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"QUANTITATIVE ESTIMATION OF ACUTE-PHASE PROTEIN (C-REACTIVE PROTEIN) IN GINGIVAL CREVICULAR FLUID IN CHRONIC PERIODONTITIS, BEFORE AND AFTER NON SURGICAL THERAPY"-CLINICO- BIOCHEMICAL STUDY

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Keywords:
C-reactive protein, GCF, Periodontitis.

Abstract

The present clinical study was to designed to quantify C - reactive protein levels of Gingival Crevicular Fluid and to know the effect of non surgical therapy in minimizing the C - reactive protein levels in chronic generalized periodontitis.clinical parameters like Gingival index, Sulcusbleeding index, Probing pocket depth, Clinical attachment level and C-reactive protein levels were recorded at different points of time over a period of 45 days.

After recording clinical parameters and Collection of GCF, scaling and root planning done for all the patients and every recall visit i.e. 14th ,45th day oral hygiene instructions were reinforced and clinical parameters like Gingival index, Sulcusbleeding index, Probing pocket depth, Clinical attachment level were recorded and GCF was collected for estimation of C-reactive protein. At the end of 45 days there was a significant reduction in Gingival index, Sulcusbleeding index, Probing pocket depth and gain in Clinical attachment level and decreased C - reactive protein levels of Gingival crevicular Fluid was observed. There was a 26% reduction in probing pocket depth and 38% gain in clinical attachment level and about reduction of 65% after 14 days and 100% reduction of C - reactive protein levels in Gingival crevicular Fluid after 45 days. Thus the results show that there is presence of C - reactive protein level makes more significant in Gingival crevicular Fluid and confirms the underlying inflammatory component of the disease activity in chronic periodontitis.

INTRODUCTION

Recent years have seen the revival of the concept that periodontitis may have an etiological or modulating role in other systemic diseases. One of several explanatory mechanisms is based around the concept of periodontitis having effect by the systemic dissemination of mediators such as C - reactive protein, Interleukin – 6, Tumor necrosis factor – α. Such a response could possibly be activated either by local infection by bacteria resulting in inflammatory damage of periodontal tissue, or by the systemic spread of bacteria or their toxins and products, during the course of the periodontal disease. This bacterial pathogens, bacterial antigens, endotoxins and inflammatory cytokines like C-Reactive proteins, Interleukin-1, and tumor necrosis factor-α contribute and modify the process of atherogenesis and thromboembolic events[1].

The elevated cell and cytokine mediated markers of inflammation, including C - reactive protein, fibrinogen, and various cytokines, are associated with periodontal disease. The same elevated proinflammatory factors in periodontal disease have also been linked with atherothrombogenesis. The relation between vascular events and periodontal disease is further supported by evidence showing that oral bacteria can cause platelet aggregation and thromboembolic events by upregulating the expression of platelet aggregation – associated protein. C-reactive

protein also induces monocytes/macrophages to produce tissue factor, which stimulates the coagulation pathway and increases blood coagulability. Increased fibrinogen levels may contribute to this process. C - reactive protein stimulates the complement cascade, further exacerbating inflammation. It has been suggested that testing C - reactive protein levels in the gingival crevicular fluid may be a new way to assess cardiovascular disease risk. The most important role of C - reactive protein is its interaction with the complement system, which is one of the body's immunologic defense mechanism. Normally there is no C - reactive protein in the blood but a positive C - reactive protein may include any of a number of diseases like rheumatoid arthritis, rheumatoid fever, cancer, tuberculosis, pneumococcal pneumonia, myocardial infarction and periodontal disease.[2]

C – Reactive protein levels fluctuate from day to day, and levels increase with ageing, high blood pressure, alcohol use, smoking, chronic fatigue, coffee consumption, having elevated triglycerides, insulin resistance diabetes, taking estrogen, eating a high protein diet, suffering sleep disturbances and depression. Alcohol can cause inflammation and raise C - reactive protein. At this time, the best way we know to reduce C - reactive protein levels are exercise and a diet that includes omega – 3 fatty acids. Statins appear to protect against inflammation as well as cholesterol.[3]

Elevated levels of C-reactive protein and decreased plasma adiponectin are associated with increased risk of atherosclerosis. As periodontal disease has been suggested to act as a risk factor for atherosclerosis, Iwamoto Y and Nishimura F (2003) examined the effect of antimicrobial periodontal treatment on C-reactive protein, Adiponectin and Tumor Necrosis factor-α level. Periodontal treatment is effective in reducing C-reactive protein and Tumor Necrosis factor-α, while adiponectin does not appear to be influence by periodontal treatment. Elevated levels of C-reactive protein and Tumor Necrosis factor-α may be associated with increased risk for further development of atherosclerosis in periodontitis patient.[4]

Based on the observations with various studies, we now know that C-reactive protein along with Interleukin-6 and Tumor Necrosis factor-α is acute phase proteins and has been identified as an inflammatory marker. Levels in blood may be a new way to assess cardio vascular disease. There are also evidence suggesting correlation between altered level of C-reactive protein and alveolar bone loss around posterior teeth, thus establishing relationship between chronic periodontal disease and increased risk of cardio vascular disease.

Hence this present clinical study was undertaken to quantify C - reactive protein levels of Gingival crevicular Fluid in chronic generalized periodontitis.

MATERIALS AND METHODS

Sixty patients diagnosed with generalized chronic periodontitis having pocket depth ≥ 5mm with radiographic evidence of bone loss, were selected for this study from the Outpatient Department of Periodontics, Darshan Dental College and Hospital, Udaipur (Rajasthan)

Selection Criteria-

Inclusion criteria

1. Patients diagnosed as having generalized chronic periodontitis with probing depth of ≥ 5 mm and radiographic evidence of vertical bone loss.
2. Age group of 35-55 years.
3. Patients with good general health, without any history of systemic disease
4. Non smoker patients with acceptable oral hygiene.

Exclusion criteria

1. Patients were excluded if they had undergone oral prophylaxis or taken antibiotics 6 month prior to inclusion for the study.
2. Pregnant and nursing patient.
3. Inability to comply with the follow-up visit requirements.

Study design

A written informed consent form explaining the nature of the study and Lab investigation procedure was signed by the patient. Gingival crevicular fluid was collected using micro capillary pipette which was hand calibrated at every 1 mm till 10 mm, from selected sites (Fgure.2, with maximum pocket depth) in the subjects on 0 day (prior to phase I therapy), 14th day and 45th day and gingival index (Loe H and Sillness J), Sulcus bleeding index (Mulheman), clinical probing pocket depth, and clinical attachment loss were recorded in a prepared chart. On every recall visit i.e. 14th day, 45th day Oral Hygiene Instructions were reinforced and Gingival crevicular Fluid was collected for estimation of C - reactive protein. Clinical parameters like gingival index, bleeding index, clinical probing pocket depth and clinical attachment loss were recorded.

CLINICAL ATTACHMENT LEVEL

Clinical attachment level was measured with University of North Carolina (UNC-15) periodontal probe and it is the distance measured from the cemento-enamel junction to the base of the pocket. The customized occlusal stent (Figure 3) was placed on the selected teeth and the probe was gently inserted along the groove on the stent and the distance from the fixed reference point on the stent to the base of the pocket and distance from fixed reference point to the Cemento-Enamel Junction was recorded. Clinical attachment level was obtained by subtracting the distance of fixed reference point to cemento enamel junction from fixed reference point to base of the pocket.

Clinical Attachment Loss = Fixed Reference Point to Base of Pocket – Fixed Reference Point to Cemento -Enamel Junction

CLINICAL PROBING DEPTH

Clinical probing depth was measured from all the four surfaces of tooth by using University of North Carolina (UNC-15) periodontal probe was probed at six points on each tooth i.e. buccal, lingual, mesiobuccal, mesiolingual, distobuccal, distolingual. The pocket depth was measured using a UNC 15 probe from fixed reference point on the stent to the base of the pocket and from the fixed reference point to the free gingival margin. Probing pocket depth was obtained by subtracting distance of the Fixed Reference Point to Gingival Margin from Fixed Reference Point to Base of Pocket. Pocket Depth = Fixed Reference Point to Base of Pocket – Fixed Reference Point to Gingival Margin. If the reading was present between 2 markings, the reading was rounded off to the next highest millimeter.

METHOD OF COLLECTION OF GINGIVAL CREVICULAR FLUID

Patients selected were seated comfortably in the dental chair with a proper illumination; samples of gingival crevicular fluid were taken from the selected sites by placing calibrated volumetric microcapillary pipettes with 0-5µl range (“Sigma chemical company”. St,Lowis, Missouri USA.) using extracrevicular method.

Gingival crevicular fluid sample were collected only from those tooth which had average pocket probing depth in each subject. Supragingival plaque obstructing access to the entrance of the crevice was carefully removed using sterile periodontal probe.

These selected sites for gingival crevicular fluid sampling was isolated with cotton rolls and are air dried using three way syringes for 2 Seconds. Calibrated glass micro-pipettes (0.5 µl volume) were placed at the opening of the gingival crevice and fluid was collected for a period of 15-20 mints. It was then transferred to a glass vial containing 100 µl of phosphate buffer (pH 7, 0.05 M) solution containing 0.1% w/v bovine serum albumin, and stored at -80ºC until assay. The glass vial was immediately taken to the lab for analysis.

TECHNIQUE: (QUANTIA CRP UV)

This consists of vitros C - reactive protein slide which is dry, multilayered, analytical elements coated on a polyester support. In this format a derivative of phosphorylcholine is covalently bond to polystyrene polymer beads and in the presence of calcium serves as a capture agent. Monoclonal anti-C-reactive protein antibody conjugated to horseradish peroxidase serves as a signal generator. An 11µl of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlining layers. C-reactive protein in the sample binds to PC-linked capture beads and anti- C-reactive protein antibodies labeled with horseradish peroxidase to form an insoluble sandwich complex. The subsequent addition of vitros immune-wash fluid to the slide removes unbound material

from the bead area, while also providing the H^2O^2 required for the enzyme mediated oxidation of leucodye. (Fgure1).

Reaction sequence:

CRP + PC + Ab.HRP $\xrightarrow{Ca2+}$ CRP – Ab.HRP + Ab.HRP

Immune wash + PC – CRP – Ab.HRP + Ab.HRP $\xrightarrow{wash}$ Ab.HRP

H2O2 + leukodye + PC – CRP – Ab.HRP $\xrightarrow{dye}$ H2O

PC = phosphorylcholine beads

Ab.HRP = anti – CRP monoclonal antibody labelled with horseradish peroxidise

Now this slide is transferred into an automated analyser. This analyser measures the reflection density of the dye at 670nm. To determine if an adequate wash has occurred, the wash detection dye is read at 540nm during the final 2.5 mint incubation period. This reflection density is directly proportional to the concentration of C-reactive protein in the sample

RESULTS

The main objective of this clinical study is to confirm the usefulness of C-Reactive protein as gingival crevicular fluid biomarkers, analyses characteristic of the active phases of periodontitis could prove valuable in identifying patients with enhanced disease susceptibility, identifying sites with active disease, predicting sites that will have active disease in the near future and/or serving as surrogate endpoints for monitoring of therapy.

Gingival index

Analysis of gingival index at base line revealed mean score of 1.530±0.250, mean gingival score reduced to 0.920±0.250 at end of 14th day. When comparison was done between baseline and 14th day the‘t’ value was found to be 6.350 ($p<0.01$) indicating a highly significant difference and mean value further reduced to 0.540±0.190 (45th day) which when compared to the baseline reading was found to be highly significant (‘t’ value 11.265 & $p<0.01$). Also when compare with readings of 14th day and 45th day the‘t’ value was 6.350 ($p<0.01$) and 5.483 ($p<0.01$) respectively was found to be highly significant.

When we compare between the gingival index from baseline to 14th day the improvement was 39% and at 45th day it was 64 %.(Ttable 1).

Bleeding index

Analysis of bleeding index at baseline revealed mean score of 1.539±0.500, mean gingival score reduced to 0.768±0.255at end of 14th day. When comparison was done between baseline and 14th day the‘t’ value was found to be 6.144 ($p<0.01$) indicating a highly significant difference and mean value further reduced to 0.561±0.101 (45th day) which when compared to the baseline reading was found to be highly significant (‘t’ value 8.581 & $p<0.01$). Also when compared with readings of 14th day and 45th day the‘t’ value was 6.144 ($p<0.01$) and 8.581 ($p<0.01$) respectively was found to be highly significant (Figure 4).

When we compared between the bleeding index from baseline to 14th day the improvement was 50% and at 45th day it was 63%. (Table 2) (Figure 5).

Probing pocket depth

The mean score for probing pocket depth at baseline was 6.410 1.220 which reduced to 5.270 0.990 at the end of 14th day and further reduced to 4.730 0.770 at 45 day. On comparison from baseline to 14th day a mean change of 1.140 0.350 with't' value of 3.239 indicated a statistically less significant reduction (P<0.05). While comparing baseline from 14th day to 45th day a mean change of 0.540 0.280 was seen with't' value of 1.929 indicating a statistically non significant reduction at the end of 45th day (p>0.05). From baseline to 45th day the mean change was 1.680 0.320 with't' value of 5.199 indicating a highly significant reduction (p<0.01) (Figure 6).

When we compared between the probing pocket depths from baseline to 14th day the improvement was 17%, from baseline to 45th day was 26% and from 14th day to 45th day improvement was 10%. (Table 3) (Figure 7).

Clinical attachment level (CAL)

The mean score for clinical attachment level at baseline was 4.470 1.300 which reduced to 3.330 1.090 at the end of 14th day and further reduced to 2.740 0.770 at 45th day. On comparison from baseline to 14th day a mean change of 1.140 0.380 with't' value of 3.003 indicated a statistically less significant reduction (P<0.05). While comparing baseline from 14th day to 45th day a mean change of 0.590 0.300 was seen with't' value of 1.971 indicating a statistically less significant reduction at the end of 45th day (p>0.05). From baseline to 45th day the mean change was 1.730 0.340 with't' value of 5.123 indicating a highly significant reduction (p<0.01).

When we compared between the clinical attachment losses from baseline to 14th day the improvement was 25%, from baseline to 45th day was 38% and from 14th day to 45th day improvement was 17%. (Table4)

C - reactive protein level (CRP)

The mean score for C - reactive protein level at baseline was 5.235 3.118 which reduced to 1.810 1.128 at the end of 14th day and further reduced to 0.000 0.000 at 45 day.[46] on comparison from baseline to 14th day a mean change of 3.426 0.742 with't' value of 4.620 indicated a statistically highly significant reduction (P<0.01). While comparing baseline from 14th day to 45th day a mean change of 1.810 0.252 was seen with't' value of 7.173 indicating a statistically highly significant reduction at the end of 45th day (p<0.01). From baseline to 45th day the mean change was 5.235 0.697 with 't' value of 7.508 indicating a highly significant reduction (p<0.01).

When we compared between the C - reactive protein levels from baseline to 14th day the improvement was 65%, from baseline to 45th day was 100% and from 14th day to 45th day improvement was 100%. (Table 5)

DISCUSSION

Epidemiological studies on oral diseases indicate that periodontal disease is a worldwide health problem and one which cannot be arrested by current professional man power levels. Prevention of periodontal destruction rather than therapy should be the goal of dental professionals. Thus valid identification of person at risk would facilitate to direct therapeutic efforts where they are most needed at an early stage disease.[5]

Joshipura et al., (1996)[6] in their prospective cohort study showed that tooth loss may be associated with a increased risk of Chronic Heart Disease, primarily among those with a positive periodontal disease history, diet was only a small mediator of this association. Recent studies have also shown that chronic periodontal disease may be a risk factor for heart disease. (Beck et al 1996),[7] renal disease, diabetes mellitus, respiratory disease, low birth weight and preterm delivery and these are also related with altered levels of C-Reactive protein.

Poor oral hygiene and periodontal or periapical infections may induce bacteremia even in the absence of dental procedures. Bacteremia may also result in activation of the hepatic acute response (Acute Phase Response). This response includes elevation of fibrinogen, C - reactive protein, heptoglobin, α_1-antitrypsin and other components of acute-phase response. There are now considerable data indicating that periodontal infections results in activation of the acute-phase response and elevation of certain serum markers. (Gore et al., 1998)[8]

Ridker et al., [9]mentioned that mild elevations in C - reactive protein placed an individual at risk for both myocardial infarction and peripheral artery disease. The rapid rise of C-Reactive protein in serum following exposure to IL-1 which is a potent bone resorber also found in gingival Crevicular fluid (Mergerhagen 1984), made the search for elevated C-Reactive protein levels in inflamed periodontal tissues of chronic heart disease patients.

Thus the primary aim of this study was to quantitatively estimate the C - reactive protein level in gingival Crevicular fluid samples and effect of Non surgical Therapy on C - reactive protein level in patients with chronic periodontitis.

The present study included a total number of 60 patients between age group of 35 to 55 years. Patient included were suffering from chronic adult periodontitis with pocket depth ≥ 5 mm with radiographic evidence of bone loss and were free from any systemic illness. Patients were excluded if they had undergone oral prophylaxis or taken antibiotics 6 month prior to inclusion for this study. Pregnant and nursing patients were excluded. At first appointment all the clinical variables were noted. Oral hygiene instruction was given and scaling & root planning was performed on both experimental and control group.
The gingival index and bleeding index of experimental period i.e. on days 0, 14th and 45th was recorded and compared. Results showed a highly significant change with regard to improvement in the gingival inflammation and reduced bleeding on probing from the baseline when compared to the 14th day and 45th day. However change was less significant between 14th day and 45th day. The definite reductions exhibited could be due to regular follow up visits and reinforcement of oral hygiene instruction followed by the patient throughout the study period. These results could be correlated with similar findings observed by various other studies. [10, 11](Graph 1 & 2).

Gains in clinical attachment levels and reduction in pocket probing depth are the most common parameters used to measure clinical improvement. There was a significant decrease in the clinical attachment loss between baseline and 45 days which was found to be 38%. Statistically significant reduction in the probing pocket depth was also observed at the end of 45th day which was 26%. These findings are in agreement with other studies by Persson et al.[12] and Adonogianaki et al.[10]

Our study demonstrated a statistically significant increased gain in the attachment levels. On comparison from baseline to 45th day the mean change was 1.730 0.340 with‘t’ value of 5.123 indicating a highly significant reduction ($p<0.01$), this finding is in correlation to the other studies done by Persson et al.[12] and Adonogianaki et al.[10]. (Graph 3 & 4).

The primary thrust of this study was to evaluate C - reactive protein in gingival Crevicular fluid which is potential indicator of disease activity. The mean score in this study for C - reactive protein level at baseline was 5.235 3.118 which reduced to 1.810 1.128 at the end of 14th day and further reduced to 0.000 0.000 at 45 day. On comparison from baseline to 14th day a mean change of 3.426 0.742 with‘t’ value of 4.620 indicated a statistically highly significant reduction ($P<0.01$). While comparing baseline from 14th day to 45th day a mean change of 1.810 0.252 was seen with‘t’ value of 7.173 indicating a statistically highly significant reduction at the end of 45th day ($p<0.01$). From baseline to 45th day the mean change was 5.235 0.697 with‘t’ value of 7.508 indicating a highly significant reduction ($p<0.01$).

The improvement from baseline to 14th day the improvement was 65%, from baseline to 45th day was 100%. (Graph 5).This can be attributed to the fact that the C-Reactive proteins are produced in inflammatory conditions[9, 13, 14, 15] and once the inflammation subsided due to treatment (scaling and Root Planning) the C - reactive protein levels decreased. Present studies have demonstrated that the extent of increase in C-reactive protein levels in periodontitis patient depends on severity of disease and elevation of C-reactive protein was associated with the presence of periodontopathic bacteria.[16] However it should be noted that C-reactive protein levels of patients tended to be higher at baseline and declined at reassessment suggesting that destructive periodontal disease are treatable and it may be possible to lower C-reactive protein value through effective management of destructive periodontal disease.[17]

SUMMARY AND CONCLUSION'S

C - Reactive protein level makes more significant in Gingival Crevicular Fluid C and confirms the underlying inflammatory component of the disease activity in chronic periodontitis.

Non-surgical periodontal treatment was effective in reducing the levels of C-Reactive protein in Gingival Crevicular fluid and this study data support the hypothesis that levels of gingival crevicular fluid biomarkers specific for three aspects of periodontitis—Degree of inflammation, collagen degradation and bone turnover—correlate with the clinical features of periodontal disease and suggest that elevated gingival crevicular fluid levels of C-Reactive protein are candidate biomarkers of periodontal disease.

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Legends:

Figure 1: C-reactive protein kit

Figure 2: GCF collection with micropipette

Figure 3: Occlusion stent with grooves

Figure 4: Gingival bleeding index at baseline

Figure 5: Gingival bleeding index at 45th day

Figure. 6: Probing pocket depth at baseline

Figure 7: Probing pocket depth at 45th day

Tables

Table 1: Mean gingival Index before and after treatment

Gingival Index	Mean ± SD	% Change from Baseline	P Value
BASELINE	1.530±0.340		
15th DAY	0.920±0.250	39%	0.000
45th day	0.540±0.190	64%	0.000

Table 2: Mean Sulcus bleeding Index before and after treatment

Table 3: Mean Probing pocket depth level before and after treatment

Probing Pocket depth	Mean ± SD	% Change from Baseline	P Value
BASELINE	6.410±1.220		
15th DAY	5.270±0.990	17%	0.002
45th day	4.730±0.770	26%	0.000

Table 4: Mean Clinical attachment level before and after treatment

Bleeding Index	Mean ± SD	% Change from Baseline	P Value
BASELINE	1.539±0.500		
15th DAY	0.768±0.255	50%	0.000
45th day	0.561±0.101	63%	0.000

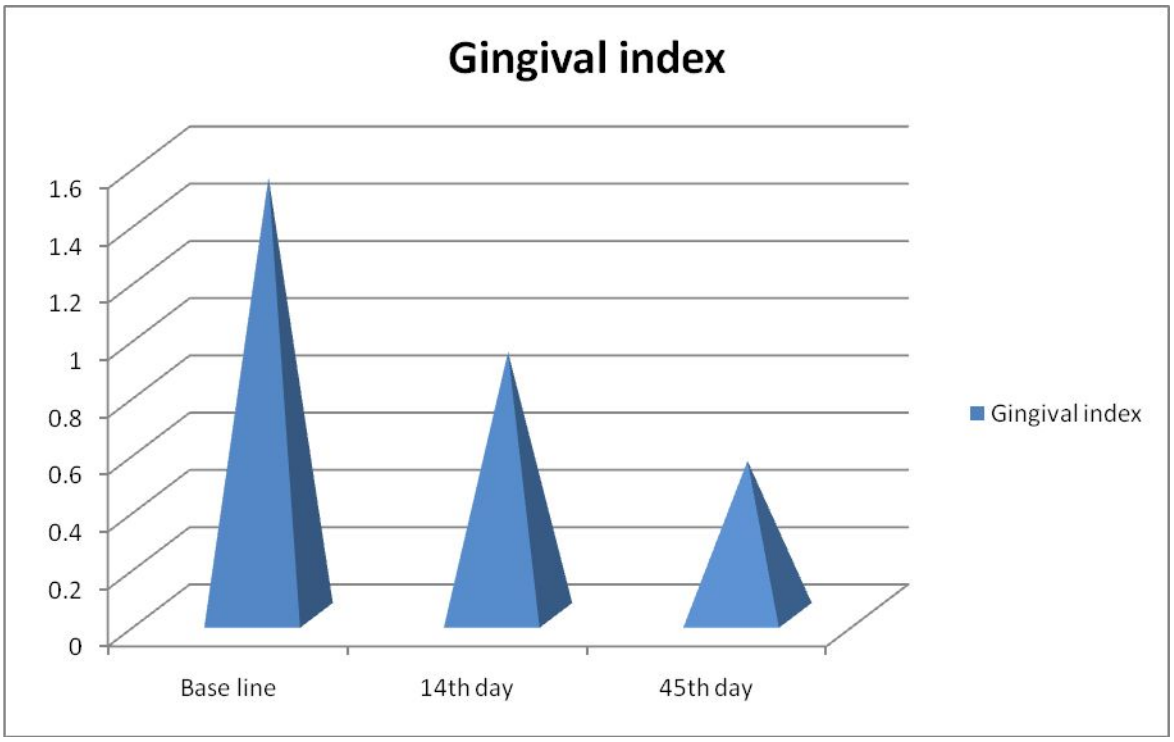
Clinical attachment level	Mean ± SD	% Change from Baseline	P Value
BASELINE	4.470±1.300		
15th DAY	3.330±1.090	25%	0.005
45th day	2.740±0.770	38%	0.000

Table 5: Mean C - reactive protein level before and after treatment

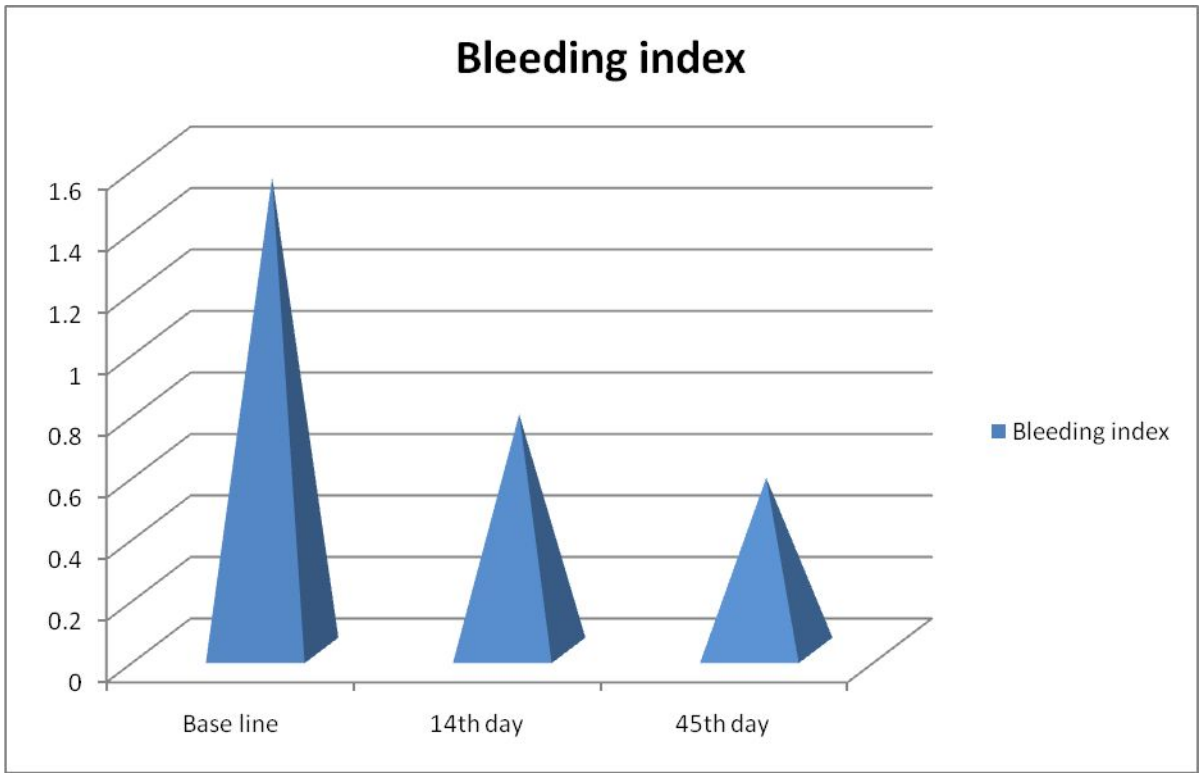
C-Reactive Protein level	Mean ± SD	% Change from Baseline	P Value
BASELINE	5.235±3.118		
15th DAY	1.810±1.128	65%	0.000
45th day	0.000±0.000	100%	0.000

Graphs

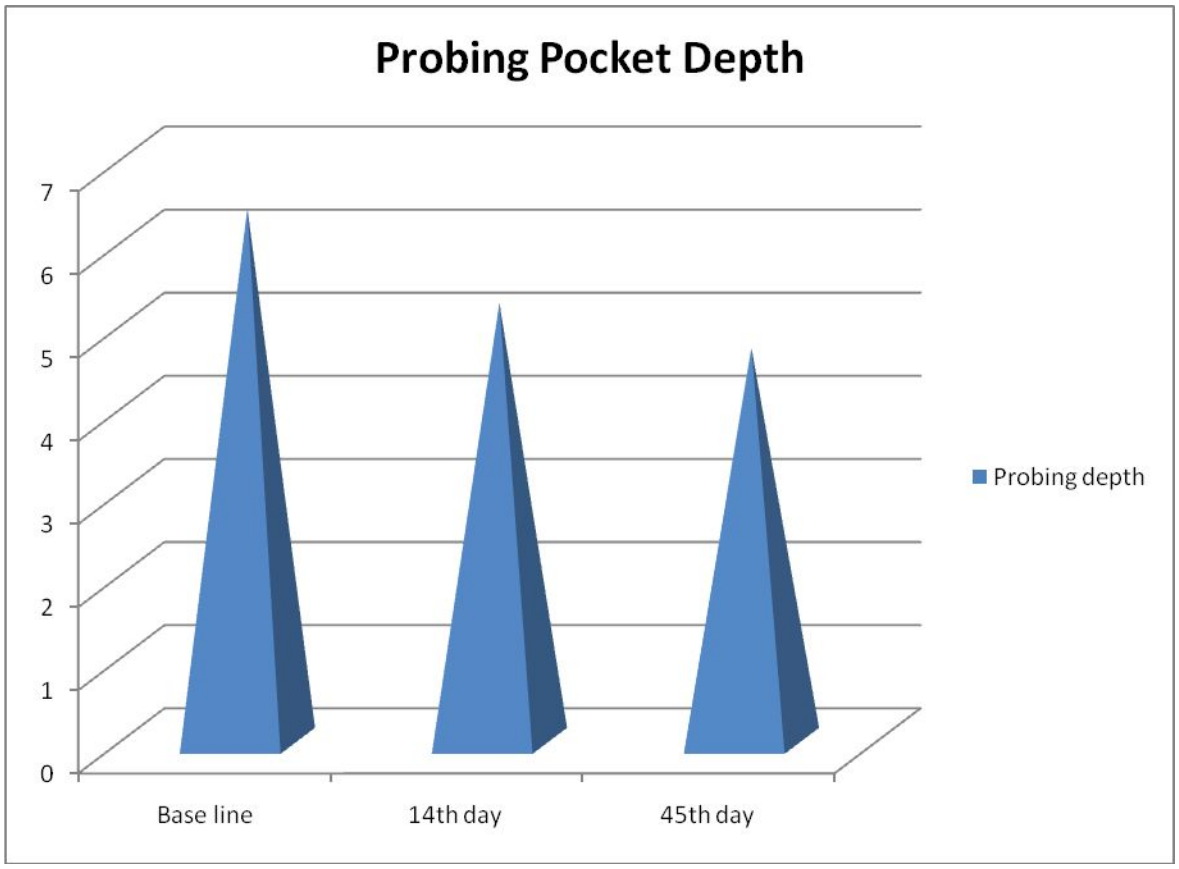
Graph 1. Comparison between Gingival Index Scores for Baseline, 14th day and 45th day



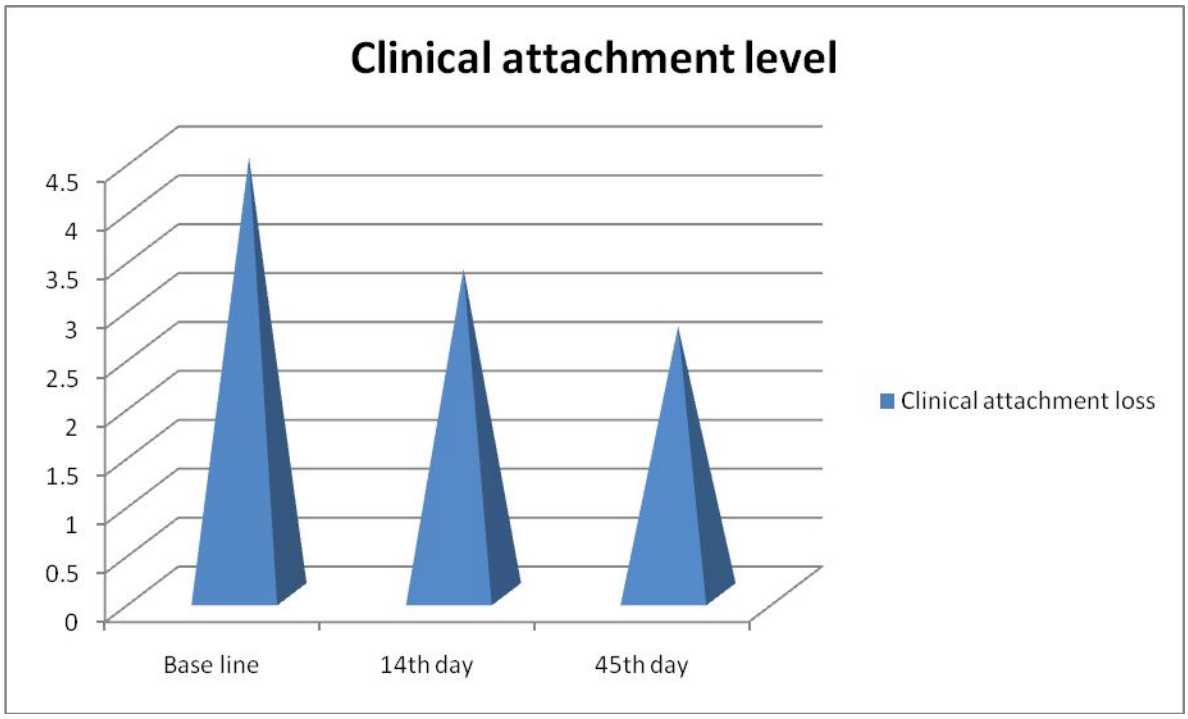
Graph 2. Comparison between Sulcus Bleeding Index Scores for Baseline, 14th day and 45th day



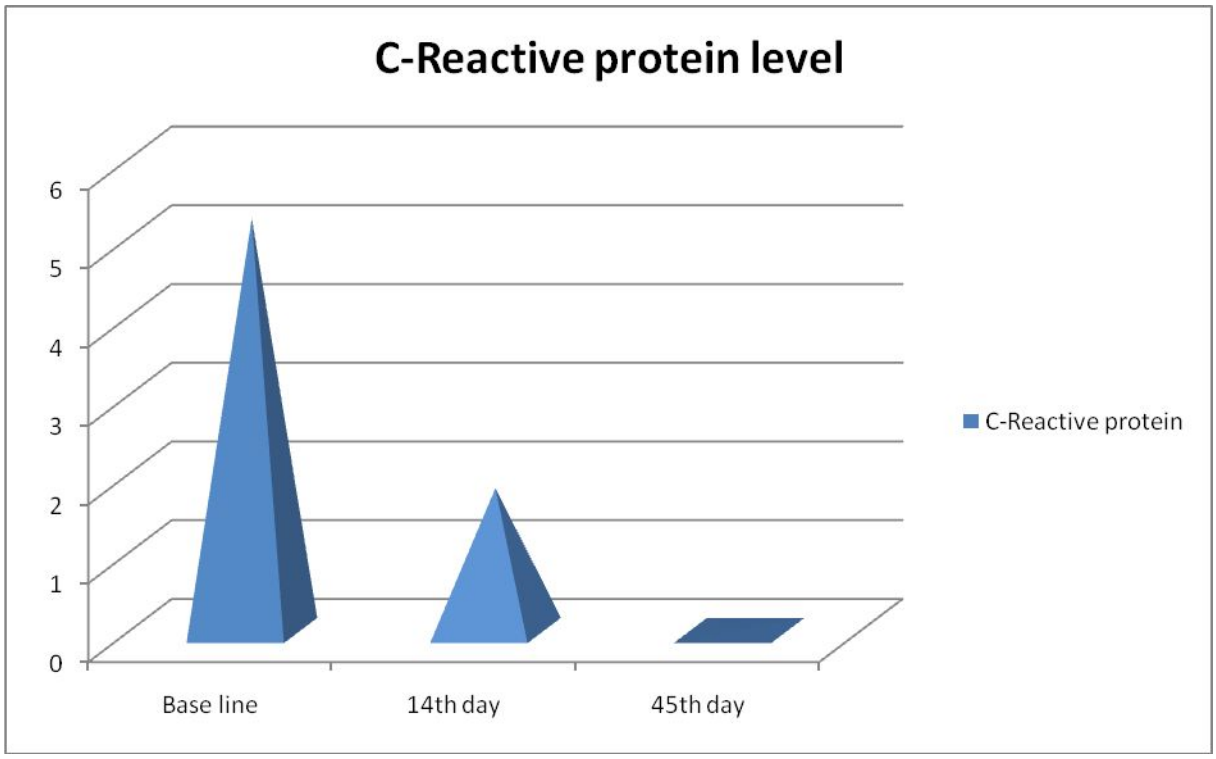
Graph 3. Comparison between Probing Pocket Depth for Baseline, 14th day and 45th day



Graph 4. Comparison between Clinical Attachment Level for Baseline, 14th day and 45th day



Graph 5. Comparison between C - reactive protein level for Baseline, 14th day and 45th day


C-Reactive protein level
6
5
4
3
2
1
0
Base line
14th day
45th day
C-Reactive protein