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Evaluation of Systemic Inflammatory Markers in Clinically Healthy Subjects and Chronic Periodontitis Patients

Dr. Rajesh K.S.[1], Dr.Spoorthi K. H.[2],Dr.Shashikanth Hegde[3], Dr.Arun Kumar M.S.[4]

[1,4,](Professor, Dept. of Periodontics, Yenepoya Dental College & Hospital, Mangalore, India)
[2](Post Graduate student, Dept. of Periodontics, Yenepoya Dental College & Hospital, Mangalore, India)
[3](Professor & Head, Dept. of Periodontics, Yenepoya Dental College & Hospital, Mangalore, India)

Abstract:Aim:*To determine whether the levels of WBCs, Platelets and Serum Protein parameters including Total Protein, Serum Albumin and Serum Globulin are altered in subjects with chronic periodontitis compared to healthy controls.*

Materials and Methods: *A total of 50 systemically healthy subjects, aged 25 to 40 years were included in the study. 25 subjects with clinically healthy gingiva were assigned to the control group and 25 subjects with chronic periodontitis were assigned to the test group. Clinical parameters including Plaque Index (PI), Gingival Index (GI), Pocket Depth (PD), Clinical Attachment Level (CAL) were measured. Venous blood samples were taken to analyze hematological parameters including WBC, Platelets, Total Protein, Serum Albumin, Serum Globulin and Albumin/Globulin ratio.*

Results: *The mean values for WBC, Platelet, Total Protein, Serum Albumin, Serum Globulin, Albumin/Globulin ratio were found to be statistically non-significant between the subjects with clinically healthy gingiva and chronic periodontitis subjects.*

Conclusion: *In conclusion, in the present study, although variations were evident in the values of hematological parameters between the subjects with clinically healthy gingiva and subjects with chronic periodontitis, it was not statistically significant.*

Keywords *–WBC, Platelets, Serum Protein, Inflammation,Periodontitis*

I. Introduction

For thousands of years blood has been regarded as the ultimate fluid that could indicate disease processes. In the past decade, there has been a renewed interest in the ways in which periodontitis may affect changes in cellular and molecular components of peripheral blood.[1] Understanding the etiology and pathogenesis of periodontal diseases and their chronic, inflammatory and infectious nature necessitates recognizing the possibility that these infections may influence events elsewhere in the body. The concept that oral diseases and systemic diseases influence each other goes back to the theory of focal infection.[2]

In 1891, Miller[3]published his theory regarding focal infection in which he indicated that microorganisms and their products are able to access parts of the body that are adjacent to or distant from the mouth. The proponents of this concept assume that microorganisms present in dental plaque and their metabolic products may enter the bloodstream, thereby causing many systemic diseases and sometimes resulting in degenerative conditions. In recent years, the concept of focal infection has changed and now mostly relies on the correlation between chronic periodontitis and systemic diseases. Offenbacher[4]introduced the concept of 'periodontal medicine' as a discipline focusing on validating this relationship and its biological plausibility using human and animal studies.

At present, it is generally known that a patient's oral status is connected with their systemic health, as poor oral health may cause serious underlying diseases concomitantly and may predispose patients to developing other systemic diseases.[5]Periodontitis has recently been identified as a potential risk factor for systemic pathologies such as cardiovascular disease. The hypothesis being that local periodontitis triggered by bacterial insult, is chronic inflammatory disease and that production of circulating cytokines may contribute to the development of atherosclerosis, cardiovascular disease, premature birth of children with small body weight and diabetes.[6]The results of several studies in the periodontal literature suggest that adult patients with periodontitis frequently present with significant differences in blood parameters when compared to healthy controls, and while the values for the different blood parameters investigated oftendo not exceed the reference values used in the clinic, the literature is consistent that periodontitis patients do have elevated systemic markers of inflammation when they are compared to subjects without periodontitis.[7]

Hence the aim of the present study was to evaluate the systemic inflammatory markers in clinically healthy subjects and chronic periodontitis patients.

II. Methodology

The present study was conducted in the Department of Periodontology, Yenepoya Dental College, Mangalore. The study was approved by Yenepoya University Ethical Committee and informed consent was obtained from all the patients. A total of 50 systemically healthy subjects between the age groups of 25-40 years were included in the study. Group 1 included 25 Subjects with clinically healthy gingiva with a gingival index score of ≤ 1 mm.Group 2 included 25Chronic periodontitis patients with a probing depth of ≥ 4mm in >30% of sites.Patients with underlying systemic diseases, patients with current or past habit of tobacco smoking or chewing, Pregnancy, patients with history of antibiotics, NSAID's, antimicrobial drugs, mouthwashes, or vitamin supplements within the past 3 months were excluded from thestudy. Clinical parameters recorded were Plaque index by Silness and Loe (1964), Gingival index by Loe and Silness (1963)[8], Probing pocket depth (PD), Clinical attachment level (CAL).

5 ml of venous blood was drawn from the ante-cubital fossa and collected in 2 vacutainers, one with Ethylene diamine tetra acetic acid (EDTA) as an anticoagulant and one without anticoagulant. 2.5 ml of anti-coagulated blood was used to estimate the WBC and platelet count using an automated hematology analyzer and 2.5 ml of blood without anticoagulant was used to estimate the Serum protein parameters using an automated biochemical analyzer. Data was statistically analyzed using student't' test and chi square test. A p-value of 0.001 or less was considered to be significant.

III. Results

In the present study, Mean WBC count for the test group was 7.428×10^9/L ± 1.465 and for the control group was 7.696×10^9/L ± 1.622, which was found to be statistically non-significant (p=0.543). (Table 1, Fig 1). Mean platelet count for the test group was 247.76×10^9/L ± 58.201 and for the control group is 259.64×10^9/L ± 50.388, which was found to be statistically non-significant (p=0.444). (Table 2, Fig 2).

Mean total protein count for the test group was 7.94 ± 1.163 and for the control group was 7.688 ± 0.401, which was found to be statistically non-significant (p=0.311). (Table 3, Fig 3). Mean serum albumin count for the test group was 4.404 ± 0.291 and for the control group was 4.296 ± 0.244, which was found to be statistically non-significant (p=0.161). (Table 4, Fig 4). Mean serum globulin count for the test group was 3.536 ± 1.051 and for the control group was 3.392 ± 0.269, which was found to be statistically non-significant (p=0.510). (Table 5, Fig 5). Mean serum albumin/globulin ratio for the test group was 1.252 ± 0.185 and for the control group was 1.228 ± 0.117, which was found to be statistically non-significant (p=0.587). (Table 6, Fig 6).

Mean GI score for the test group was 2.271 ± 0.308 and for the control group is 0.353 ± 0.042, which was found to be statistically highly significant (p=0.000). (Table 7, Fig 7). Mean PI score for the test group was 2.177 ± 0.249 and for the control group was 0.491 ± 0.038, which was found to be statistically highly significant (p=0.000). (Table 8, Fig 8). Mean Probing pocket depth value for the test group was 4.30 ± 0.08 and the Mean Clinical attachment level for the test group was 3.11 ± 0.11.(Table 9).

Table 1: Mean WBC count×10^9/L

WBC

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	7.428	1.465	6.823	8.033	.613	.543
Control group	25	7.696	1.622	7.026	8.366		NS

Table 2: Mean Platelet count×10^9/L

PLATELET

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	247.760	58.201	223.736	271.784	.772	.444
Control group	25	259.640	50.388	238.841	280.439		NS

Table 3: Mean Total Protein level (gm/dl)

TOTAL PROTEIN

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	7.940	1.163	7.460	8.420	1.024	.311
Control group	25	7.688	.401	7.522	7.854		NS

Table 4: Mean Serum Albumin level (gm/dl)

SERUM ALBUMIN

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	4.404	.291	4.284	4.524	1.422	.161
Control group	25	4.296	.244	4.195	4.397		NS

Table 5: Mean Serum Globulin level (gm/dl)

SERUM GLOBULIN

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	3.536	1.051	3.102	3.970	.664	.510
Control group	25	3.392	.269	3.281	3.503		NS

Table 6: Mean Serum Albumin/Globulin (gm/dl) ratio

ALBUMIN/GLOBULIN

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	1.252	.185	1.176	1.328	.548	.587
Control group	25	1.228	.117	1.180	1.276		NS

Table 7: Mean Gingival Index

GI - Test

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	2.271	.308	2.144	2.398	30.841	.000
Control group	25	.353	.042	.336	.370		HS

Table 8: Mean Plaque Index

PI -Test

	N	Mean	Std. Deviation	95% Confidence Interval for Mean		t value	p value
				Lower Bound	Upper Bound		
Test group	25	2.177	.249	2.074	2.280	33.416	.000
Control group	25	.491	.038	.475	.507		HS

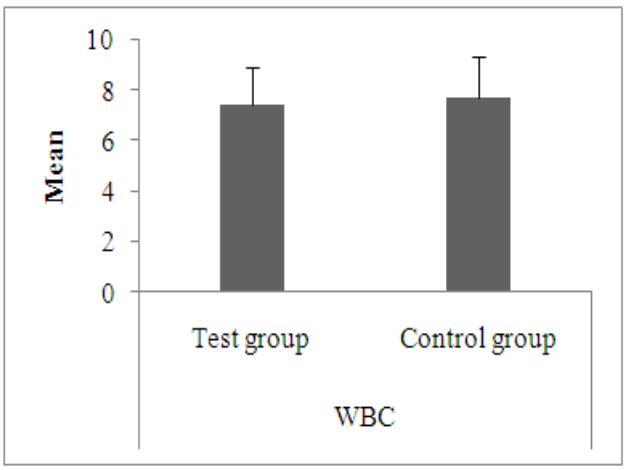

Figure 1: Mean WBC count×10^9/L

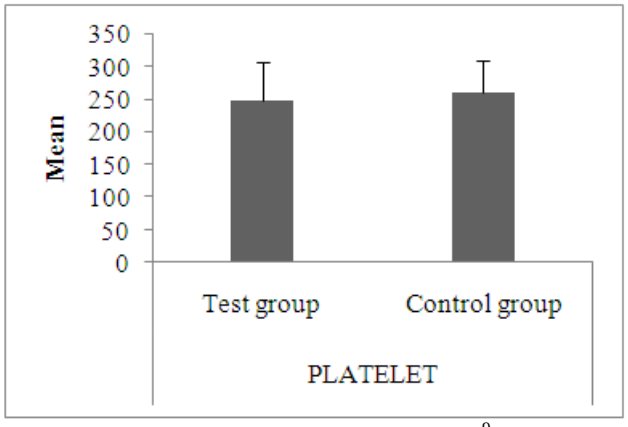

Figure 2: Mean Platelet count×10^9/L

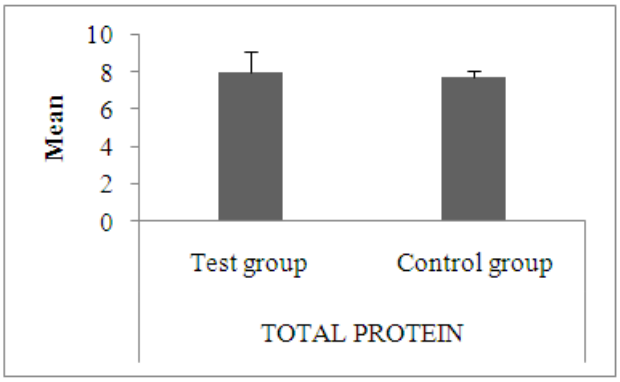

Figure 3: Mean Total Protein level (gm/dl)

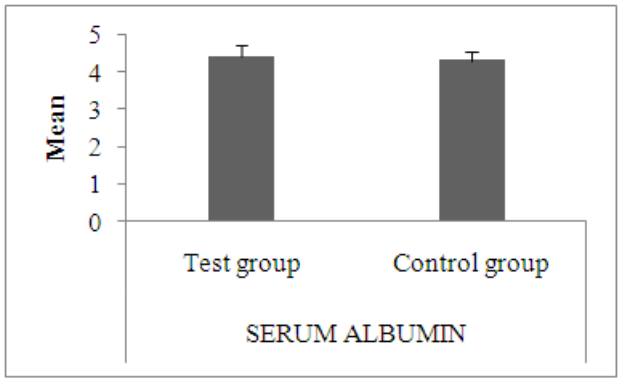

Figure 4: Mean Serum Albumin level (gm/dl)

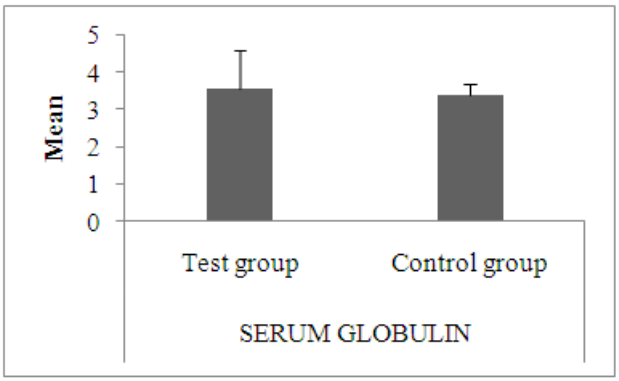

Figure 5: Mean Serum Globulin level (gm/dl)

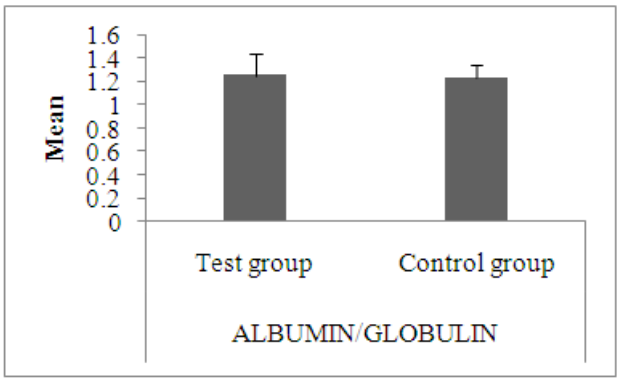

Figure 6: Mean Serum Albumin/Globulin (gm/dl) ratio

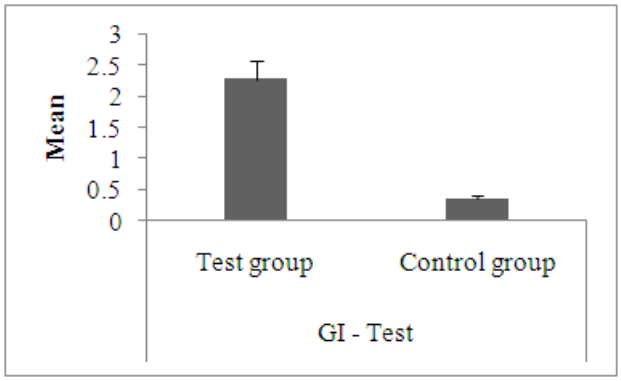

Figure 7: Mean Gingival Index

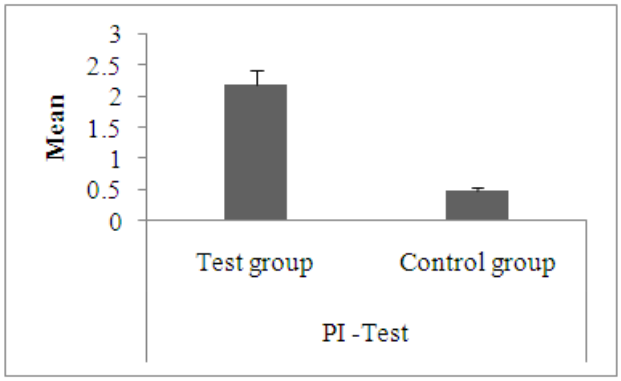

Figure 8: Mean Plaque Index

IV. Discussion

A total blood analysis or hemogram is frequently used to assess the presence of infection or inflammation, and the question whether periodontal infections affect hematological parameters such as the differential counts of white blood cells, red blood cells, and/or platelets has interested several research groups. Some of these studies have documented differences in the hematological parameters for subjects with chronic periodontitis compared to healthy subjects, although the findings are not always conclusive.[7]

In the present study, the difference in WBC count between the healthy and control group was found to be statistically non-significant. The mean WBC count was similar in patients with periodontitis and in subjects with clinically healthy gingiva. But higher level of lymphocytes was observed in the patients with periodontitis compared to subjects in the healthy group which can be attributed to the presence of infection. This can lead to chronic inflammation of the periodontium leading to periodontitis. Our findings corroborate with the findings reported by Loos BG et al. (2000)[9], Gustafsson and Asman (1996)[10], Frederiksson et al. (1998)[11], Frederiksson et al. (1999)[12] andFokkema et al. (2002).[13]In the above studies, they found an elevated level of total WBC count in the periodontitis patients, which was explained by the presence of higher number of neutrophils. Hence, they concluded that periodontitis predispose affected patients to cardiovascular diseases (CVD).

Platelets are essential for primary hemostasis and endothelial repair, but also play a key role in atherogenesis and thrombus formation. Platelet count has been associated with vascular and non-vascular death and a recent meta-analysis showed that mean platelet volume is a predictor of cardiovascular risk. In the present study, the mean platelet count in patients with periodontitis was found to be lower in number compared to subjects with healthy gingiva, which was statistically non-significant. This was in agreement with a study by Kumar BP et al. (2014)[14] where it was concluded that there is no significant role of platelets in periodontal infection even though it has a major role in atherogenesis.

In the present study, the mean total protein level for the chronic periodontitis group was slightly higher compared to control group, which was statistically non-significant and the mean serum albumin level for chronic periodontitis group was slightly elevated compared to the control group, which was statistically non-significant. This was in agreement with studies conducted by Ogawa H. et al. (2006)[15], Kolte RA et al. (2010)[16] where a correlation between periodontal disease events and serum albumin levels could not be established suggesting an inverse relationship between the serum albumin concentration and chronic periodontal disease. Iwasaki M. et al. (2008)[17]concludedthat serum albumin concentration is a significant risk predictor of periodontal disease progression among elderly non-smokers. Furthermore, a low serum albumin concentration may impair the periodontal condition. It is observed in several studies that a lower serum albumin level might be responsible for periodontal disease progression. The serum albumin value might be a good marker for a subject's general health condition.

In the present study, the mean serum globulin level for chronic periodontitis was slightly higher compared to the control group, but was statistically non-significant and the mean albumin/globulin ratio for chronic periodontitis was similar to the control group, which was statistically non-significant.Shi D et al. (2008)[18] concluded that patients with aggressive periodontitis (AgP) may have elevated peripheral leukocyte number and serum globulin levels as well as decreased serum albumin levels and albumin/globulin ratios compared to controls. These changes might be associated with the severity of periodontal destruction. In the present study, mean gingival index for the chronic periodontitis group was higher compared to the control group, which was statistically highly significant and the mean plaque index for the chronic periodontitis group was higher compared to the control group, which was statistically highly significant.

V. Conclusion

In conclusion, in the present study, although variations were evident in the values of hematological parameters between the subjects with clinically healthy gingiva and subjects with chronic periodontitis, it was not statistically significant.

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