing interest in the probable connection between periodontitis and coronary heart disease as measured by C-Reactive Protein (CRP), a widely recognized systemic inflammation indicator. CRP is an inflammatory marker and potential mediator in the pathophysiology of atherosclerosis; it has a long history of association with an increased likelihood of cardiovascular incidents [10]. Another key factor in the development of plaques characterized by atherosclerosis is dyslipidemia, which is defined by dysregulated lipid profiles which include elevated levels of Low-Density Lipoprotein (LDL) cholesterol and reduced levels of High-Density Lipoprotein (HDL) cholesterol [11]. Foam cells filled with lipids and cholesterol form in the endothelium lumen as a consequence of the disruption of Low-Density Lipoprotein (LDL) distribution brought about by the enhanced oxidation seen in CHD and periodontitis. Atherosclerosis and endothelial dysfunction are the ensuing consequences of this mechanism. This may represent the most likely pathway connecting the two diseases [12]. The investigation of the association between periodontitis and cardiovascular disease has underscored the significance of inflammatory mediators generated by oral microorganisms. These mediators infiltrate the endothelium, eliciting inflammatory responses that augment cardiovascular risk. The relationship between inflammatory indicators and lipid molecules in chronic periodontitis, as well as their combined influence on the onset of cardiovascular disease, is not sufficiently comprehended.

The present study aimed to clarify the relationship between these two pathways, enhancing the knowledge regarding how inflammatory processes and dyslipidemia in periodontitis may lead to cardiovascular disease.

METHODS

This case-control research comprised 88 men and women aged 30–60 years. Sixty-six individuals had periodontal and coronary heart disease, while twenty-two were healthy individuals. 22 patients were allocated to the CP, CHD, CP + CHD, and control groups (clinically healthy individuals). The individuals were recruited using a consecutive sampling technique. Before participating in the investigation, each participant executed a written informed consent form. In the Chronic Periodontitis (CP) group, inclusion criteria specified a minimum of 16 natural teeth, with at least 35% of sites exhibiting a clinical attachment level (CAL) of 3 millimeters or greater, a probing depth (PD) of 4 mm or greater, and 40% of sites showing bleeding on probing. The Coronary Heart Disease (CHD) cohort included 30-60-year-olds with ACS, or stable ischemic disease. Participants were confirmed to have stenosis of a minimum of one coronary artery (≥50%) confirmed through coronary angiogram, CABG, or PCI. Patients were also asked about cardiovascular risk elements, medications, underlying medical disorders, previous echocardiography, electrocardiography, and coronary angiography. Similar criteria were used to define the inclusion criteria for the chronic periodontitis and coronary heart disease (CP + CHD) group. Patients in the control group were systemically healthy and did not present with any locations with PD or CAL beyond 4 mm. Radiographs showed no alveolar bone loss and ≤10% BOP locations. All individuals were excluded for diabetes, insulin resistance, chronic renal disease, and SLE. Patients taking hormonal contraceptives, antibiotics, anti-inflammatory drugs, or immunosuppressive drugs for three months before enrolling in the research were excluded. Also, women who were pregnant or nursing, along with individuals who smoked or drank, were exempt. Patients who had periodontal therapy within three months of the study or patients receiving Cyclosporin A, Hydantoin, Nifedipine, and other gingival hyperplasia-causing drugs were not enrolled in the study. In collaboration with the Periodontology outpatient department of Bahria University Dental College Hospital, the Cardiac Care Unit (CCU) of PNS Shifa Hospital, and the Multidisciplinary Research Laboratory, the study was executed in the Biochemistry department of BUHSCK. The Bahria University Health Sciences Campus and Dental College Faculty Review Committee (FRC) (FRC-BUHS-50/2022-508) and Ethical Review Committee (ERC) (ERC 04/2023) approved the six-month study starting from December 2022 to May 2023. The sample size was estimated using ADMA levels as the major outcome variable. G Power version 3.1.9.2 (mean of F test, one-way ANOVA with fixed effects) calculated that 88 subjects divided into four groups were needed, assuming an effect size of 0.379, an a priori power of 0.80, an alpha level probability, and four groups. Twenty-two people per cohort were recruited [14]. Data were stored and analyzed using IBM-SPSS version 23.0. The normality of the data was evaluated using the Shapiro-Wilk test. Counts with percentages were reported on baseline demographics across the four studied groups. Median with interquartile range (75^{th} percentile-25^{th} percentile) was reported for CRP, lipid profile, and periodontal parameters. The association of baseline characteristics was tested using the Pearson Chi-Square

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test. To report the correlations between biochemical and periodontal parameters, Spearman rank correlation analysis was implemented. All p-values less than 0.05 were considered statistically significant.

RESULTS

In the current study there were four study groups, each contained twenty-two samples. In Control group there were 59.1% individuals who were aged less than or equal to 40 years old among which 40.9% were females. In CP group there were 59.1% were aged 41-50 years old among which 18.2% were female gender. In CHD group there were 54.5% were aged more than 50-years old, 4.5% were female gender, whereas in CP + CHD groups there were 68.2% individuals who were aged more than 50-years old, none was female gender. The Pearson Chi Square test provided a significant association of age group and gender with studied groups ($p<0.05$) (Figure 1).

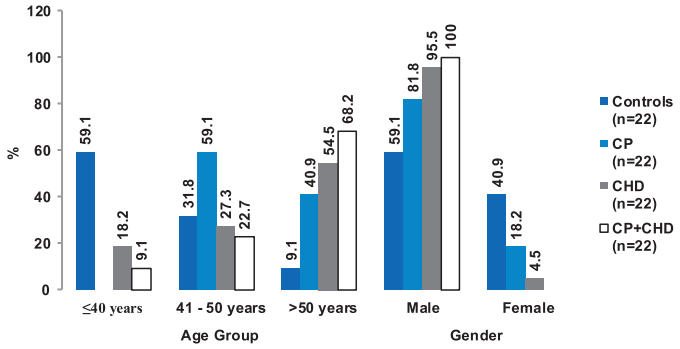

Figure 1: Distribution of Study Participants According to Age and Gender

Lipid profile demonstrated that total cholesterol was significantly higher for CP group compared to CHD and CP + CHD groups. The difference between the tested groups was not significant for triglycerides. HDL was significantly low in CHD group compared to both groups CP and CP + CHD. LDL levels between groups were significantly different depicting low concentrations for CP + CHD group while higher values for CP, CHD and controls ($p<0.01$) (Table 1). Patients with CP and CP + CHD presented with a higher median values of PD and CAL compared with CHD and healthy individuals ($p<0.01$) (Table 1).

Table 1: Comparison of CRP, Lipid Profile and Periodontal Parameters across the Studied Groups

Variables	Controls	CP	CHD	CP + CHD	p-Value
	Median (Q3 – Q1)	Median (Q3 – Q1)	Median (Q3 – Q1)	Median (Q3 – Q1)	
CRP mg/L	1.05 (1.5-0.7)	3.55 (4.4-2.9)	3.6 (7.15-0.82)	6.85 (9.2-4.36)	<0.01*
TC (mg/dL)	185 (195-180)	185.85 (190 -182.35)	160.61 (205.11 -127.32)	147.45 (172.67 -135.45)	<0.01*
TG (mg/dL)	131 (140-122)	116.1(137.8 -109.4)	140.36 (170.7 -100.32)	135.45 (158.4 -110.88)	0.38
HDL-C (mg/dL)	50 (55-45)	37.6 (41.9-35.6)	34.64 (42.96-29.41)	37.75 (48.02-30.19)	<0.01*
LDL-C (mg/dL)	120 (125-110)	104.6 (108.2 -101.2)	116.49 (144.35-81.27)	85.95 (95.2-77.4)	<0.01*
PD (mm)	1.5 (2-1)	4.5 (5-4)	2 (2.5-1)	5.5 (6-5)	<0.01*
CAL (mm)	2.25 (2.5-2)	7 (8-6)	2.75 (3.5-2.5)	7.75 (9-7)	<0.01*

Group CP= Chronic Periodontitis, Group CHD= Coronary Heart Disease, Group CP + CHD= Chronic Periodontitis and Coronary Heart Disease, TC= Total Cholesterol, TG= Triglycerides, HDL-C= High Density Lipoprotein Cholesterol, LDL-C= Low Density Lipoprotein Cholesterol, PD= Probing Depth, CAL= Clinical Attachment Loss, Test applied: Kruskal Wallis Test, *p<0.05 considered statistically significant. CRP has a negative correlation with Total Cholesterol (31.3%), LDL cholesterol (46.5%), HDL cholesterol (21.8%), number of teeth (63.1%) and a positive correlation with probing depth (55.3%), clinical attachment loss (51.9%), bleeding on probing (61.9%) and plaque index score (43.6%). Total cholesterol has a negative correlation with probing depth (50.3%), clinical attachment loss (31.7%) and bleeding on probing (32.5%) and a positive correlation with LDL cholesterol (70.2%) and number of teeth (37.3%). LDL cholesterol has a negative correlation with probing depth (64.6), clinical attachment loss (56%), bleeding on probing (48.9%) and plaque index score (35.3%) and a positive correlation with number of teeth (46.8%). HDL cholesterol has a negative correlation with bleeding on probing (25.2%) and plaque index score (34.4%) and a positive correlation with number of teeth (28.8%) (Table 2).

Table 2: Correlation between Lipid Profile and Periodontal Parameters (Spearman Rank Correlation)

Variables	CRP (mg/L)	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL Cholesterol (mg/dL)	HDL Cholesterol (mg/dL)
CRP mg/L	1	-	-	-	-
Total Cholesterol (mg/dL)	-0.313**	1	-	-	-
Trigly -cerides (mg/dL)	0.09	0.085	1	-	-
LDL Cholesterol (mg/dL)	-0.465**	0.702**	0.106	1	-
HDL Cholesterol (mg/dL)	-0.218*	0.16	-0.016	0.125	1
Number of Teeth	-0.631**	0.373**	-0.017	0.468**	0.288**
PD (mm)	0.553**	-0.503**	-0.097	-0.646**	-0.181
CAL (mm)	0.519**	-0.317**	-0.072	-0.560**	-0.141
BoP %	0.619**	-0.325**	0.041	-0.489**	-0.252*
PI Score	0.436**	-0.086	-0.098	-0.353**	-0.344**
** Correlation is Significant at the 0.01 Level (2-Tailed)					
* Correlation is Significant at the 0.05 Level (2-Tailed)					

DISCUSSION

Inflammation in the development of Cardiovascular Diseases (CVD) has received considerable focus in recent times, with chronic periodontitis being identified as a possible causative element. Among the indicators of inflamed tissues, C-Reactive Protein (CRP) was especially

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significant because of its well-established link with endothelial dysfunction, which was a precursor to atherosclerosis. C-Reactive Protein (CRP) serves as both an indicator of inflammation and an active contributor to the inflammatory pathways, establishing a connection between periodontal disease and cardiovascular disorders. Endothelial dysfunction, demonstrated by the compromised capacity of blood vessels to expand, was a pivotal occurrence in the initial phases of atherosclerosis. The blood lipid profile, characterized by increased concentrations of LDL and diminished concentrations of HDL, was essential in the progression of endothelial dysfunction. The presence of dyslipidemia, in conjunction with increased CRP levels, intensifies the inflammatory reaction, therefore facilitating the development of atherosclerosis. In the present study, 59.1% of individuals were aged between 41 and 50 in the CP group. Among the CHD and CP + CHD groups, 54.5% and 68.2% were aged above 50 years. Males outnumbered females in all four groups (59.1%, 81.8%, 95.5%, 100%). This may be because male dental hygiene and periodontal health did not meet enrollment standards for a healthy periodontium [13-15]. In this study, CP patients had considerably higher median CRP readings than controls. Aoyama N *et al.*, found similar hsCRP results in CP and healthy people [16]. In CHD patients, C-reactive protein was much higher than in controls. Some of the participants had just experienced an angina episode, which may have boosted their levels. Similarly, Esteves-Lima RP *et al.*, found that hs-CRP was strongly linked to CHD [17]. Compared to controls and CP groups, CP + CHD patients showed significantly higher C-reactive protein values. These results lined up with literature reported in previous studies [18-20]. CVD was exacerbated by inflammation [21]. The synthesis of CRP in hepatocytes was enhanced by pro-inflammatory cytokines generated locally at infection or inflammation sites, which were known to increase the risk of atherosclerotic implications. CRP may stimulate human endothelial cell adhesion molecule expression and atherosclerotic lesion development [21, 22]. Thus, severe periodontal disease may be linked to atherosclerotic lesions. Based on the findings of the investigation, it was found that CHD and CP + CHD patients had lower cholesterol levels compared to CP individuals and controls. It was attributed to the fact that these individuals were on cholesterol-lowering therapy. HDL has an atheroprotective effect that is mediated by reverse cholesterol transport. This study found that patients who had both chronic periodontitis and Coronary Heart Disease (CHD) consistently exhibited lower levels of High-Density Lipoprotein (HDL), which was consistent with the well-established association between reduced HDL and increased cardiovascular risk. The decrease in HDL levels can be ascribed to the persistent inflammatory state present in both disorders, which was recognized to adversely affect lipid metabolism. This result highlights the possible influence of systemic inflammation in worsening lipid abnormalities, hence contributing to the advancement of cardiovascular disease in these individuals. The highest median values for parameters PD and CAL were recorded in individuals having combined disease (CP + CHD). 23.Gupta S *et al.*, conducted a study on periostin levels to evaluate the correlation between CHD and persistent periodontal disease [23]. They found out that the highest mean values were found in the group CHD-CP for periodontal parameters.Furthermore, these findings were by the literature documented by Kumar KR *et al.*, which demonstrated the highest mean values for PD and CAL in CP + CHD patients [25]. Clinical periodontal parameters and lipid profiles have substantial correlations, highlighting the complicated link between systemic inflammation, lipid metabolism, and periodontal health. Inflammatory markers, such as CRP, were negatively correlated with total cholesterol, LDL, HDL, and tooth count, while positive correlations were found for probing depth, CAL, BoP, and PI score ($p<0.01$). This suggested that inflammation may autonomously affect periodontal and cardiovascular health. These findings were aligned with the literature reported by Kumar KR *et al* [24, 25]. However, total cholesterol and LDL were positively associated with periodontal parameters, including tooth count, demonstrating a complex relationship between lipid status and oral health. According to research by Katz J *et al.*, males suffering from periodontitis had far greater levels of LDL and total cholesterol in their blood compared to those with healthy periodontal tissue or gingivitis [26, 27]. One of the study's key drawbacks was its limited sample size, which may limit the findings' applicability to a larger population. A limited sample size can reduce statistical power, making it difficult to identify subtle but clinically significant variations between groups. Another restriction was the potential bias imposed by the exclusion criteria, which were particularly relevant to lifestyle factors such as smoking, alcohol consumption, and poor eating habits. While these eliminations were essential to control for confounding variables, they may limit the study's application to real-world groups that exhibit such behaviours. By omitting people with these lifestyle characteristics, the study may not accurately represent the average periodontitis patient group, which frequently has complicated connections between oral health, lifestyle, and cardiovascular disease. This may result in an underestimation of the cumulative influence of these factors on interest-related outcomes. The clinical implications of the correlation between dyslipidemia, C-Reactive Protein (CRP), Coronary Heart Disease (CHD), and Chronic Periodontitis (CP) were significant. CP patients

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may be at an increased risk for cardiovascular events due to high levels of CRP and dyslipidemia, which were established risk factors for CHD. Additionally, the results indicate that specific measures, such as lipid-lowering compounds or anti-inflammatory therapies, may be beneficial for CP patients in reducing cardiovascular morbidity.

CONCLUSIONS

There was a complex relationship between dyslipidemia, inflammation, and periodontal health, as evidenced by the strong correlations between systemic indicators of inflammation, lipid profiles, and periodontal parameters. The potentially detrimental impact of inflammation on both cardiovascular and periodontal diseases was underscored by the negative associations between CRP and lipid levels and the positive associations between CRP and periodontal parameters. The role of inflammation in modulating lipid metabolism and periodontal disease was supported by these insights, necessitating additional research to investigate such relationships and the consequences for treatment strategies in both cardiovascular and periodontal health.

Authors Contribution

Conceptualization: AZ
Methodology: AZ
Formal analysis: AZ, AB
Writing, review and editing: FT, SBA, HA, ANK

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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