ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE 1 THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS

A Dissertation submitted in

partialfulfillment of the requirements

for the degree of

MASTER OF DENTAL SURGERY

BRANCH – II

PERIODONTICS

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

Chennai – 600 032

2016 - 2019

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This is to certify that the Dissertation entitled**"ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE 1 THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS"**is a bonafide work done by **Dr.J.HEMALATHA**, Post Graduate student (2016–2019) in the Department of Periodontics, under the guidance of **Dr. K MALATHI**, HOD and Professor (Guide), Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai – 600 003.

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PLACE OF STUDY	**TAMIL NADU GOVERNMENT DENTAL COLLEGE AND HOSPITAL, CHENNAI – 600 003.**
DURATION OF THE COURSE	**3 YEARS**
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I hereby declare that this dissertation titled **"ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE 1 THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. K. MALATHI, M.D.S,** Head of the Department and Guide, Department of Periodontology, TamilNadu Government Dental College and Hospital, Chennai -600003.

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This agreement herein after the "Agreement" is entered into on this day ----------between the Tamil Nadu Government Dental College and Hospital represented by its **Principal** having address at Tamil Nadu Government Dental College and Hospital, Chennai – 600 003, (hereafter referred to as, 'the college')

And

Dr. J.HEMALATHA, aged 38 years currently studying as **Post Graduate student** in Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai – 600 003, (hereafter referred to as 'the PG student and principal investigator')

And

Mrs. Dr.K MALATHI aged 50years working as **HOD and Professor** in Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai (herein after referred to as the 'Co- Investigator'),

Whereas the PG student as part of his curriculum undertakes this research on **"ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE 1 THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS"** for which purpose the Co-investigators and the college shall provide the requisite infrastructure based on availability and also provide facility to the PG student as to the extent possible as a principal investigator.

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Sub: IEC review of the research proposals,

Title of the work: **Estimation of serum albumin levels before and after phase I therapy in patients with Chronic Periodontitis**

Investigator: **J. Hemalatha**
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Department : **Department of Periodontics**
Tamil Nadu Govt. Dental College & Hospital ,

Thank you for submitting your research proposal , which was considered at the Institutional Ethics Committee meeting held on the 30-09-2016, at TN Govt. Dental College. The documents related to the study referred above were discussed and reviewed.

The decision of the members of the committee , the secretary and the Chairperson IEC of TN Govt. Dental College is here under:

Approved	Approved and advised to proceed with the study
Approved with suggestions	----------

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ABSTRACT

BACKGROUND

Many studies have been carried out till date that talk about severity of chronic periodontitis but none correlates with Albumin level in blood before and after instituting phase I periodontal therapy. Since albumin is an important biomarker for systemic inflammation, it must be interesting to check whether periodontal health status has anything to do with the albumin level in blood. Hence, present study was carried out to determine albumin level in blood before and after instituting phase I periodontal therapy and to find out whether it has any effect on systemic health.

AIM

The aim of this study is to determine whether there is an alteration in the level of serum albumin in patients with chronic periodontitis before and after phase I therapy.

METHODS

Fifteen patients with chronic periodontitis and 15 systemically healthy subjects without periodontal disease were selected. Serum albumin levels in individuals with healthy periodontium (control group) and chronic periodontitis (study group) were compared. Serum albumin levels and clinical periodontal parameters (pocket depth, clinical attachment level, gingival index, bleeding index and plaque index) were measured at baseline and 3 months after non-surgical periodontal treatment in chronic periodontitis patients. Data were analyzed with descriptive statistical methods (means ± standard deviations). Independent samples t-test was used to compare serum albumin levels and clinical variables between the test and control groups. Paired samples t-test was used in the test group for comparisons before and after treatment. Statistical significance was set at $P < 0.05$.

RESULTS

The mean serum albumin levels in patients with chronic periodontitis (3.956 ± 0.049 mg/dL) were significantly less than that in periodontally healthy subjects (4.73 ± 0.052mg/dL). Three months after periodontal treatment, the serum albumin level increased significantly (4.567 ± 0.017 mg/dL) and approached the levels in periodontally healthy subjects ($P < 0.05$).

CONCLUSION

The decrease and increase in serum albumin levels with periodontal disease and periodontal treatment respectively indicated an inverse relationship between serum albumin levels and chronic periodontitis.

Keywords: Albumin, chronic periodontitis, inflammation, scaling, root planing

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LIST OF ABBREVIATIONS

Ig	**Immunoglobulin**
PPD	**Probing Pocket Depth**
CAL	**Clinical Attachment Level**
MWF	**Modified Widman Flap**
BOP	**Bleeding On Probing**
PI	**Plaque Index**
GBI	**Gingival Bleeding Index**
CRP	**C Reactive Protein**
HD	**Hemo Dialysis**
RBC	**Red Blood Cells**
TNF	**Tumour Necrosis Factor**
GCP	**Generalized Chronic Periodontitis**
IL	**Interleukin**
CEJ	**Cemento Enamel Junction**
ESRD	**End Stage Renal Disease**
ALT	**Alanine Transaminase**
ALP	**Alkaline phosphatase**
SGA	**Subjective Global Assessment Score**
SRP	**Scaling And Root Planing**
AAP	**American Association Of Periodontology**
MCV	**Mean Corpuscular Volume**
MCH	**Mean Corpuscular Hemoglobin**
MCHC	**Mean Corpuscular Hemoglobin Concentration**

INTRODUCTION

Periodontitis is a destructive chronic infection of the gingiva, periodontal ligament and bone that supports the teeth.[1] The primary clinical features of periodontitis include gingival inflammation, periodontal pocketing, clinical attachment loss and alveolar bone loss. Chronic periodontitis is considered to start as plaque induced gingivitis, a reversible condition that is left untreated may develop into chronic periodontitis. Chronic periodontitis distinguished by loss of attachment and bone is regarded as irreversible.

Periodontitis results in the formation of many inflammatory mediators like C -reactive proteins. Also this will stimulate antibody formation like IgG. A significant association between serum albumin concentration and IgG has been reported [2].C -reactive protein may be used to identify the presence of inflammation in individuals with a lower serum albumin concentration. [3]

Serum albumin is a negative acute phase protein and serum albumin might be the practical marker of general health status **(Phillips et al).**[4] Chronic inflammatory conditions such as liver diseases and renal diseases reduces the serum albumin levels. **(Herrmann et al).**[5] Serum albumin levels remain virtually unchanged even in the presence of protein calorie malnutrition in otherwise healthy individuals until terminal starvation. Hence, serum albumin concentration should be a criterion, which indicates general health.

Recently report says that the number of untreated teeth was a significant factor associated with serum albumin concentration in elderly. Therefore, it is suggestive that oral disease burden might be monitored by the levels of serum albumin.[6] Inflammation and malnutrition both reduce the serum albumin concentration by

decreasing its rate of synthesis. This suggests that the periodontal disease severity might be indicated and monitored by the levels of serum albumin. Therefore, serum albumin can be used as a risk predictor for periodontal disease.

AIM

The aim of this study is to determine whether there is an alteration in the level of serum albumin in patients with chronic periodontitis before and after phase I therapy.

OBJECTIVES

- To measure serum albumin levels in control group and in study group at

  T0: Before phase I therapy,

  T1: After 3 months of Phase I therapy.
- To correlate the clinical parameters in the control, with study group before and after phase I therapy along with serum albumin levels.
- To compare serum albumin levels in individuals with healthy periodontium (control group) and chronic periodontitis (study group).
- Estimation of serum albumin levels before and after phase I therapy in patients with chronic periodontitis.

REVIEW OF LITERATURE

Periodontitis is a chronic inflammatory condition with a polymicrobial infectious in nature, which destroys the tooth-supporting tissues and results in attachment and bone loss. After accumulation of bacterial biofilm on the external tooth surfaces, periodontitis is induced as an inflammatory–immune process and results in the production of proinflammatory cytokines, local and systemic inflammatory responses, and formation of periodontal pockets.[7]

The diagnosis of periodontitis relies on clinical measurements, including pocket depth, clinical attachment level, and radiographic findings. However, such measurements have limited efficacy in periodontal diagnosis in many cases because these parameters mainly indicate previous periodontal disease rather than the current disease activity[8].It is necessary to develop new diagnostic tests so that it would be possible to diagnose active disease, predict future disease activity and evaluate response to periodontal treatment after clinical recovery of patients from periodontitis. Advances in diagnosis of periodontal diseases include moving toward diagnostic techniques that can evaluate periodontal disease risk by measuring biomarkers. Biomarkers are produced by both healthy individuals and those with specific systemic conditions. They are the molecules that is used to monitor the health status, disease initiation and response to treatment.[9]

Acute phase proteins are some of these biomarkers. The acute phase reaction consists of a group of metabolic and systemic changes that are induced by an inflammatory stimulus. The most critical components of this reaction are acute phase proteins that consist of a heterogeneous group of serum proteins synthesized by the liver.[10]

A change in the concentration of acute phase proteins, as a patho-physiologic phenomenon, occurs subsequent to a wide range of disorders, including inflammation, trauma, infection and infarction. Despite their name, such proteins are expressed due to both acute and chronic inflammation.[11]

Acute phase proteins are categorized into two groups:

1. Positive acute phase proteins such as C-reactive protein, serum amyloid-A, pentraxin-3, ceruloplasmin and ferritin, whose serum levels increase during the inflammatory response.

2. Negative acute phase proteins are proteins such as albumin and transferrin, whose serum levels decrease with an increase in inflammation severity.[12]

SERUM ALBUMIN

Serum albumin is the most abundant protein in human blood plasma, it constitutes about half of serum protein .It is produced in the liver. It is soluble and monomeric. Albumin transports hormones, fatty acids and other compounds, buffers, and maintain oncotic pressure, among other functions.

Albumin is synthesized in liver as preproalbumin, which has an N- terminal peptide that is removed before the nascent protein is released from the rough endoplasmic reticulum. The product, proalbumin, is in turn cleaved in golgi vesicles to produce the secreted albumin.

SERUM ALBUMIN LEVELS

The reference range for albumin concentration in serum is approximately 35-50g/L (3.5-5.0g/dl)[13].It has a serum half-life of approximately 20 days.It has a

molecular mass of 66.5 KDa. For children less than 3years of age, the normal range is broader, 2.9 – 5.5 g/dl.

Low albumin (hypoalbuminemia) may be caused by liver disease, nephrotic syndrome, burns and protein losing enteropathy, malabsorption, malnutrition[14], late pregnancy, genetic variation and malignancy.

High albumin (hyperalbuminemia) is almost always caused by dehydration.[15] In some cases of retinol (vitamin A) deficiency, the albumin levels can be high values .This is because retinol causes cells to swell with water .This swelling also likely occurs during treatment with 13-cis retinoic acid .In lab experiments it has been shorn that all- trans retinoic acid down regulates human albumin production. Hyperalbuminemia has also been associated with high protein diets.[16]

FUNCTIONS

- Maintains oncotic pressure
- Transports thyroid hormones
- Transport other hormones in particular ones that are fat – soluble
- Transports fatty acids (" free " fatty acids) to the liver and to monocytes for utilization of energy
- Transports unconjugated bilirubin
- Transports many drugs ; serum albumin can affect the half – life of drugs
- Competitively binds calcium ions
- Serum albumin, which is a negative acute- phase protein , that is down – regulated in inflammatory states. As such, it is not a valid marker of nutritional status; rather it is a marker of inflammatory state.

- Prevents photo degradation of folic acid

MEASUREMENT

Serum albumin is commonly measured by recording the change in absorbance upon binding to a dye such as bromocresol green or bromocresol purple.[17]

PROPERTIES

Albumin is a globular, water soluble unglycosylated serum protein of approximate molecular weight of 65,000Daltons. Albumin is negatively charged. The glomerular basement membrane is also negatively charged in the body, some studies suggest that this prevents filtration albumin in urine. According to this theory, the negative charge plays a major role in the selective exclusion of albumin from the glomerular filtrate. A defect in this property results in nephrotic syndrome leading to loss of albumin in urine. Nephrotic syndrome patients are sometimes given albumin to replace lost albumin.

STRUCTURE

The general structure of albumin is characterized by several long alpha helices allowing it to maintain a relatively static shape, which is essential for regulating blood pressure.

Serum albumin consists of eleven distinct binding domains for hydrophobic compounds. One hemin and six long –chain fatty acids which can bind to serum albumin at the same time.[18]

TYPES

Human serum albumin

Bovine serum albumin (often used in medical molecular biology labs)

THERAPEUTIC USES

Human albumin solution is available for medical use, usually at concentration of 5-25% .Human albumin often used to replace blood volume in trauma, burns and surgery patients.

Human serum albumin is used as a component of a frailty index.[19]

It has been shown to give better results than other fluids when used to replace volume, but is frequently used in conditions where loss of albumin is a major problem, such as liver disease with ascites.

Human serum albumins have been used to potentially reverse drug / chemical toxicity by binding to free drug / agent.

GLYCATION

Human blood protein like haemoglobin[20] and serum albumin[21] may undergo a slow non – enzymatic glycation, mainly by formation of Schiff base between amino groups of lysine residues and glucose molecules in blood. This reaction is inhibited in the presence of antioxidant agents.[22] Elevated glycoalbumin is absorbed in diabetes mellitus.[23]

Glycation has potential to alter the biological structure and function of serum albumin protein.[24] Moreover, the glycation can result in formation of Advanced Glycation End Products, which leads to tissue damage via alteration of the structures

and function of tissue proteins and generation of reactive oxygen intermediates. They also interfere with the normal product of nitric oxide in cell.[25]

OXIDATION

The albumin is the predominant protein in most body fluids, its Cys34 represents the largest fraction of free thiol exists in both reduced and oxidized forms[26]. In plasma of healthy young adults , 70-80% of total HSA contains the free sulfhydryl group of Cys34 in reduced form or mercaptoalbumin[27] .However in pathological states characterized by oxidative stress and during aging process ,the oxidized form could predominate[28].The albumin thiol reacts with radical hydroxyl, hydrogen peroxide and reactive oxygen species as peroxynitrite and have been shown to oxidize Cys34 to sulfenic acid derivative ,it can be recycled to mercaptoalbumin ,however at high concentration of reactive species leads to irreversible oxidation to sulfonic acid affecting it structure[29].Presence of reactive oxygen species , can induce irreversible structural damage and alter protein activities.

LOSS VIA KIDNEY

In healthy kidney, albumin size and negative electric charge exclude it from excretion in the glomerulus. This is not always the case, as in some diseases including diabetic nephropathy, which can sometimes be a complication of uncontrolled or of longer term diabetes in which protein can cross the glomerulus. The lost albumin can be detected by a simple urine test. Depending on the amount of albumin loss, a patient may have normal renal function, microalbuminuria or albuminuria.

SERUM ALBUMIN IN RELATION TO PERIODONTITIS

Hermann et al. (1992) detected that many conditions, such as inflammatory states, liver diseases, and renal diseases, have been indicated to reduce serum albumin levels.[30] Serum albumin is the main protein synthesized by the liver. In elderly individuals it seems to be imperative that the impaired dentition status and a lean life style along with the possibility of compromised systemic health status would reflect within the values of serum albumin concentration.[31]

Serum albumin is a negative acute phase protein supports the contention that serum albumin is a marker of inflammation.[32] Chronic diseases are associated with inflammation and the release of inflammatory cytokines such as interleukin-1, interleukin-6, and tumor necrosis factor-α, which cause a decrease in serum albumin **(Schalk et al. 2004).**[33] Moreover, malnutrition may also be monitored by means of serum albumin concentration **(Don and Kaysen 2004).** Therefore, albumin concentration is associated with nutrition and inflammation **(Kaysen et al. 2002).**

There has also been a linkage of serum albumin level and mortality rate. Investigations by **Corti et al.**[34] have reported graded increase in mortality rate with decreasing serum albumin levels. However, it seems more evident that serum albumin levels below 4 g/dL have higher mortality rate.

Shibata et al. reported significantly different 10-year survival rate with a quartile of serum albumin levels. Therefore, the periodontal disease status has a substantial influence not only on the subject's serum albumin levels but also on general health aspects.[35]

Iwasaki et al evaluated albumin, a negative acute phase protein, in patients with periodontitis and concluded that there might be an inverse relationship between periodontitis and albumin serum levels.[36]

Kshirsagar et al [37] studied levels of serum albumin and CRP among patients on HD. In this study, patients with severe periodontitis had low serum albumin levels (odds ratio 8.20; 95% confidence interval 1.61-41.82; p = 0.01) compared with individuals without severe periodontitis disease after adjustment for several factors. There was no association of CRP levels and severity of the disease.

Shirmohamadi et al 2016 [38]The mean transferrin serum level in patients with chronic periodontitis (213.1 ± 9.2 mg/dL) was significantly less than that in periodontally healthy subjects (307.8 ± 11.7 mg/dL). After three months periodontal treatment, the transferrin serum level increased significantly (298.3 ± 7.6 mg/dL) and approached the levels in periodontally healthy subjects ($P < 0.05$).

There is decrease in transferrin serum levels with periodontal disease and increased after periodontal treatment, respectively, indicated an inverse relationship between transferrin serum levels and chronic periodontitis.

Sawant et al[39] don**e** a study to evaluate the role of serum albumin and liver enzymes in the cause effect relationship of chronic periodontitis .And found that there is Significant reduction in serum albumin levels in chronic periodontitis patients. And concluded there was significant inverse association between serum albumin and clinical attachment loss in patients with chronic periodontitis, also found that there was significant increase in serum ALT and ALP levels in chronic periodontitis patients as compared to healthy subjects. Such a study will help the clinician for dental referral of the patient with high ALT and ALP levels.

Navkiran Kaur[40] conducted the study to evaluate the relationship between periodontal health status and serum albumin levels. The findings of this clinical trial suggest an inverse relationship between the serum albumin concentration and chronic periodontal disease.

In a study by **Bhuvan et al**[41] it was found that Periodontal condition is related to the control of glucose level among patients with type 2 diabetes and periodontal disease. Periodontal treatment can effectively reduce the level of glucose and Glycated albumin as well as it improves the periodontal condition in type-2 diabetes patients with periodontal disease.

In a study by **Veisa et al**[42] on Albumin as a Prognostic Factor for Malnutrition and Inflammation in Chronic Kidney Disease, with main goal to evaluate, for the first time, the correlation between albumin level, periodontal disease and inflammation in a cohort of haemodialysis patients. It was found that in HD patients, albumin and nutritional status (evaluated by SGA score) were associated with a significantly increased death risk. Further evidence is needed in order to support inflammation markers as a long term predictor for decline in ESRD patients.

In a study by **Pimpale S et al** to correlate between periodontal disease and serum albumin concentration in chronic periodontitis patients and suggested that serum albumin concentration is an important risk indicator in chronic periodontitis patients.

NON-SURGICAL THERAPY:

The main goal of the treatment of patients with periodontitis is to establish proper infection control, i.e, to reduce the bacterial load below the individual threshold level for disease. Cause-related periodontal therapy is usually the initial phase of periodontal treatment. The aims are to eliminate supra- and sub-gingival bacterial deposits and to prevent their recurrence **(Haffajee et al. 1997)**.[43] even in the most severe cases of periodontal disease, cause-related periodontal therapy most often precedes surgical therapy. This is done so that the periodontal infection is

reduced and the overall tissue quality is improved prior to surgery. This procedure may also limit the areas requiring surgery.

The aims can be achieved by the following procedures/measures:

- Motivate the patient to understand periodontal disease and the importance of co-operation in order to combat the disease. Optimal treatment results and stable long term conditions depend on optimal self-performed oral hygiene and compliance towards regular maintenance visits.
- Provide the patient with custom-made oral hygiene instruction and reassess the results from time to time and reinforce the technique, if necessary. Maintaining optimal personal hygiene is paramount because at least one study showed that instrumentation alone without improving patient's plaque control may lead to microbial repopulation of the instrumented sites shortly after scaling and root planing **(Magnusson et al. 1984)**[44]

Non-surgical periodontal therapy include scaling and root planing to remove bacterial plaque and calculus subgingivally by mechanical means, using either manual instruments such as hand scalers/curettes or machine-driven instruments such as sonic or ultrasonic scalers. **Badersten et al (1981)[45], Torfason et al (1979)**[46] have shown that both instrumentation approaches could achieve similar results, but according to **Leon & Vogel (1987)[47]** ultrasonic instruments might be more suitable in furcation areas.

Although the word "planing" means to remove substance for achieving a smooth surface, **Nyman et al (1988)**[48] showed that it was not necessary to plane the root surfaces until smooth or to remove the so called "diseased/ contaminated" cementum**.** A clinical study by **Oberholzer & Rateitschak 1996** concluded that the

establishment of a smooth and hard root surface was not a critical factor in periodontal therapy**.**

Non-surgical treatment improves clinical parameters including bleeding on probing, probing depths and probing attachment levels, provided that patients can obtain and maintain a proper plaque control**.**

Singletary et al 1982[49]**, Greenewell et al 1984** [50]**, Lavanchy et al 1987**[51] reported a significant reduction in gingival inflammation 1-3 months after phase I therapy. **Hughes & Caffesse 1978**[52] demonstrated that reduction of probing depth following mechanical instrumentation results from a combination of gain in clinical attachment and increase in gingival recession**. Proye et al 1982**[53] noted recession after one week and a gain of clinical attachment by 3 weeks after phase I therapy. After a single episode of scaling and root planing, pockets were reduced to 1.36 mm. This consisted of 0.84 mm recession and 0.52 mm attachment gain.

Morrison et al 1982[54] in another examination of data from the previously mentioned 8-year longitudinal study analyzed the effect of gingivitis scores on probing depth and attachment levels. For pockets 1 to 3 mm and 4 to 6 mm there was no difference in pocket reduction maintenance. For attachment there was no significant difference in 1 to 3 mm probing depths and in 4 to 6 mm pockets, lower gingivitis scores had better gain the first 2 years but thereafter no difference was recorded. For 7 to 12 mm pockets, the lower gingivitis scores seemed to result in better probing levels and attachment gain for the first 3 years but this was not maintained throughout the experiment. And the severity of gingivitis did not affect the maintenance of pocket depth reduction or clinical attachment levels.

Ramfjord et al 1982[55] reported that deepest sites demonstrated the greatest pocket reduction after instrumentation. They conducted a study in pockets with 1-

3mm, 4-6mm and ≥7mm for 5years and found the greatest pocket reduction in group with ≥7mm pocket depth. But they also found a greater amount of recession in this group.

Cobb 1996[56] reported that for shallow pockets with initial probing depth 1-3mm, the mean reduction of probing pocket depth (PPD) was 0.03mm and loss of probing attachment level (PAL) was 0.3mm. For moderate pockets with initial PPD of 4-6mm, the mean reduction of PPD was about 1.29mm and the PAL gain was 0.55m **.** For deep pockets with initial PPD ≥ 7mm, the mean reduction of PPD was about 2.16mm and the PAL gain was 1.19mm.

The number of sites that bleed on probing also markedly reduces following nonsurgical therapy. **Cobb (2002)**[57] reviewed many studies and found that the mean reduction in bleeding on probing from baseline levels was about 45%.

Non-surgical periodontal therapy has been proved to be effective in controlling the disease in most patients **(Badersten et al. 1984, Claffey & Egelberg 1995)**[58,59] Furthermore, most of the stabilized periodontal conditions can be maintained throughout time if the patients are committed to have supportive periodontal care **(Axelsson & Lindhe 1981, Renvert & Persson 2004).**[60,61]

Philstrom et al. (1981)[62] in a 4-year study utilizing multi-rooted teeth, compared scaling and root planing to modified Widman surgery. Seventeen patients received thorough scaling and root planing as well as oral hygiene instructions. A modified Widman flap was then performed on one half of each subject's dentition. Patients were recalled 3 to 4 times a year for 4 years. The data were separated into 3 groups by initial pocket depth; 1 to 3 mm, 4 to 6 mm, and > 7 mm. Both methods resulted in increased probing depth and loss of attachment in the 1 to 3 mm group, in the 4 to 6 mm group both procedures resulted in reduction in probing depth and

maintenance of attachment levels with the root planing resulting in slightly more gain in attachment. The > 7 mm group showed the greatest reduction in probing depth and gain in attachment with better results in the flap procedures. The results indicate that both procedures were effective in treating moderate to advanced periodontitis. The additional flap procedure tended to result in greater probing reduction and attachment gain for deeper pockets.

Philstrom et al. (1983)[63] in a second report analyzed the 6.5 year results of the previous study. This report concludes that scaling and root planing alone or in combination with modified Widman flap surgery resulted in sustained decreases in gingivitis, plaque, and calculus and neither procedure appears to be superior with respect to these parameters. Seventeen patients diagnosed with moderate-advanced periodontitis were utilized in a split-mouth design study to compare the effects of scaling and root planing alone and combined with modified Widman flap surgery. Data were collected at baseline, 6 months following active therapy and every year up to 4 years, then at 5 1/2 and 6 1/2 years. Probing depth did not change for 1 to 3 mm pockets treated by either scaling or root planning alone or in combination with modified Widman flap surgery. For pockets 4 to 6 mm, both treatment procedures resulted in equally effective sustained pocket reduction. Deep pockets (> 7 mm) were initially reduced more by the flap procedure. After 2 years, no consistent difference between treatment methods was found in degree of pocket reduction. For pockets initially 4 to 6 mm in depth, attachment level was sustained by both procedures. Pockets > 7 mm in depth treated by either procedure resulted in a sustained gain in attachment.

Philstrom et al. (1984)[64] in a third report examined the response of molar and non-molar teeth to scaling and root planing alone or scaling and root planing plus a

flap procedure. At 6 1/2 years, non-molar teeth had an average of about 1.0 mm less probing depth than molar teeth irrespective of type of procedure performed. There was greater probing depth and more apical attachment level on molar than on non-molar teeth treated by either method for 4 to 6 mm pockets. In > 7 mm pockets, the flap resulted in less pocket depth on non-molars than molars, but no difference in the attachment level for either method. Nineteen of the 453 teeth included in the study were extracted throughout the study; 11 of these were extracted after therapy.

Hill et al. (1981)[65] published a 2-year study of scaling and root planing compared to modified Widman surgery. This 90-patient study included multi-rooted teeth. Following a hygienic phase which included scaling and root planing and oral hygiene instructions;, each quadrant was treated by 1 of 4 treatments (pocket elimination, modified Widman flap (MWF), subgingival curettage, and scaling and root planing). Measurements which included pocket depth and attachment levels were taken at the initial exam, after the hygienic phase and 1 and 2 years after treatment. In the 1 to 3 mm crevices there was a slight loss of attachment after all types of treatments. In the 4 to 6 mm pockets there was a significant reduction in probing depth after all modalities with the greatest reductions after pocket elimination and modified Widman flap, and a loss of attachment for pocket elimination and a gain for curettage and scaling. In the > 7 mm pockets there was a significant reduction after all modalities with the greatest reduction after pocket elimination, and no significant differences in attachment results among the 4 methods. None of the surgical modalities had any better effect than scaling and root planing alone in maintenance of periodontal support which was not directly related to reduction in pocket depth.

Cercek et al. (1983)[66] reported results of a 2-year study that compared supragingival plaque control to subgingival plaque control to scaling and root planing. Seven patients with chronic periodontitis were observed during 3 phases of treatment: 1) tooth brushing and flossing; 2) Perio-Aid used subgingivally; and 3) subgingival debridement. Plaque scores ranged from 38 to 99% with a mean of 74% at the initial exam. These scores were reduced to 5 to 15% and were maintained throughout the study. The mean bleeding score of 71.7% was reduced to 40.9% in Phase I, no change in Phase II, and reduced to 23% in Phase III. Deeper sites showed more bleeding than shallower sites throughout the study. The mean probing depth of 4.4 mm was reduced to 4.0 mm in Phase I, no improvement in Phase II, and reduced to 3.2 mm after instrumentation. Probing attachment level showed a mild loss through Phase II, but improved attachment levels were found after instrumentation. An increasing gingival recession was noted during the study. Less effect was derived from patient-performed plaque control, whether supra- or subgingival. The bulk of the effect was derived from professional subgingival instrumentation (SRP).

Badersten et al 1981[45] in a 13-month study of patients with moderate periodontitis compared the effect of hand versus ultrasonic instrumentation on attachment levels of single-rooted teeth. Incisors, canines, and premolars in 15 patients with moderately advanced periodontitis were treated by hand and ultrasonic non-surgical therapy. Improvements in plaque scores, bleeding on probing, decreased probing and attachment levels were similar for both treatment methods. It was shown that shallower sites had a slight loss of attachment while deeper sites showed some improvement.

Badersten et al. 1984A[58] reported 24-month results of a study comparing hand to ultrasonic instrumentation in patients with severe periodontitis. Sixteen

patients with severely advanced periodontal disease were treated by hand or ultrasonic non-surgical therapy. Comparable results were obtained by both methods. It has been shown that the deep probing depths could be successfully treated non-surgically based on probing depth, probing attachment levels, bleeding on probing, plaque, and gingival recession. It was shown that shallower sites were at risk of losing attachment, while the deep sites were more likely to gain attachment. Deeper residual probing sites were more likely to bleed on probing.

Badersten et al. 1984B[67] compared the effect of a single session of scaling and root planing to repeated sessions of scaling and root planing. Incisors, canines, and premolars were studied in 13 patients with severe periodontitis. Teeth were instrumented using ultrasonic instruments, and repeated instrumentation in one side of the jaw was performed after 3 and 6 months. A gradual and marked improvement took place during the first 9 months. There is no differences in results could be observed when comparing the effects of a single versus repeated instrumentation. Deep periodontal pockets in incisors, canines, and premolars may be treated by plaque control and one episode of instrumentation.

Badersten et al (1985A) [68] reported a study of the effect of operator variability on the results of the scaling and root planing procedure. Twenty patients whose dentition displayed generalized severe periodontal destruction were selected for the study. The incisors, canines, and premolars in either the maxilla or the mandible were studied. The periodontal pockets were debrided using either hand and/or ultrasonic instruments under local anesthesia by a periodontist or by 1 of 5 dental hygienists. A split mouth design was used with measurements of dental plaque, bleeding on probing, probing depth, and probing AL recorded at the initial exam and at every third month by an examiner not involved with treatment. The results indicate

that deep periodontal pockets in incisors, canines, and premolars may be successfully treated by plaque control and one episode of instrumentation and that operator variability between highly skilled clinicians is minimal.

Badersten et al. (1985B) [69] examined patterns of probing attachment loss following scaling and root planing. Incisors, canines, and premolars in 33 patients with generalized periodontal destruction were studied for patterns of probing attachment loss. Patients received supra- and subgingival debridement after oral hygiene instructions, and were followed for 24 months. Measurements were made every third month and 7 patterns of probing attachment were identified. Seventy-three percent (73%) of sites showed a gradual change. Seventeen percent (17) showed an early loss followed by a stabilization of attachment levels. Shallower sites showed a pattern of early loss followed by stabilization while deeper sites showed a gradual loss.

In general, clinicians should assess healing four to six weeks after performing root planning **(AAP 1989)**[70]**. Cercek and coworkers (1983)** noted clinical improvements continued for 8 months however, most of the healing occurred during the first month.

Similarly, **Kaldahl and colleagues (1988)** demonstrated that the repair process extended for 1 year. It appears that the greatest changes with respect to probing depth reduction and gain of clinical attachment can be recorded after 4 to 6 weeks, but gradual repair and maturation of the periodontium may occur over 9 to 12 months.

Longitudinal studies by **Ramfjord et al (1982)**[55] indicated that scaling and root planing arrested attachment loss as well as surgical therapy regardless of probing depths. However, information relating to length of therapy, skill level of therapists,

compliance of patients with recall intervals and plaque control, responsibilities of clinicians for long-term maintenance and disease activity status need further clarification. Concerns regarding these issues must be integrated into therapeutic decisions, because they can dramatically affect interpretation and application of reported data.

In a study by **Kaur H et al**[71] it was shown that lower number of erythrocytes (RBS's) and haemoglobin in periodontitis patients compared to healthy individuals, and no remarkable differences in levels of MCH, MCHC, and MCV were found between Test group and Control group. Thus, based on these results, it can be concluded that, like any other chronic condition, chronic periodontitis can be a causative factor for anaemia proving its influence systemically.

. **Abdul S Aziz et al**[72] evaluated the effect of non-surgical therapy on some oxidative stress markers in patients with chronic periodontitis and found that chronic periodontitis patients show higher inflammatory manifestations and oxidative stress. SRP helps in lowering inflammatory burden and hence, it may be a useful support in the control and prevention of various inflammatory diseases associated with chronic periodontitis.

In a study by **Sawant et al** significant reduction in serum albumin levels in chronic periodontitis patients were seen. There was significant inverse association between serum albumin and clinical attachment loss in patients with chronic periodontitis.

Based on the observations above, we hypothesized that a decrease in serum albumin levels might be related to chronic periodontitis and a return to its normal levels can be expected in response to periodontal treatment.

Therefore, the present study was undertaken to:

- To measure serum albumin levels for control group and for study group at

  T0: Before phase I therapy,

  T1: After 3 months of Phase I therapy.
- To correlate the clinical parameters in the control, with study group before and after phase I therapy along with serum albumin levels.
- To compare serum albumin levels in individuals with healthy periodontium (control group) and chronic periodontitis (study group).
- Estimation of serum albumin levels before and after phase I therapy in patients with chronic periodontitis.

MATERIALS AND METHODS

SOURCE

The study population was selected from the outpatient section of the Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai.

STUDY DESIGN

The study is of a case control type. The study participants will be recruited prospectively in this study.

SAMPLE SIZE

A total of 30 patients need to be selected for study.

Group I- 15 subjects with healthy periodontium. (control group)

Group II- 15 subjects with chronic periodontitis (study group)

Levels of serum albumin will be compared between patient affected with chronic periodontitis before and after phase I therapy and healthy individuals.

ELIGIBILITY CRITERIA

Inclusion criteria

- Periodontally healthy individuals - For control group
- Either sex.
- Study group: Patient affected with chronic periodontitis showing probing pocket depths > 5mm.
- Age 30 – 50 years.
- Agreement to participate in study program.

Exclusion criteria

- Smokers.
- Patients with systemic diseases such as cardiovascular diseases, renal diseases, liver diseases and medically compromised patients such as uncontrolled diabetes, immunosuppression, bleeding disorders, cancer, stroke and sever osteoporosis.
- Aggressive periodontitis
- Patient who underwent periodontal treatment in past 6 months.
- Patient under drugs which affects serum albumin levels.
- Pregnancy and lactation
- Patients under bisphosphonates medication
- Patients under the uses of antibiotics within past 6 months.

ARMEMENTATIUM

For clinical examination

1. Mouth mirror
2. William's Probe
3. Explorer
4. Dental tweezers
5. Cotton rolls
6. Sterilized disposable gloves
7. Disposable face masks and head cap

For collection of blood sample

1. Sterile cotton
2. Surgical spirit
3. Disposable syringe with 22gauge needle
4. Tourniquet.

For Phase I therapy

1. Mouth mirror
2. Explorer
3. Scalers and curettes
4. Kidney tray
5. Cotton Rolls
6. Disposable Gloves
7. Disposable Facemask
8. Disposable Head cap
9. Disposable syringe 24 gauge needle
10. Local Anaesthetic solution
12. 0.9 % Normal saline
13. Ultrasonic scaling unit manufacturer Wood pecker

STUDY DESIGN

The study was carried out in 30 subjects divided into 2 groups with 15 subjects in each group: Group I-15 subjects with healthy periodontium (control group), Group II -15 subjects with chronic periodontitis (study group) . Ethical clearance was obtained from the Institutional Ethical Committee and ethical principles were meticulously followed throughout the study. After explaining the study protocol, informed consent was obtained from all the selected subjects. A thorough medical and dental history of the subjects was taken. All the subjects underwent full-mouth periodontal probing and charting and clinical and laboratory evaluation

CLINICAL PARAMETERS ASSESSMENT

The following clinical parameters were evaluated for all the subjects:

1. Plaque index – ***Silness and Loe 1964***[73]
2. Gingival bleeding index – ***Ainamo and Bay 1975*** [74]
3. Probing depth in mm (PD) – ***Carranza 10th ed*** [75]
4. Clinical attachment level in mm (CAL) – ***Carranza 10th ed***[75]

Plaque Index (Silness and Loe 1964)

All teeth were examined at 4 sites each (disto-facial, facial, mesio-facial, lingual / palatal) and were scored as follows:

Scoring Criteria

Score 0: No plaque in the gingival area.

Score 1: A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque is recognized only by running a probe across the tooth surface.

Score 2: Moderate accumulation of plaque within the gingival pocket and on the gingival margin and / or adjacent tooth surface that can be seen by the naked eye.

Score 3: Abundance of soft deposits within the gingival pocket and / or on the gingival margin and adjacent tooth surface.

CALCULATION

Plaque index per tooth = Total score/4

Plaque index per individual = Total P I per tooth / Total number of teeth examined

Interpretation

Score 0 – Excellent oral hygiene

0.1 to 0.9 – Good oral hygiene

1.0 to 1.9 – Fair oral hygiene

2.0 to 3.0 - Poor oral hygiene

Gingival Bleeding Index *(Ainamo & Bay 1975)*

Starting distobuccally, the probe was gently inserted into the sulcus and run to the buccal and mesial surfaces of every tooth at an angle of about 45º. This was repeated for all the teeth present. Similarly probing was carried out at palatal/lingual sites. The total number of bleeding sites per tooth was thus recorded for every tooth except the third molar.

Scoring Criteria

Positive score (1) - Presence of bleeding within 10 seconds

Negative score (0) - Absence of bleeding

$$\% \text{ of bleeding sites} = \frac{\text{Total number of positive score} \times 100}{\text{Total number of surfaces of all teeth}}$$

Probing Pocket Depth (PPD)

Probing Pocket Depths were measured from the gingival margin to the base of the pocket in millimeters using William's Periodontal Probe. The probe was walked within the gingival sulcus along the circumference of the tooth. Keeping the probe parallel to the long axis of the selected tooth, six measurements were made per tooth (Mesiobuccal, Distobuccal, Midbuccal, Mesiolingual, Distolingual and Midlingual).

Clinical Attachment Level (CAL)

Clinical Attachment Level was measured from the Cemento – Enamel Junction (CEJ) to the base of the pocket using William's Periodontal Probe.

When the gingival margin was located on the anatomic crown, the level of the attachment was determined by subtracting from the probing depth, the distance from the gingival margin to the CEJ. If both were the same, the loss of attachment was calculated to be zero.

When the gingival margin coincided with the CEJ, the loss of attachment was calculated as equaling the probing depth. When the gingival margin was located apical to the CEJ, the loss of attachment was greater than the probing depth and therefore the distance between the CEJ and the gingival margin were added to the PD.

Three measurements were made on the buccal aspect and three on the lingual aspect of each tooth – total of six sites per tooth (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, and Distolingual).

b) Radiographic parameters

- Intra oral periapical radiographs were taken to assess the bone loss.

c) Routine blood investigation

- Haemoglobin
- Bleeding time
- Clotting time
- Total leukocyte count
- Differential leukocyte count
- Random sugar

d) Estimation of Serum Albumin Levels

METHOD OF COLLECTION OF BLOOD SAMPLE

Venous blood was drawn from the participants those were selected for the study. The subjects were informed, and consent was taken. They were made to tighten a fist so that vein was more palpable, and antecubital vein was selected for veni puncture. A tourniquet was applied about 1-2 inches above the antecubital fossa. After disinfecting the puncture site with 10% isopropanol solution, blood was withdrawn using a syringe with 24 gauge needle. Tourniquet was released as the blood flow began. After drawing 3 ml of blood, sterile cotton ball was placed on the puncture site and needle was withdrawn. The subjects were instructed to apply mild finger pressure on the site for few minutes to avoid oozing out of blood.

PRINCIPLE OF THE ASSAY

This kit uses Bromocresol green for the Quantitative determination of albumin.

The method is based on the specific binding of bromocresol green (BCG), an anionic dye, and the protein at acid pH produce a color change of the indicator from yellow –

green to green –blue with the resulting shift in the absorption wavelength of the complex. The intensity of the color formed is proportional to the concentration of albumin in the sample.

BCG + Albumin PH 4.3 → BCG-albumin complex

PROCEDURE

1. Assay conditions :

Wavelength ……..630 nm(600-650)

Cuvette ……1 cm light path

Temperature ……15-25 oC

2. Adjust the instrument to zero with distilled water.

3. Pipette into a cuvette :

4. Mix and incubate for 10 minutes at room temperature (15-250C).

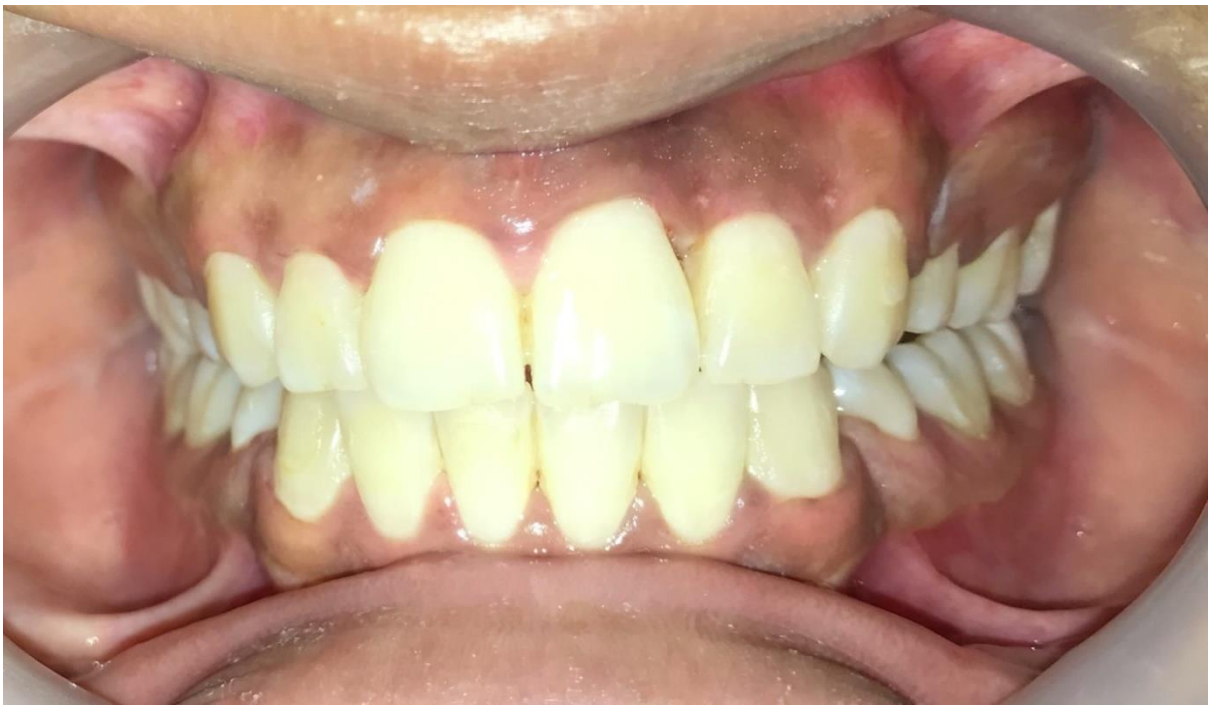
5. Read the absorbance (A) of the samples and standard, against the blank.

The color is stable for 60 minutes at room temperature

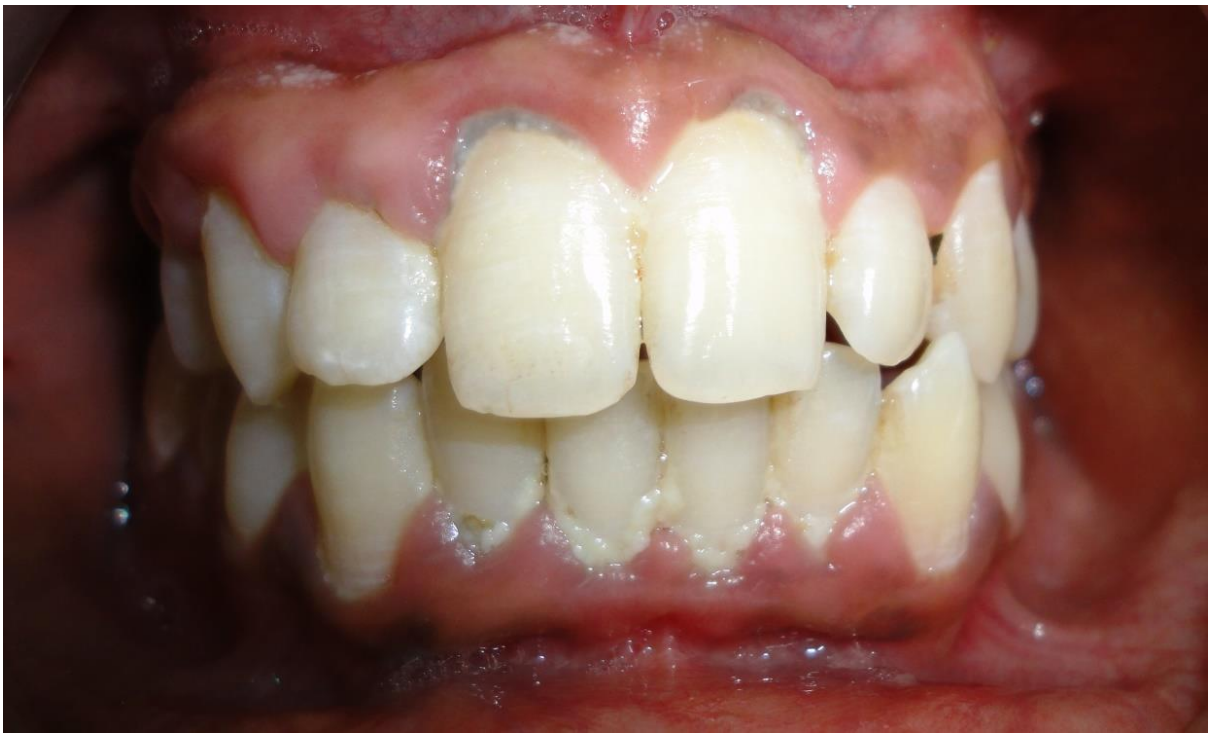
Tubes	Blank	Sample	Standard
Reagent	1.0ml	1.0ml	1.0ml
Sample	-	5µml	-
Standard	-	-	5µml

Calculations:

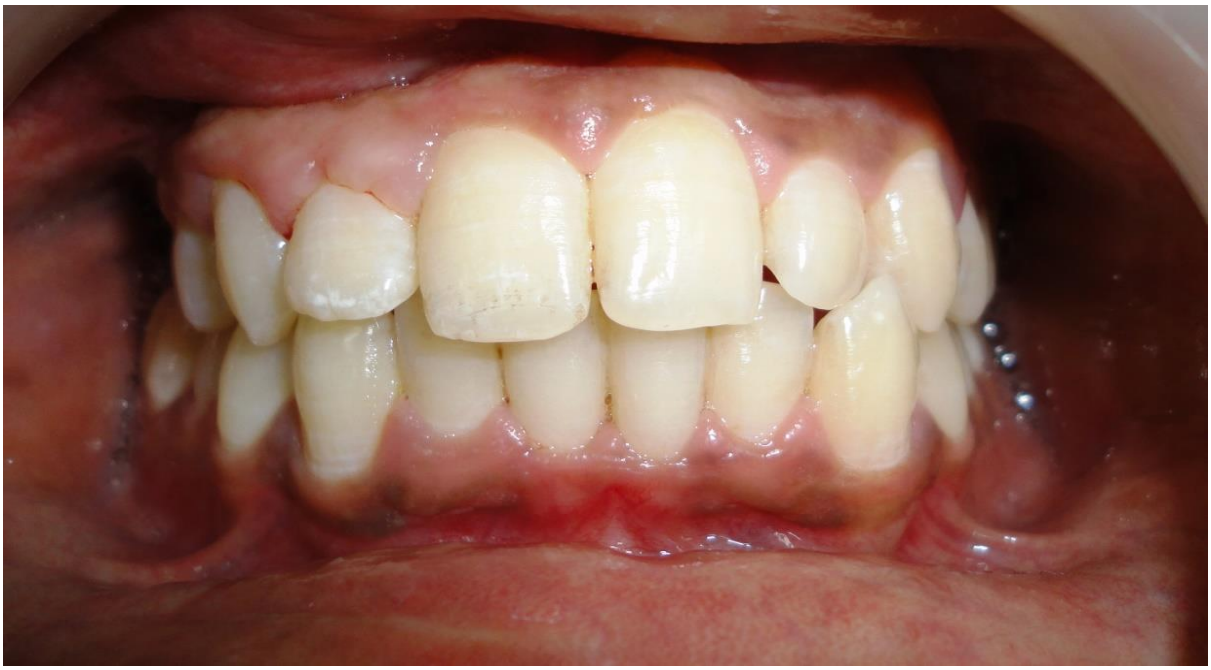
(A) Sample ÷ (A) Standard × 5(standard concentration) = g/dl albumin



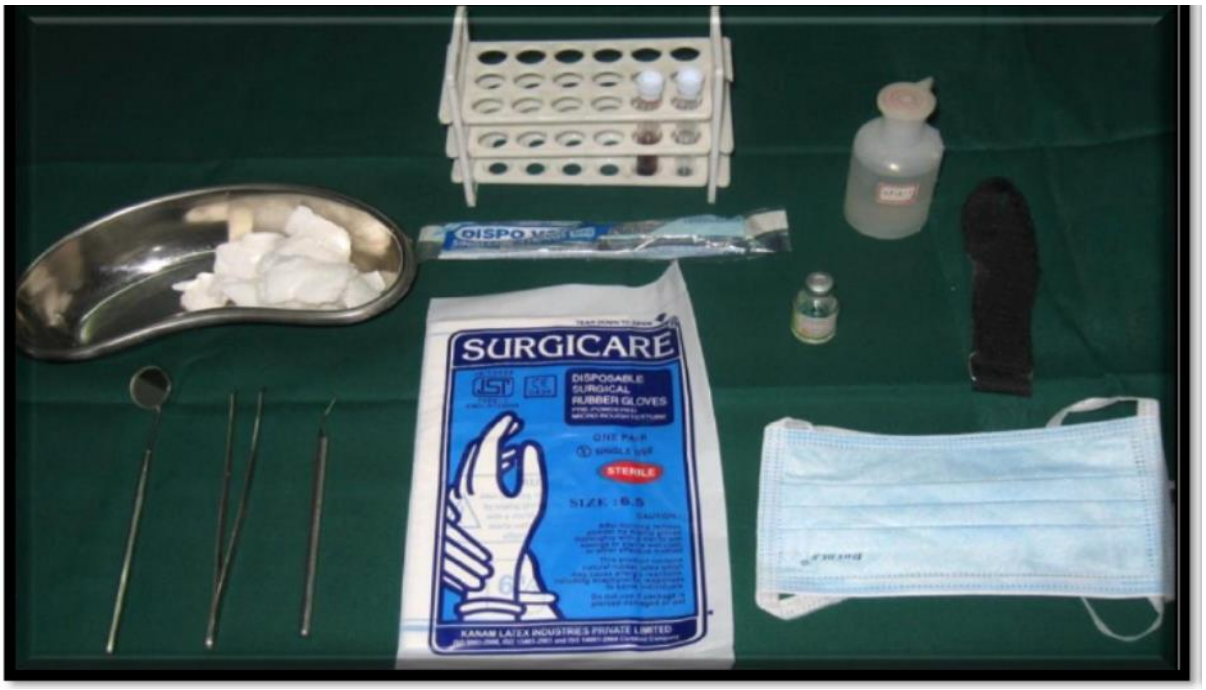
PHOTOGRAPH 1: GROUP I-CONTROLGROUP



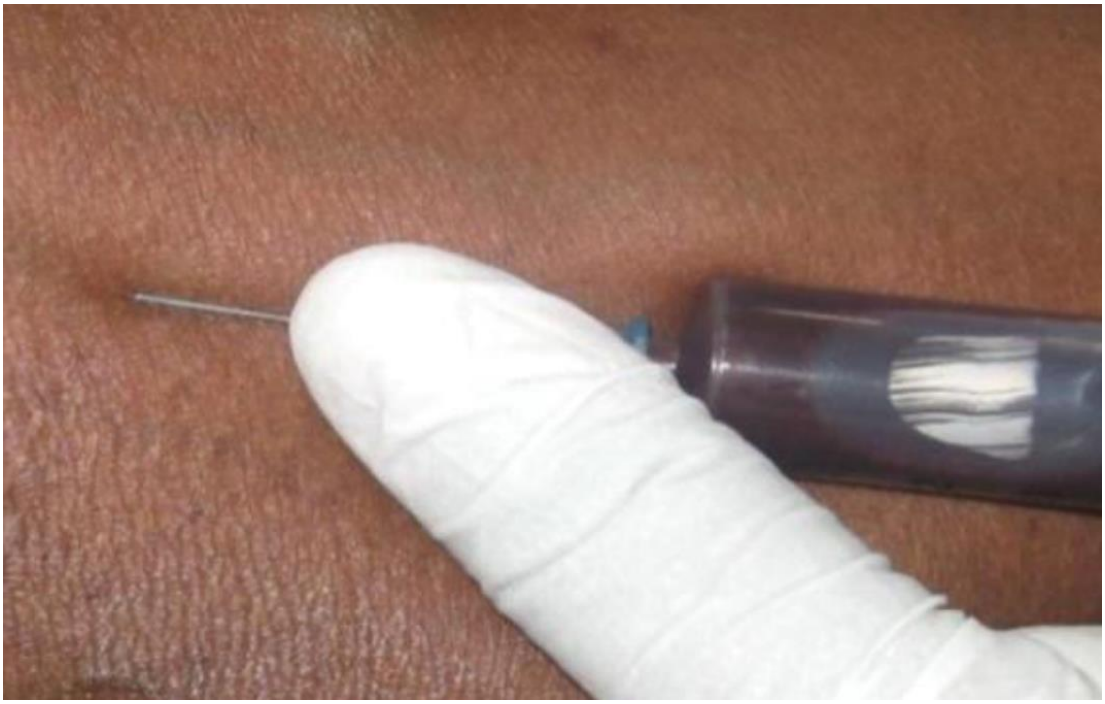
PHOTOGRAPH 2: GROUPII-GENERALISED CHRONIC PERIODONTITIS (BASELINE)



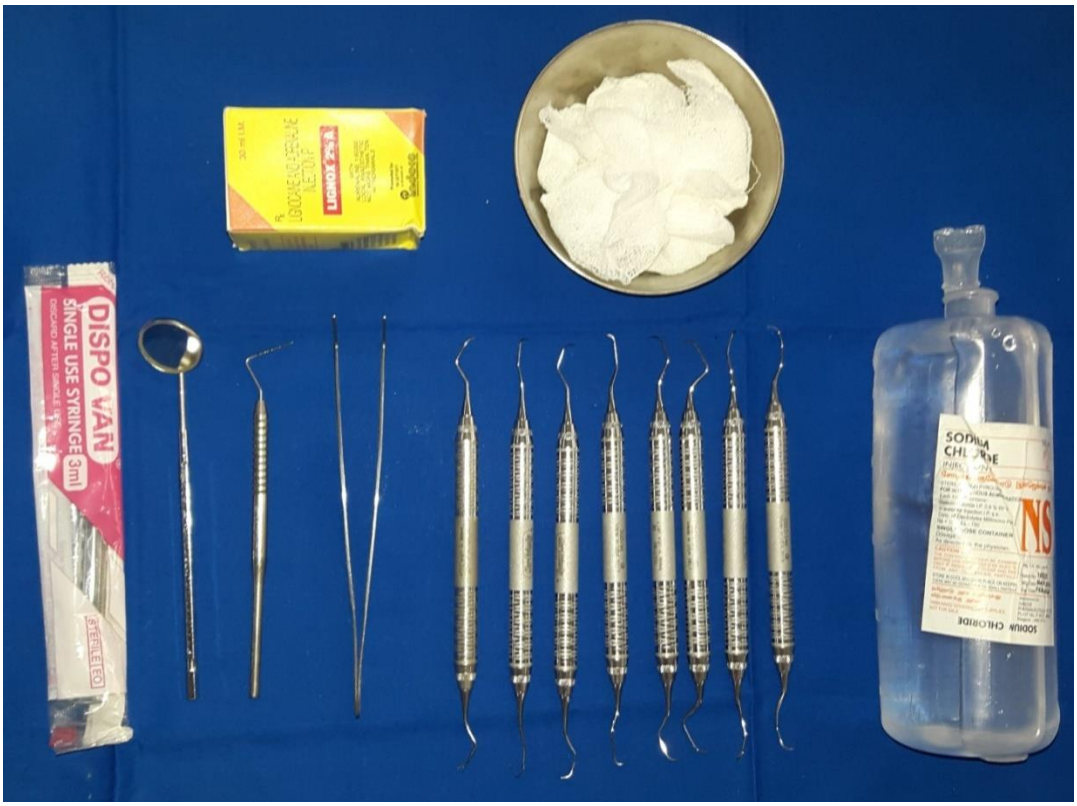
PHOTOGRAPH 3: GROUP II-GENERALISED CHRONIC PERIODONTITIS (3 MONTHS POST OP)



PHOTOGRAPH 4: ARMAMENTARIUM FOR CLINICAL EXAMINATION AND SAMPLE COLLECTION



PHOTOGRAPH 5 : COLLECTION OF THE VENOUS BLOOD SAMPLE



PHOTOGRAPH – 6 ARMAMENTARIUM FOR PHASE I THERAPY

PHOTOGRAPH – 7 : CENTRIFUGAL MACHINE

PHOTOGRAPH – 8 ALBUMIN KIT

PHOTOGRAPH -9: BIOCHEMICAL ANALYZERS

STATISTICAL ANALYSIS

The statistical analysis was done using the computer software program SPSS version 20 (Statistical Package for Social Science, Version 20). Data was expressed as mean ± standard deviation of the parameters evaluated. In Group I, the clinical and laboratory parameters were evaluated at baseline and in Group II at baseline and 3 months post operatively.

STATISTICAL TESTS USED:

1. Tests of normality: **Shapiro wilk's test.**
2. To compare the parametric values between baseline and 3 months in both groups **Paired sample t test** was performed.
3. To compare the parametric values between two groups **Independent sample t test** was performed.
4. To determine the correlation serum albumin with clinical parameters **Pearson rank correlation** coefficient was performed.

P value of <0.05 is considered significant in the present study.

RESULTS

In the present interventional study, 30 subjects were included, among them were Group **I**- 15 subjects with healthy periodontium. (Control group) **Group II- 15** subjects with Generalised Chronic periodontitis (study group). The parameters assessed were plaque index, gingival bleeding index, probing depth and clinical attachment level (clinical) and serum albumin level (laboratory) in Group I and at baseline and 3 months post-operatively in Group II patients.

INTRAGROUP COMPARISON

1. PLAQUE INDEX

Group I: The mean plaque index score in group I at baseline was 0.644±0.033

Group II : The mean plaque index score in group II at baseline was 2.476± 0.065 and at 3 months was 0.984 ±0.98 .Mean difference in plaque index score in group II between baseline and 3 months post-operative value was 1.492±0.118 which was found to be statistically significant (p= 0.000)

INTERGROUP COMPARISION

Mean difference between the baseline values of PI between Group I and Group II 1.832± 0.074 which was found to be statistically significant. (p= 0.000)

2. PROBING POCKET DEPTH

Group I: The mean probing pocket depth score in group I at baseline was 2.15±0.041

Group II: The mean PPD index in group II at base line was 5.174 ±0.292 and at 3 months was 3.306 ±0.210.

Mean difference in plaque index score in group II between baseline and 3 months post-operative value was 1.868 ±0.159 which was found to be statistically significant (p= 0.000)

INTERGROUP COMPARISON

Mean difference between the baseline values of PPD between Group I and Group II 0.340± 0.104which was found to be statistically significant. (p= 0.001)

3. CLINICAL ATTACHMENT LEVEL

Mean CAL index in group II at base line was 5.024 ±0.319 and at 3 months was 3.454 ±0.223. Mean difference in plaque index score in group II between baseline and 3 months post-operative value was 1.570±0.297 which was found to be statistically significant

(p= 0.000)

4. GINGIVAL BLEEDING INDEX

Mean GB index in group II at base line was 74.820 ± 3.022 and at 3 months was 15.113 ±1.487. Mean difference in plaque index score in group II between baseline and 3 months post-operative value was 59.706±2.910 which was found to be statistically significant (p= 0.000)

5. SERUM ALBMIN LEVEL

INTRA GROUP COMPARISON

Group I: The mean serum albumin level in group I at baseline was 4.73±0.052.

Group II: Mean serum albumin level in group II at base line was 3.956 ± 0.049 and at 3 months was 4.567 ±0.017. Mean difference in serum albumin level in group II between baseline and 3 months post-operative value was 0.611± 0.045 which was found to be statistically significant. (p= 0.000)

INTER GROUP COMPARISON:

Mean difference of serum albumin levels between group I and Group II at baseline was 0.777 ± 0.072 which was found to be statistically significant. (p=0.000)

TABLE 1 : MASTER CHART – GROUP 1 (*CLINICAL PARAMETERS)* *CONTROL GROUP*						
S. NO	**AGE**	**SEX**	**BASELINE PI**	**BASELINE PPD**	**BASELINE CAL**	**BASELINE GBI**
1.	25	F	0.65	2.27	0	0
2.	30	M	0.45	2.41	0	0
3.	29	F	0.85	2.02	0	0
4.	28	M	0.83	1.98	0	0
5.	35	M	0.56	2.02	0	0
6.	28	F	0.5	2	0	0
7.	29	M	0.56	2.34	0	0
8.	28	F	0.64	2.13	0	0
9.	30	F	0.5	2.16	0	0
10.	25	F	0.56	2.13	0	0
11	30	F	0.81	2.1	0	0
12	29	M	0.78	2.2	0	0
13	32	F	0.55	2.31	0	0
14	30	M	0.7	1.9	0	0
15	25	F	0.72	2.4	0	0

**TABLE 2: MASTER CHART – GROUP II (*CLINICAL PARAMETERS)*
*CHRONIC PERIODONTITIS***

S. NO	AGE	SEX	BASE LINE PI	BASE LINE PPD	BASE LINE CAL	BASE LINE GBI	3MONTH PI	3MONTH PPD	3MONTH CAL	3MONTH GBI
1.	40	F	2.38	5.81	5.1	83.7	0.87	3.32	3.65	13.4
.	45	F	2.13	4.18	4.07	68.4	0.62	2.14	2.56	10.5
3.	43	F	2.76	6.26	6.18	79.4	0.98	4.21	4.33	22.6
4.	45	F	2.67	6.12	6.14	80.5	0.42	4.02	4.22	17.6
5.	45	M	2.56	7.12	6.98	91.3	1.05	5.02	5.22	24.6
6.	38	F	2.45	6.38	6.24	67.5	0.76	4.02	4.12	11.2
7.	40	M	2.78	6.28	6.24	87.6	0.76	3.98	3.5	24.4
8.	32	M	1.98	5.13	5.08	55.6	0.32	3.03	3.13	9.5
9.	42	M	2.8	5.5	5.25	89.5	1.05	3.28	3.22	10.5
10.	42	M	2.34	4.6	4.4	75.4	1.32	2.3	2.33	8.6
11.	34	M	2.7	3.8	4.55	51.2	1.1	2.2	2.44	21.2
12.	42	F	2.52	4.1	5.1	71.4	1.3	3.09	2.42	10.3
13.	34	M	2.46	4.98	3.18	66.2	1.2	2.8	2.88	10.8
14.	34	F	2.1	3.7	4.44	74.5	1.82	2.2	3.4	18.2
15.	30	F	2.52	3.66	4.4	80.1	1.2	3.1	2.2	13.3

TABLE 3: MASTER CHART – GROUP I (LABORATORYPARAMETER)

S. NO	AGE	SEX	BASELINE SERUM ALBUMIL
1.	37	F	4.8
2.	39	M	4.7
3.	41	M	4.8
4.	33	F	4.9
5.	37	F	4.9
6.	48	M	4.9
7.	48	M	4.9
8.	32	M	4.6
9.	38	F	4.8
10.	47	M	4.9
11	35	F	4.7
12	30	M	4.9
13	44	F	4.8
14	33	M	4.7
15	42	F	4.6

TABLE 4: MASTER CHART – GROUP II (LABORATORYPARAMETER)

S. NO	AGE	SEX	BASELINE SERUM ALBUMIN	3 MONTH POST-OP SERUM ALBUMIN
1.	45	F	3.8	4.2
2.	39	M	3.4	3.8
3.	41	M	3.8	4.1
4.	53	F	4.0	4.3
5.	37	F	3.8	4..0
6.	45	M	3.9	4..1
7.	48	M	4.0	4..2
8.	32	F	4.2	4.5
9.	38	F	4.0	4.3
10.	47	M	3.6	3.8
11.	45	F	4.1	4.3
12.	30	M	3.6	4.0
13.	40	F	3.6	3.9
14.	33	M	3.8	4.2
15.	42	F	3.7	4.0

TABLE-5

GROUP-I

Descriptive Statistics

Variables	N	Minimum	Maximum	Mean		Std. Deviation
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
BASELINE PI	15	.45	.85	.644	.033	.131
BASELINE PPD	15	1.9	2.41	2.15	.041	.160
BASELINE CAL	15	0	0	0	0	0
BASELINE GBI	15	0	0	0	0	0
BASELINE SERUM ALBUMIN	15	4.36	5.01	4.73	.052	.203

TABLE-6

CORRELATION BETWEEN CLINICAL PARAMETERS AND SERUM ALBUMIN LEVEL IN GROUP 1

CLINICAL PARAMETERS		BASELINE SERUM ALBUMIN
BASELINE PI	Pearson Correlation	.245
	Sig. (2-tailed)	.398
BASELINE PPD	Pearson Correlation	.324
	Sig. (2-tailed)	.258
BASELINE CAL	Pearson Correlation	-
	Sig. (2-tailed)	-
BASELINE GBI	Pearson Correlation	-
	Sig. (2-tailed)	-

TABLE-7

GROUP 2

Descriptive Statistics						
Variables	**N**	**Minimum**	**Maximum**	**Mean**		**Std. Deviation**
	Statistic	**Statistic**	**Statistic**	**Statistic**	**Std. Error**	**Statistic**
BASELINE PI	15	1.98	2.80	2.476	.065	.255
BASELINE PPD	15	3.66	7.12	5.174	.292	1.131
BASELINE CAL	15	2.42	6.98	5.024	.319	1.238
BASELINE GBI	15	51.20	91.30	74.820	3.022	11.707
BASELINE SERUM ALBUMIN	15	3.65	4.32	3.956	.049	.192
3 MONTHS PI	15	.32	1.82	.984	.098	.380
3 MONTHS PPD	15	2.14	5.02	3.306	.210	.814
3 MONTHS CAL	15	2.33	5.22	3.454	.223	.866
3 MONTHS GBI	15	8.60	24.60	15.113	1.487	5.762
3 MONTHS SERUM ALBUMIN	15	4.43	4.65	4.567	.017	.066

TABLE-8

CORRELATION BETWEEN CLINICAL PARAMETERS AND SERUM ALBUMIN LEVEL IN GROUP II

CLINICAL PARAMETERS		BASELINE SERUM ALBUMIN
BASELINE PI	Pearson Correlation	-.098
	Sig. (2-tailed)	.729
BASELINE PPD	Pearson Correlation	-.117
	Sig. (2-tailed)	.677
BASELINE CAL	Pearson Correlation	-.061
	Sig. (2-tailed)	.830
BASELINE GBI	Pearson Correlation	-.015
	Sig. (2-tailed)	.957

CLINICAL PARAMETERS		3 MONTHS SERUM ALBUMIN
3 MONTHS PI	Pearson Correlation	.150
	Sig. (2-tailed)	.595
3 MONTHS PPD	Pearson Correlation	.246
	Sig. (2-tailed)	.377
3 MONTHS CAL	Pearson Correlation	.212
	Sig. (2-tailed)	.448
3 MONTHS GBI	Pearson Correlation	.183
	Sig. (2-tailed)	.515

TABLE-9

CORRELATON BETWEEN CLINICAL PARAMETER AT BASE LINE & 3 MONTHS IN GROUP II

Paired T Test

Variables		Paired Differences					t	Df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	BASELINE PI - 3 MONTHS PI	1.492	.457	.118	1.238	1.745	12.627	14	.000
Pair 2	BASELINE PPD - 3 MONTHS PPD	1.868	.618	.159	1.526	2.210	11.710	14	.000
Pair 3	BASELINE CAL - 3 MONTHS CAL	1.570	1.151	.297	.932	2.207	5.282	14	.000
Pair 4	BASELINE GBI - 3 MONTHS GBI	59.706	11.273	2.910	53.463	65.949	20.511	14	.000
Pair 5	BASELINE SERUM ALBUMIN - 3 MONTHS SERUM ALBUMIN	.611	.176	.045	.708	.513	13.386	14	.000

TABLE-10

UNPAIRED T TEST BETWEEN THE BASELINE SERUM ALBUMIN LEVEL OF HEALTHY PERIODONTIUM AND CHRONIC PERIODONTITIS

Variables	Differences				T	Df	Sig. (2-tailed)
	Mean	Std. Error Mean	95% Confidence Interval				
			Lower	Upper			
SERUM ALBUMIN LEVEL OF HEALTHY PERIODONTIUM vs. SERUM ALBUMIN LEVEL OF CHRONIC PERIODONTITIS	.777	.072	.629	.925	10.745	28	.000

TABLE-11

UNPAIRED T TEST BETWEEN THE BASELINE VALUES OF CLINICAL PARAMETERS OF CONTROL AND STUDY GROUP

Variables	Differences				t	Df	Sig. (2-tailed)
	Mean	Std. Error Mean	95% Confidence Interval				
			Lower	Upper			
CG PI BL - SG PI BL	1.832	.074	1.680	1.984	24.725	28	.000
CG PPD BL - SG PPD BL	.340	.104	.127	.553	3.274	28	.001

*CG – CONTROL GROUP, SG – STUDY GROUP, BL – BASELINE

Figure 1 : **Comparison of plaque index between group I and group II**

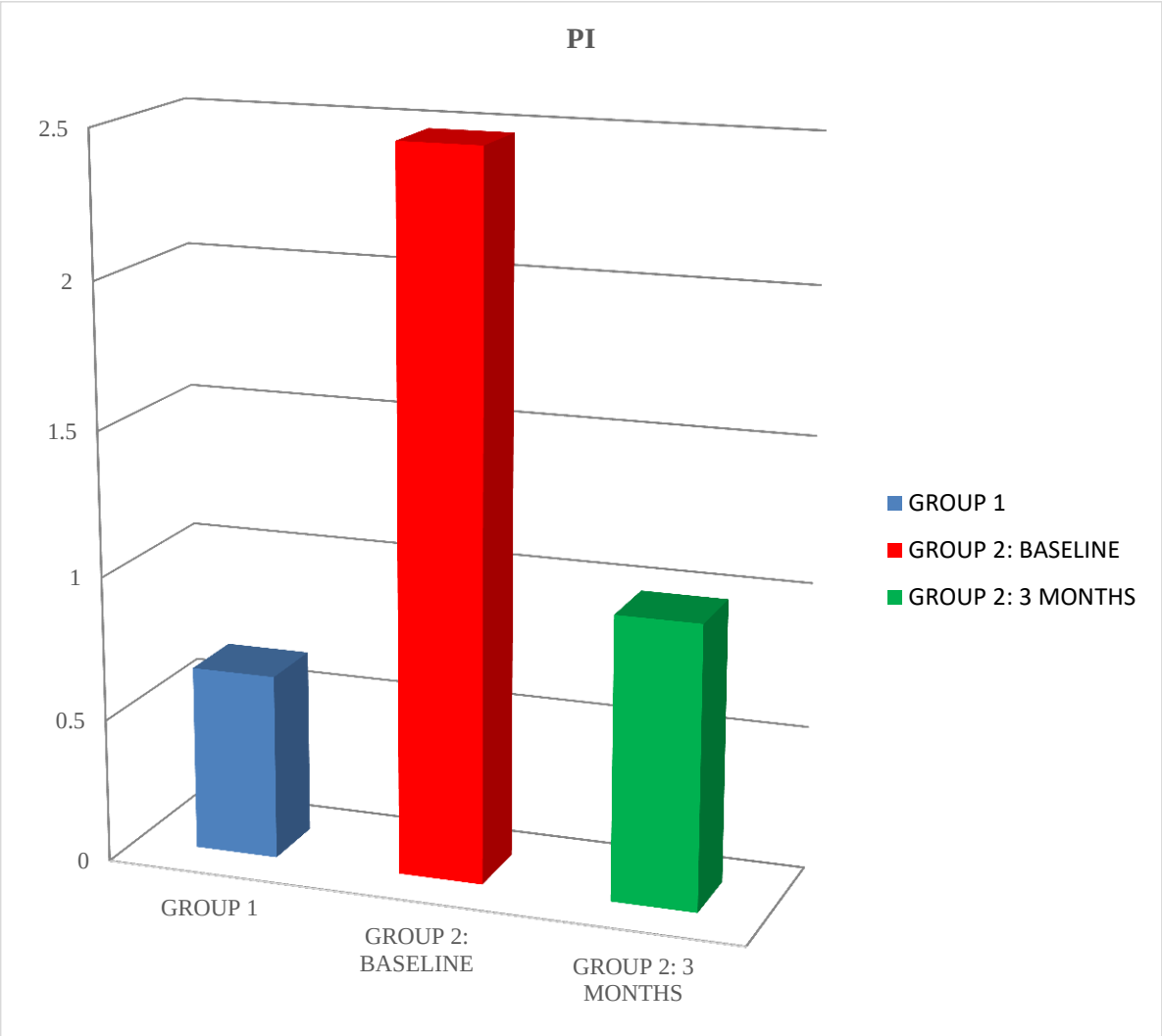


Figure 2 : **Comparison of Probing pocket depth between group I and group II**

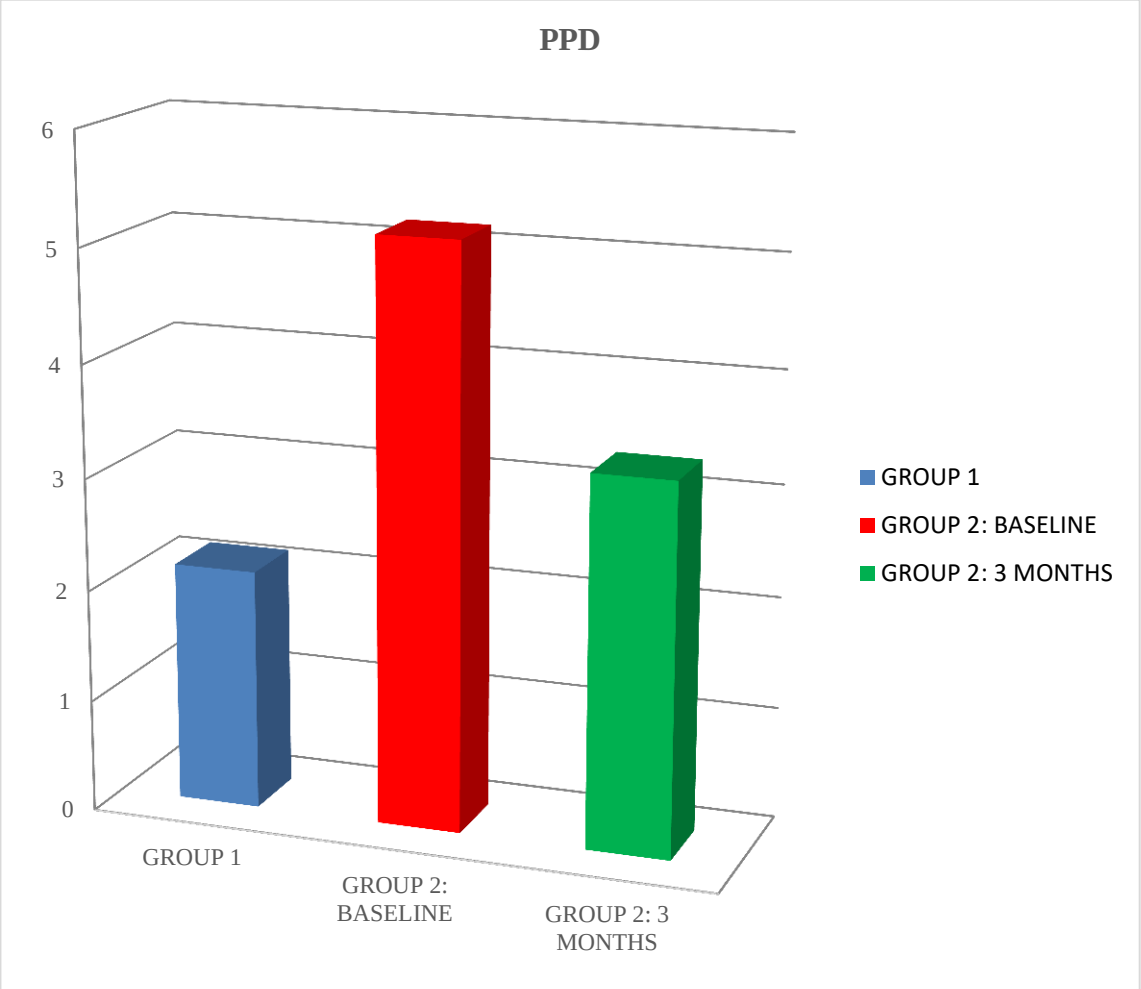


Figure 3 **: Comparison of Clinical attachment level between group I and group II**

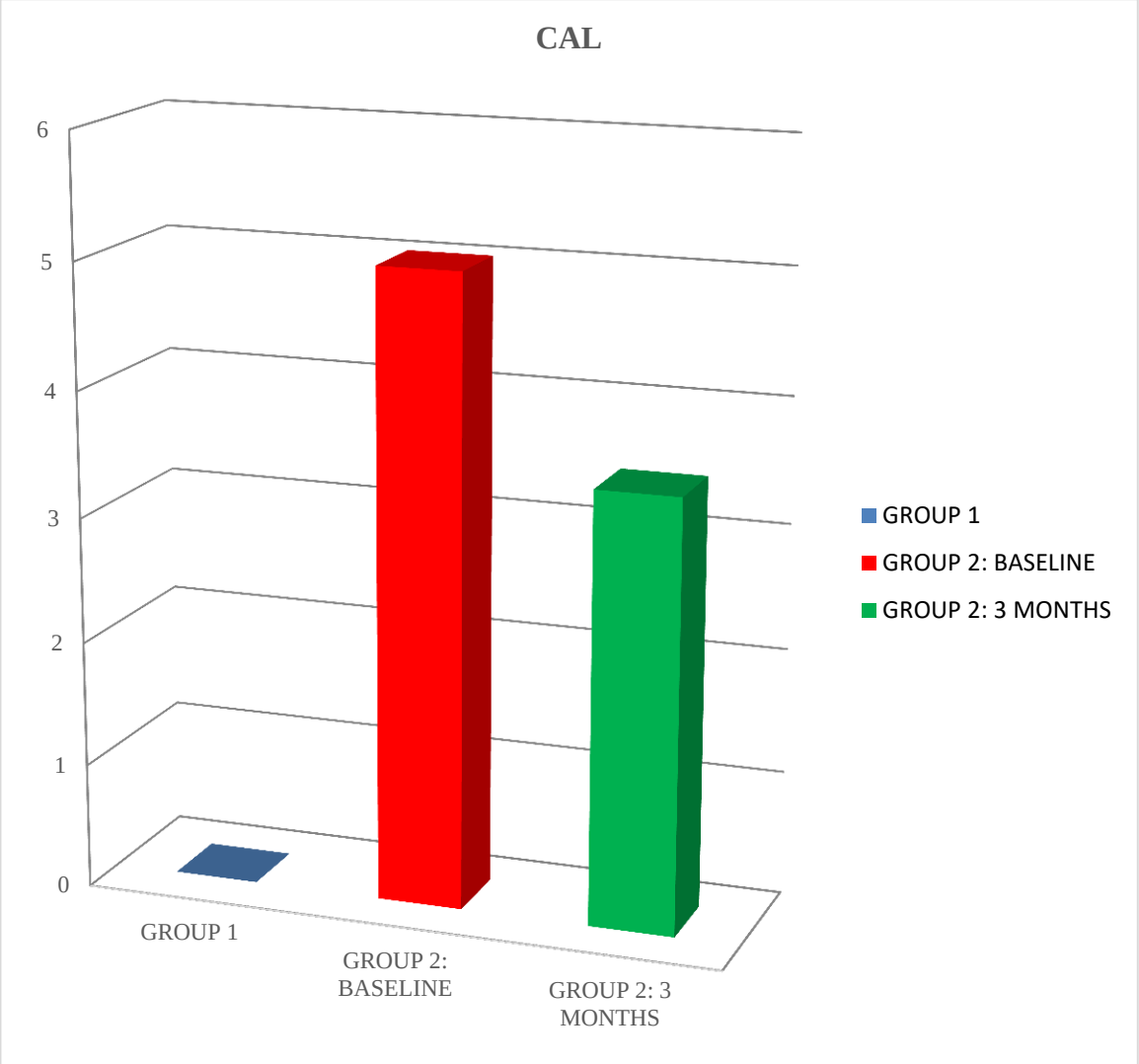


Figure 4 **: Comparison of gingival bleeding index between group I and group II**

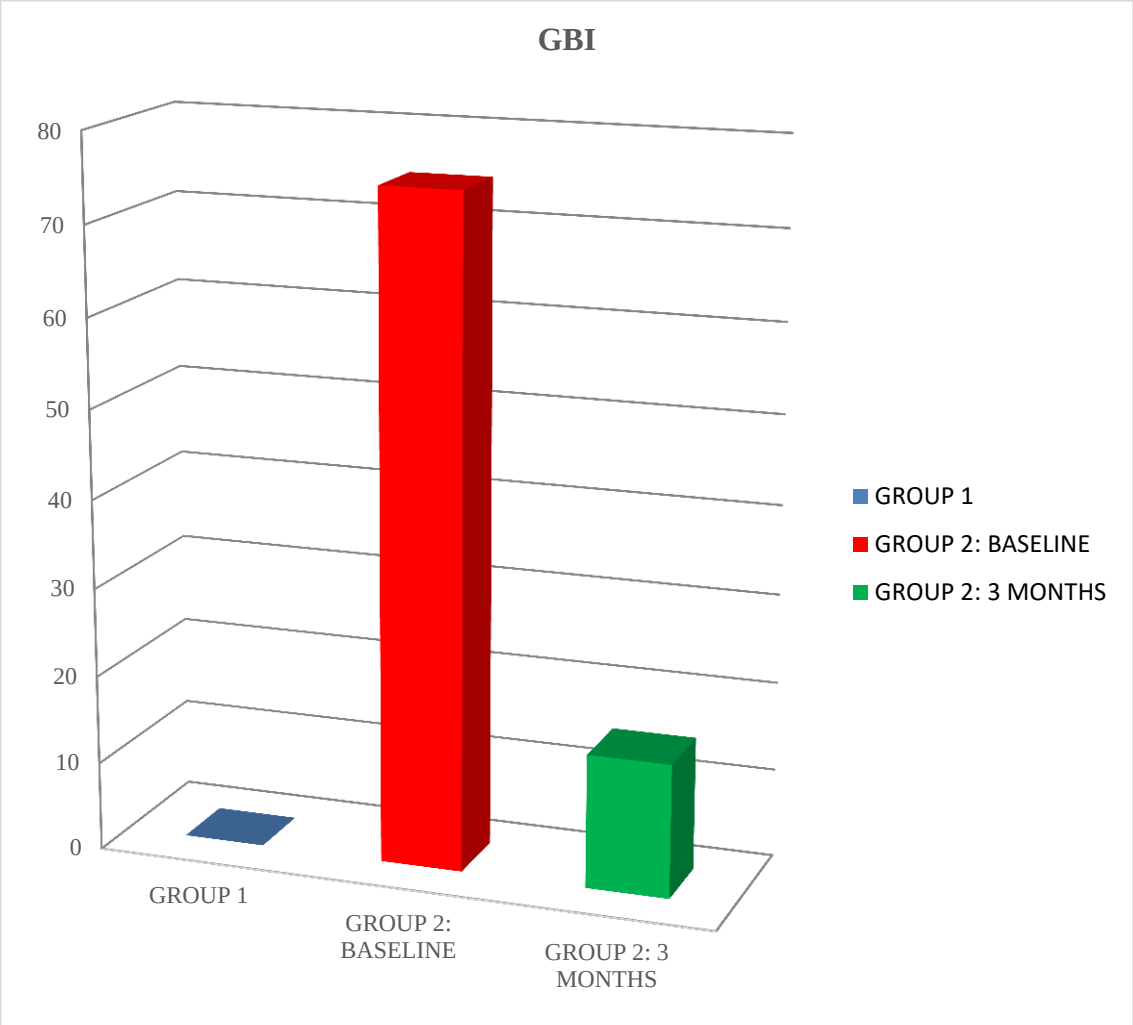
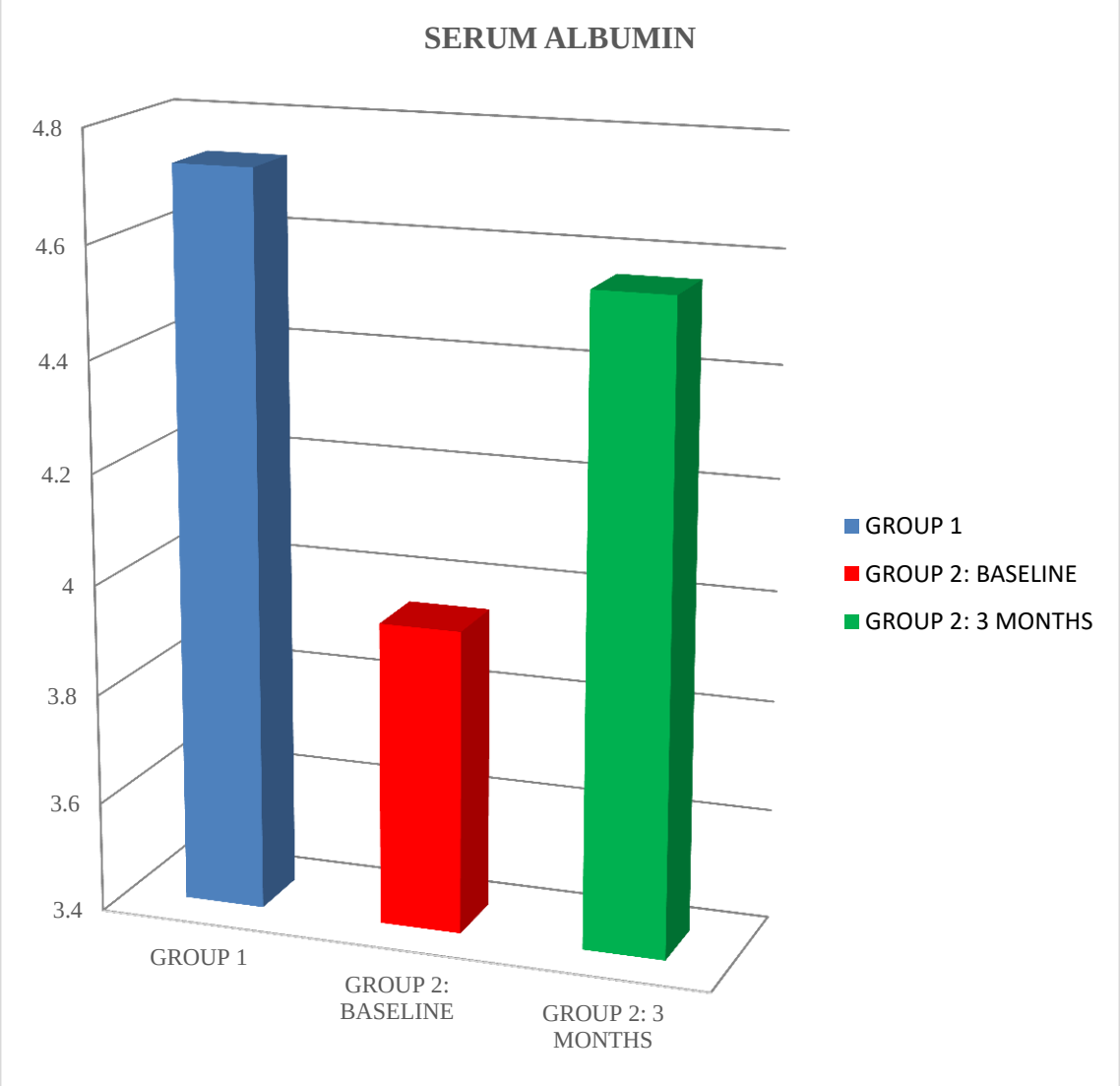


***Figure 5*: Comparison of serum albumin levels between group I and group II**



DISCUSSION

Periodontitis is a chronic inflammatory disease caused by anerobic gram negative bacteria affecting the supporting structures of the teeth. Serum Albumin concentration is a negative acute phase protein associated with inflammation and it might be a practical marker for general health status. Periodontitis results in the formation of many inflammatory mediators like C -reactive proteins. Also this will stimulate antibody formation like IgG. A significant association between serum albumin concentration and IgG has been reported .C -reactive protein may be used to identify the presence of inflammation in individuals with a lower serum albumin concentration.

Recently report says that the number of untreated teeth was a significant factor associated with serum albumin concentration in elderly. Therefore, it is suggestive that the oral disease burden might be monitored by the levels of serum albumin.

Inflammation and malnutrition both reduce the serum albumin concentration by decreasing its rate of synthesis. This suggests that periodontal disease severity might be indicated and monitored by the levels of serum albumin. Therefore, serum albumin can be used as a risk predictor for periodontal disease.

The present study was carried out to estimate the levels of serum albumin at baseline level and 3 months after phase I therapy in patients with chronic periodontitis.

In the present study, 30 subjects were selected, among them

15 subjects with clinically healthy individuals were categorized as- **Group I** (T0) and

15 subjects with generalized chronic Periodontitis were categorized as- **Group II** (T1).

Several studies have been carried out to evaluate albumin levels in serum in patients with chronic periodontitis but none of the studies were there to compare the serum albumin levels before and after phase 1 therapy in chronic periodontitis.

In the present study, subjects with any acute or chronic systemic conditions like diabetes or inflammatory conditions like rheumatoid arthritis, cardiovascular disease etc. have been excluded because these conditions can cause decreased albumin levels on their own, which may lead to confoundin g effect in the study. Smoking is also a potential confounding and environmental risk factor periodontitis and hence smokers were also excluded from the study.

Patients under medications like antibiotics, corticosteroids, anti-inflammatory drugs for past 3 months and those underwent periodontal therapy within past six months have been excluded because these therapies can suppress the inflammatory process and may lead to confounding effect in the study. Patients under medications like antibiotics, corticosteroids, anti-inflammatory drugs for past 3 months and those underwent periodontal therapy within past six months have been excluded because these therapies can suppress the inflammatory process and may lead to confounding effect in the study.

Quantitative estimation of serum albumin levels done in the present study in both groups was done by BROMOCRESOL GREEN method.

In the present study, clinical parameters like plaque index, PPD, CAL, gingival bleeding index were also assessed and compared among the two groups to correlate with the levels of serum albumin to establish a relationship between altered serum albumin levels and periodontal disease status. In present study PI and PPD, Serum albumin values at baseline in group I was (0.644 ± 0.033 2.15 ±0.041, 4.73±0.052.) respectively .

The mean base line value for PI, PPD, CAL, GBI and serum albumin was (2.476±0.065, 5.174±0.295, 5.024±0.319, 74.820±3.022 and 3.956± 0.49) and 3 months value after phase I therapy was(0.984± 0.098,3.306±0.210,3.454±0.223 ,15.113±1.487 and 4.567±0.0170)

The mean difference between the baseline values of PI, PPD,and serum albumin was(1.832± 0.074, 0.340± 0.104 , 0.777 ± 0.072) which was found to be statistically significant(p= 0.000) .

And mean difference in PI ,PPD,CAL ,GBI and serum albumin in **Group II** at baseline and 3 months post operative value was (1.492±0.118, 1.868 ±0.159, 1.570±0.297 , 59.706±2.910, 0.611± 0.045) which was found to be statistically significant(p= 0.000) .

The results of present study indicate the difference between serum albumin levels in Group I and Group II were found to be statistically significant ($P \leq 0.001$). The findings of this clinical trial suggest an inverse relationship between the serum albumin concentration and chronic periodontal disease which is consistent with the finding of **Navkiran Kaur**[40] who done a study to evaluate the correlation of serum albumin levels with chronic periodontitis.

The results observed from the present study shows that the mean serum albumin levels in patient with chronic periodontitis (3.65 ± 0..049) was significantly less than that in periodontally healthy subjects(4.73±0.052). And after three months of phase I therapy, the serum albumin levels increased significantly (4.43±0.017) and approached the levels in periodontally healthy subjects. And there was improvement in all the clinical parameters such as reduced BI, PI, PPD and gain in clinical attachment level. This finding might be attributed to a decrease in proinflammatory cytokine levels, especially IL-6, after periodontal intervention in patient with chronic periodontitis since IL-6 is the chief agent for the hepatocytic secretion of the majority of acute phase proteins. It appears logical that if periodontal inflammation decreases serum albumin levels, these levels should increase subsequent to non-surgical periodontal treatment which decreases the load of systemic inflammation

The results are consistent with **Shirmohamadi et al**[38] who done a study to evaluate Effect of non-surgical periodontal treatment on transferrin serum levels in patients with chronic periodontitis. Serum Transferrin which is also a negative acute phase protein similar to that of albumin and results were the mean transferrin serum level in patients with chronic periodontitis (213.1 ± 9.2 mg/dL) was significantly less than that in periodontally healthy subjects (307.8 ± 11.7 mg/dL). After three months periodontal trseatment, the transferrin serum level increased significantly (298.3 ± 7.6 mg/dL) and approached the levels in periodontally healthy subjects ($P < 0.05$). There is decrease in transferrin serum levels with periodontal disease and increased after periodontal treatment, respectively, indicated an inverse relationship between transferrin serum levels and chronic periodontitis.

In the present study the levels of serum albumin is low in all chronic periodonititis patients which might be consistence with the results reported

Kshirsagar et al[31] who studied levels of serum albumin and CRP among patients on HD. In this study, patients with severe periodontitis had low serum albumin levels (odds ratio 8.20; 95% confidence interval 1.61-41.82; p = 0.01) compared with individuals without severe periodontitis disease after and 3 months adjustment for several factors.

In a study by **Pimpale S et al**[76] to correlate between periodontal disease and serum albumin concentration in chronic periodontitis patients and suggested that serum albumin concentration is an important risk indicator in chronic periodontitis patients. Thus, in present study it would seem appropriate to infer that the lower serum albumin concentrations were effected by inflammatory component of chronic periodontitis.

In a study done to evaluate the effect of Non-Surgical Periodontal Therapy on Serum Albumin Levels of Patients on Maintenance Hemodialysis Therapy suggest that hypoalbuminemia has been demonstrated to be a strong predictor of death in chronic renal failure. Some studies have however suggested that hypoalbuminemia may be more indicative of underlying inflammation, rather than nutritional status, especially in patients with kidney disease. Inflammation is an important predictor of low serum albumin levels among dialysis patients, independent of nutritional status. Low albumin is often indicative of malnutrition, but chronic inflammation appears to be the culprit in half of the patients with low levels of albumin.

The above study were consistent with the present study suggesting that in which periodontitis is also a chronic inflammatory condition in which low levels of serum albumin has been observed inferring that it might be a marker of inflammatory disease .

The finding of our study shows statistically significant inverse relationship between serum albumin levels and chronic periodontal disease. Hence serum albumin levels might be an important risk indicator for chronic periodontal disease. In order to explore the actual cause to effect relationship between periodontal disease and serum albumin concentration, longitudinal evaluations with a larger population would be necessary.

Summary and Conclusion

The present study was conducted in order to evaluate the serum albumin levels in chronic periodontitis patients before & after phase 1 therapy .A total of 30 patients were selected and divided into two groups Group I - control group and Group II- chronic periodontitis. The clinical and laboratory data were assessed at baseline for control group and for chronic periodontitis patients at baseline and 3 months after phase 1 therapy the values were subjected to statistical analysis.

The following conclusions were drawn from the study:

- Lower serum albumin levels were detected in patients with chronic periodontitis.
- Non-surgical periodontal treatment resulted in an increase in serum albumin levels in chronic periodontitis group, which reached the levels in periodontally healthy subjects.

Within the limits of the present study, it can be concluded that there is a statististically significant inverse association between serum albumin concentration and chronic periodontal disease. Further studies are necessary with larger sample sizes to determine whether serum albumin levels can be used as diagnostic markers for periodontal diseases. In order to explore the actual cause to effect relationship between periodontal disease and serum albumin concentration, longitudinal evaluations with a larger population would be necessary.

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Annexure 1: Participant Information Sheet- English

PARTICIPANT INFORMATION SHEET

TITLE OF THE STUDY:

Estimation of serum albumin levels before and after phase I therapy in patients with chronic periodontitis.

Assessment of effect of phase I periodontal therapy in altering serum albumin levels in patients with chronic Periodontitis.

NAME OF THE RESEARCH INSTITUTION:

Tamil Nadu Government Dental College and Hospital, Chennai.

PURPOSE OF THE STUDY:

The purpose of this study i**s** to "**Estimate Serum Albumin levels before and after phase I therapy in patients with chronic periodontitis".**

PROCEDURE:

Case history, Intra Oral examination X rays will be taken. About one table spoon of blood will be drawn from your hand. Scaling (cleaning of the teeth surface) and root planning will be done under Local anaesthesia. For some of the participants the blood sample will be collected after 3months once again collected blood will be sent for estimation of serum albumin level. Clinical parameters such as gingival bleeding index, plaque index, probing pocket depth and clinical attachment level will be done.

RISK OF PARTICIPATION:

Pain, swelling can happen during blood sample collection.

Radiation exposure during IOPA view radiographs procedure.

Pain and discomfort due to local anaesthetic effect.

BENEFITS OF PARTICIPATION:

Patients get scaling and root planning therapy for periodontal disease.

CONFIDENTIALITY:

The identity of the patients participating in the research will be kept confidential throughout the study. In the event of any publication and presentation resulting from the research, no personally identifiable information will be shared.

PARTICIPANTS RIGHTS:

Taking part in the study is voluntary. You are free to decide whether to participate in the study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.

COMPENSATION : Nil

Name of the participant **Signature of the participant**

For queries related to study:
xxxxxxxxxxxxxxx

For queries related to rights of participant
xxxxxxxxxxxxxxxxx

Annexure 2: Informed Consent Form: English

INFORMED CONSENT FORM

ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE I THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS

"I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care."

_____ _____ _____

Date Name of the participant Signature/Thumb impression of the participant

[The literate witness selected by the participant must sign the informed consent form. The witness should not have any relationship with the research team; If the participant does not want to disclose his/her participation details to others, in view of respecting the wishes of the participant, he/she can be allowed to waive from the witness procedure. (This is applicable to literate participant ONLY). This should be documented by the study staff by getting signature from the prospective participant.]

"I have witnessed the accurate reading of the consent form to the potential participant and the individual has had opportunity to ask questions. I confirm that the individual has given consent freely."

Date Name of the witness Signature of the witness

Date Name of the interviewer Signature of the interviewer

Annexure 3: Participant Information Sheet: Tamil

ஆராய்ச்சி பற்றிய தகவல் படிவம்

ஆராய்ச்சி மேற்கொள்பவர்: **வழி நடத்துபவர்:**

மருத்துவர்.ஜே.ஹேமலதா மருத்தவர்.கே.மாலதி

ஆராய்ச்சியின் தலைப்பு

“ஈறு அழற்சி நோய் உள்ளவர்களின் இரத்தத்தில் ஆல்புமின் அளவை அறுவை சிகிச்சையில்லா ஈறு மருத்துவத்திற்கு முன்பும் பின்பும் ஒப்பிட்டு மதிப்பீடு செய்தல்”

செய்முறை:

கீழ்கண்ட ஆய்வுகள்/பரிசோதனைகள் உங்களுக்கு செய்யப்படும்:

- வாய் பரிசோதனை - உட்புறம், வெளிப்பறம்.
- நோயுற்ற பகுதியின் ஊடுகதிர் படம் எடுக்கப்படும்.
- வழக்கமான இரத்தப் பரிசோதனை செய்யப்படும்.
- உங்கள் கையிலிருந்து பரிசோதனைக்காக 2 மிலி அளவு இரத்தம் எடுக்கப்படும்.
- ஒவ்வாமை ஏற்படுகிறதா என்பதை தெரிந்து கொள்ள 0.5மிலி 2% லிக்னோகெயின் என்னும் மரத்துப்போக செய்யும் மருந்து உங்களின் கையில் பரிசோதனைக்காக செலுத்தப்படும். பின்பு நோயுற்ற பகுதியில் இம்மருந்து செலுத்தப்படும்.
- அல்ட்ரா சோனிக் ஸ்கேலர் மற்றும் கைக்கருவிகள் பயன்படுத்தி பல் மற்றும் பல்லின் வேர் சுத்தம் செய்ப்படும். உப்பு நீர் கொண்டு நோயுற்ற பகுதி சுத்தம் செய்யப்படும்.

பங்கேற்பதினால் வரக்கூடிய பக்க விளைவுகள்

பக்க விளைவுகள் ஏற்படாமல் தடுக்க உரிய முறைகள் பின்பற்றப்படும்.

- ஊடுகதிர் படம் எடுக்கப்படும் பொழுது லெட் ஏப்ரன், தைராய்டு காலர் போன்ற பாதுகாப்பு உபகரணங்கள் பயன்படுத்தப்படும்.
- சிறந்த தரம் மற்றும் சுத்தமான கருவிகள் பயன்படுத்தப்படும்.
- சிகிச்சைக்குப் பின் தேவைபட்டால் வலி நிவாரண மருந்துகள் கொடுக்கப்படும்.

பங்கேற்பதினால் விளையும் நன்மைகள்

உங்களின் ஈறு அழற்சி நோய்க்கு அறுவைசிகிச்சையில்லா ஈறு மருத்துவ முறையின் படி சிகிச்சை அளிக்கப்படும்.

இரகசியகாப்பு:

உங்களை பற்றிய குறிப்புகள் பிறர் அறியாவண்ணம் ஆராய்ச்சி முடியும் வரை இரகசியமாக பாதுகாக்கப்படும். அதை வெளிப்படுத்தும் நேரங்களிள் எந்த தனி நபர் அடையாளங்களும் வெளிப்பட வாய்ப்பு கிடையாது.

தன்னார்வ பங்கேற்பு:

இந்தஆராய்ச்சியில் பங்கு பெறுவது தங்களின் தனிப்பட்ட முடிவு மற்றும் இந்த ஆராய்ச்சியிலிருந்து தாங்கள் எப்பொழுது வேண்டுமானாலும் விலகிக் கொள்ளலாம். தங்களின் இந்த திடீர் முடிவு தங்களுக்கோ அல்லது ஆராய்ச்சியாளருக்கோ எவ்வித பாதிப்பம் ஏற்படுத்தாது என்பதை தெரிவித்துக் கொள்கிறோம்.

நோயாளியின் பெயர் நோயாளியின் கையொப்பம்

_____

ஆராய்ச்சி தொடர்புடைய தகவல்களுக்கு: பங்கேற்பாளரின் உரிமை தொடர்புடைய தக வல்களுக்கு:

XXXXXXXXXXXXXXXXXXXX XXXXXXXXXXXXXX

Annexure 4: Informed Consent Form: Tamil

ஆராய்ச்சிஒப்புதல்படிவம்

ஆராய்ச்சிதலைப்பு

“ஈறு அழற்சி நோய் உள்ளவர்களின் இரத்தத்தில் ஆல்புமின் அளவை அறுவை சிகிச்சையில்லா ஈறு மருத்துவத்திற்கு முன்பும் பின்பும் ஒப்பிட்டு மதிப்பீடு செய்தல்”

பெயர்: வயது/பால்:

முகவரி: தொலைபேசி:

புறநோயாளி எண்:

ஆராய்ச்சி சேர்க்கை எண்:

நான் _____ வயது _____
என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள ஒப்புதல் அளிக்கிறேன்.

கீழ்காணப்படும் நிபந்தனைகளுக்கு நான் சம்மதிக்கிறேன்:

நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் செய்முறைகள் பற்றி முழுமையாக தெரிவிக்கப்பட்டுள்ளேன்

இந்த பரிசோதனைக்காக பற்களை சுத்தம் செய்ய வேண்டியுள்ளதாக அறிகிறேன்

என் உடல் நலம்பாதிக்கப்பட்டாளோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய் குறிகள் தென்பட்டாளோ அதற்கு சிகிச்சை பெற்று கொள்வதற்கும் முழு உரிமை உள்ளதாக அறிகிறேன்.

நான் எற்கனவே உட்கொண்ட மற்றம் உட்கொள்கின்ற மருந்துகளின் விபரங்களை ஆராய்சியாளரிடம் தெரிவித்துள்ளேன்.

என் மருத்துவ குறிப்பேடுகளை இந்த ஆராய்ச்சியில் பயன்படுத்திக் கொள்ள சம்மதிக்கிறேன்.

இந்த ஆராய்ச்சி மையமும் ஆராய்ச்சியாளரும் என்னுடைய விபரங்கள் அனைத்தயும் இரகசியமாக வைப்பதாக அறிகிறேன்.

_____	_____	_____
நோயாளியின் பெயர்	கையொப்பம்	தேதி

_____	_____	_____
ஆராய்ச்சியாளரின் பெயர்	கையொப்பம்	தேதி

Annexures 5: Proforma

DEPARTMENT OF PERIODONTICS

TAMILNADU GOVERNMENT DENTAL COLLEGE AND HOSPITAL

CHENNAI – 600 003

ESTIMATION OF SERUM ALBUMIN LEVELS BEFORE AND AFTER PHASE I THERAPY IN PATIENTS WITH CHRONIC PERIODONTITIS

PROFORMA

OP No: DATE:

NAME: PATIENT ID No:

AGE/SEX:

ADDRESS:

MOBILE No:

OCCUPATION:

INCOME:

CHIEF COMPLAINT:

HISTORY OF PERSENTING ILLNESS:

PAST MEDICAL HISTORY:

PAST DENTAL HISTORY:

HISTORY OF HABITS:

INTRA ORAL EXAMINATION:

1) No. of teeth present:

2) Gingival examination

Colour

Contour

Consistency

Texture

Position

Pigmentation

Mobility :

Recession :

GINGIVAL BLEEDING INDEX

3																
B																
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38
B																
3																

CALCULATION

BASELINE

SCORE: **INFERENCE:**

3 MONTHS

SCORE: **INFERENCE:**

PROBING POCKET DEPTH AND CLINICAL ATTACHMENT LEVEL

3	CAL																
	PPD																
B	CAL																
	PPD																
3	CAL																
	PPD																
B	CAL																
	PPD																
		18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
		48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38
B	PPD																
	CAL																
3	PPD																
	CAL																
B	PPD																
	CAL																
3	PPD																
	CAL																

INVESTIGATIONS

Blood Investigation:

Hb count

Total Leucocyte count

Differential Leucocyte count

Bleeding Time

Clotting Time

Random Blood Sugar

Serum albumin level

SAMPLE	BASELINE	3 MONTHS
Serum albumin		

DIAGNOSIS

PROGNOSIS

TREATMENT

PHASE I:

S.NO	CALCULATIONS	BASELINE	3 MONTHS
1	Plaque index		
2	Gingival bleeding index		
3	Probing pocket depth		
4	Clinical attachment level		
5	Serum albumin		

SIGNATURE OF PG STUDENT SIGNATURE OF THE GUIDE

DATE