Biospectra : ISSN: 0973-7057, Vol. 18(2), September, 2023, pp. 249-254 **Medical Science**
An International Biannual Refereed Journal of Life Sciences

ISSN : 0973-7057

Evaluation of C-reactive protein in adult periodontitis : prior & later of non-invasive periodontal treatment

Shikha Sinha*, Padma R. & Vivek Kumar
Department of Periodontics, Awadh Dental College, Jamshedpur, Jharkhand, India

Received : 18th May, 2023 ; Revised : 17th June, 2023
DOI:- https://doi.org/10.5281/zenodo.12032426

Abstract- Human CRP is the prototypically intense stage reactant, the circulating congregation of which rises quickly and broadly in a cytokine-mediated reaction to tissue harm, contamination and aggravation, serum CRP values are routinely measured, experimentally, to identify and screen human maladies. In any case, CRP is likely to have critical have guard, rummaging and metabolic capacities through its capacity for calcium subordinate authoritative to exogenic and autologous iotas accommodate phosphocholine (PC) and after that sanctioning the classical complement pathway. CRP may moreover have pathogenic impacts and the later revelation of a prognostic affiliation between expanded CRP generation and coronary atherothrombotic occasions is of specific intrigued. The prevention of extracellular proteolytic enzyme, coagulation, fibrinolysis, adjust of secure cell work, the neutralization & removal of harmful elements from the circulation have all been revealed to be fundamentally dependent on the intense stage proteins. The Point of the think about was to assess C- receptive protein in unremitting periodontitis some time recently and after scaling and root planning. The study population included 30 patients who reported to the branch of Periodontology & Implantology, Awadh Dental college and Hospital, Jamshedpur, Jharkhand. Sample size was calculated using Yamane's formula. With respect to ethical approval, criteria for inclusion and exclusion included and study design was conducted. Selected patients were grant to two groups (group I & group II). clinical specification were recorded at baseline in group I & II at baseline and 3 months after SRP in group II. Significant difference ($p<0.01$) was found when baseline CRP values were compared with 3 months post therapy in Group II. The cruel CRP levels diminished with non-surgical periodontal treatment. This advantageous impact cannot be generalized as our think about did not incorporate patients with extreme periodontal pulverization. Inside the restrictions of the think about it can be draw the inference that periodontal treatment can be one of the points within the expectation of unfavorable cardiovascular circumstances.

Key words: Human CRP, extracellular proteolytic enzyme, Yamane's formula

INTRODUCTION

Chronic periodontitis is damaging incendiary infection of the supporting tissues of the teeth.[1] Thinks about demonstrate that the periodontal injury isn't entirely a restrain handle, instead it may lead to systemic changes within the secure work. It has been hypothesized that periodontitis either contributes to or tweaking portion in cardiovascular events and unfavorable pregnancy result.[2]

*Corresponding author :
Phone : 9304530283
E-mail : shikha.sinha14@gmail.com

The dissemination of plaque deduced antigens past the adjacent injury into the passage or arterial blood probably accounts for this.[3]

A few implements have been presented to clarify consonant hypotheses. One of them is predicated on the possibility that localized mediators like C-reactive protein (CRP), interleukins–1beta (IL-1ß) and –6 (IL-6) and tumor necrosis factor-alpha (TNF-a) could have an impact on the provocation of peiodontitis.[4] Human CRP is the classical intense stage reactant, the circulating concentration of

which rises quickly and broadly in a cytokine-mediated reaction to tissue harm, disease and irritation, serum CRP values are routinely measured, tentatively, to recognize and screen human maladies.[5] The intense stage proteins have been found to have an fundamental part within the restraint of extracellular proteases, blood clotting, fibrinolysis, tweak of safe cell work and the neutralization and clearance of hurtful components from the circulation. The union of these intense stage proteins has been appeared to be directed by cytokines and to a lesser degree by glucocorticoid hormones. The larger part of the intense- stage proteins are glycoproteins, which play a variety of parts within the homeostatic reaction to harm. The acute stage proteins in individuals differentiate considerably within the significance of their rise after onset of damage. The level of some of those proteins in the serum increments quickly amid contamination and concentrations can increment 2 to 100 crease and stay lifted all through contamination.[6]

CRP was found by Tillett and Francis in 1930.[7] The title CRP emerged since it was to begin with recognized as a substance within the serum of patients with intense aggravation that responded with the "c" carbohydrate counter acting agent of the capsule of pneumococcus.8 CRP could be a pentameric plasma protein with homologs that participate within the systemic reaction to irritation.[9] C-reactive protein could be a substance that the liver makes in reaction to aggravation.[10] A few thinks about propose that raised CRP levels reflect certain low-grade diseases related with coronary heart illness , like persistent periodontitis and contamination caused by cytomegalovirus and Chlamydia pneumonia.[11]

Rheumatoid joint torment, ankylosing spondilitis, Granulomatosis with polyangittis, systemic necrotizing vasculitis, Behcet's clutter, neonatal septicemia, systemic lupus erythematosus, post operator infection, thrombo embolic complications following major surgery, ulcerative colitis , myocardial localized necrosis have all been linked to elevated CRP levels.[12] CRP rise could be a portion of the intense stage reaction (non-particular) to intense and constant inflammation[13] while its diminishes in sum with die down of the infection and quiet recovery.[14] Numerous epidemiological thinks about have appeared that serum CRP levels were hoisted in patients with unremitting periodontitis as compared with unaffected control population.[15] CRP levels increment to hundreds of µg/ml inside hours taking after disease.[16]

It was proposed prior that one of the most capacities of CRP relates to its capacity to recognize outside pathogens particularly and to start their end by connection with humoral and cellular effector frameworks[17] CRP has both master provocative and hostile to-incendiary properties. It plays a part within the acknowledgment and clearance of remote pathogens and harmed cells by official to the phosphocholine, phospholipids, histone, chromatin and fibronectin.[18] The display consider was planned to assess the impact of periodontal treatment on serum CRP levels in patients with unremitting periodontitis. . Alterations were made for other variables that are known to be related with lifted levels of CRP such as age and body mass file (BMI) to way better disconnect the impacts of periodontal contamination on systemic C-reactive protein levels.

MATERIALS & METHODS

The think about populace included 30 patients who detailed to the department of Periodontology and Implantology, Awadh Dental college and Hospital, Jamshedpur, Jharkhand.

Ethical clearance for current study was given by the ethical committee of the institution.

The nature and intention of the study, the procedure involved, risks or benefits associated with study protocols were explained to each participant and an informed consent was obtained.

Inclusion criteria includes:

- Patients within the age range of 21 to 50 years.
- Patient having a least of 20 teeth.
- Patients with a sulcus depth of less than or equal to 3mm, no clinical attachment loss, bleeding on probing present in less than 20% sites- for control group.
- Patients having a Probing pocket depth of less than or equal to 4 mm and clinical attachment loss = 5mm in 2 or more sites in least of 3 quadrants -for test group.
- Patients Willingness to participate.

Exclusion criteria includes: -

- Patients using any form of tobacco.
- Patients with systemic disease such as coronary artery disease, hypertension, metabolic disorders like diabetes mellitus that can alter the course of periodontal disease.

- Patients having taken antibiotics /anti-inflammatory drugs in the preceding 6 months.
- Patients who had received periodontal therapy or extraction in the preceding 6 months.
- Pregnant women and nursing mothers.
- Patients with inability to comply with the follow up visit.

Study design:

Selected patients were allocated to two groups (group I & group II):

Group I (CONTROL GROUP)- 15 subjects with healthy gingiva, no change in color or edema and bleeding on probing in = 20% of site.

Group II (TEST GROUP)- consisted of 15 subjects diagnosed with chronic generalized periodontitis having a probing pocket depth of = 4mm and CAL = 5mm in 2 or more sites in minimum of 3 quadrants.

Clinical Parameters:

The following clinical specification were recorded at baseline in group I and at baseline and 3 months after SRP in group II:

- Oral Hygiene Index-Simplified by Greene and Vermilion (1964)[19]
- Sulcus bleeding index by Muhlemann H.R and Sons (1971)
- Gingival Index by Loe and Sillness (1963)[20]
- Plaque Index by Sillness and Loe (1964)[21]
- Pocket Depth (PPD)
- Clinical attachment loss (CAL)[22]

Study Design:

The selected patients were designated to two groups (control group and test group). Control Group consisted of 15 subjects with healthy gingiva, showing no change in color and bleeding on probing in =20% of sites. Test Group consisted of 15 patients diagnosed with chronic periodontitis having a pocket depth of =4mm and CAL =5mm in two or more sites in minimum of 3 quadrants. The following clinical specification were recorded in Group I at baseline & at baseline and 3 months after scaling and root planning (SRP) in Group II. Probing depth and CAL were measured using UNC 15 probe. All the measurements were done by a single operator.

Methodology:

A comprehensive (noninvasive) stage I treatment, which included tireless instruction and motivation, plaque control, scaling and root arranging (SRP) was performed within the test gather. Review visits were planned after 3 months to record all the clinical parameters. Blood tests were collected at pattern in both the bunches and 3 months post treatment in test bunch. Venous blood tests were gotten from the central veins through the skin within the cubital fossa. 3ml of blood was drawn employing a 5cc expendable syringe and 23 gage needles. The blood was at that point centrifuged at 3,000 r.p.m (transformations per miniature) for 5 mins and serum was isolated, from that point it was put away at -4^{o}C until advance examination. The test was defrosted at room temperature some time recently measure and all measure were performed in research facility. The serum hence gotten was utilized for testing the CRP levels utilizing QUANTIA CRP semi programmed analyzer which is based on agglutination response guideline.

Statistical Analysis

The information collected were subjected to statistical examination, utilizing the measurable bundle for the social science computer program for excel. Intergroup comparisons were made by one – way ANOVA taken after by Mann-Whitney test for pairwise comparisons. Categorical information was analyzed by Fisher's exact test. P value less than 0.05 was considered for significance.

RESULTS

Table 1- Comparison of parameters at baseline and after 3 months in test group.

	OHI	PI	GI	SBI
F-value	217.8	20.5	17.04	23.7
Z	4.6	3.5	2.9	3.1
P	<0.1	0.0001	0.0016	0.0001
SIGNIFICANCE	SIG	SIG	SIG	SIG

Significant difference were found when OHI, PI, GI & SBI was compared between baseline & 3 months post therapy in group II, showing p-value of <0.1,0.0001, 0.0016 & 0.0001 respectively (Table I).

Table 2- Comparisons of parameters of group I (control group) & group II (test group) at 3 months.

	OHI	PI	GI	SBI
F-value	2.7	128.5	140.5	114.4
Z	0.60	4.64	4.64	3.9
P	0.27	<0.0001	0.21	0.0001
SIGNIFICANCE	N.S.	SIGNIFICANT	N.S.	SIGNIFICANT

No significant difference was found in respect to OHI and GI, when the control group was compared to the 3 months post therapy results of test group. However, PI & SBI showed a significant difference with a p-value of <0.0001 & 0.0001 respectively (Table II).

Table 3- CRP values [Paired t test]

		Mean	S.D.	f-value	p- value
Group I		1.22	0.54	10.21	0.34
Group II at 3 months		1.78	0.38		**NON-SIGNIFICANT**
Group II	At baseline	2.28	0.55	35.78	<0.01
	At 3 months	1.78	0.38		**SIGNIFICANT**

No significant difference was found in CRP values of Group I and II at 3 months showing a p value of 0.34, however a significant difference ($p<0.01$) was found in CRP value when baseline values were compared with 3 months post therapy (Table III).

DISCUSSION

More seasoned age, repetitive bacterial diseases, and incessant bronchial aggravation are recognized chance components for "high-normal" readings of CRP within the common populace. Be that as it may, raised CRP levels have been seen in individuals who do not show up to have any known hazard variables for raised CRP, proposing that other obsessive conditions may constitute an extra jolt for a systemic fiery response.[23]

A hoisted serum concentration of CRP is proven of a dynamic tissue – harming prepares and CRP estimation in this way gives a straight forward screening test for natural infection and inflammatory diseases.[24] There's considerable prove to appear that CRP contributes directly to the pathogenesis of atherothrombosis. CRP may be a ligand official protein that ties to the cell layer of disturbed microbes and have cell. The complement cascade is activated when CRP binds to certain ligands. Additionally, macrophages, monocytes and neutrophils all contain CRP receptors. This bound CRP targets the bacterial and injured cells for phagocytosis subsequently intensifying the adjacent fiery response to the contamination.[25]

CRP advances artheroscelortic injury arrangement and actuates grip atom expression in known endothelial cells. Subsequently proposing the plausibility that atherosclerotic complication is straight forwardly related with periodontal infection.[26]

Prove appears that patients with genuine periodontitis have extended serum level of CRP when compared to control populace.[27] But the ponders drop brief in appearing that periodontitis was the cause for the observed serum CRP levels as CRP levels alter with various bewildering factors like maturing, tall blood weight, decrease physical development, tall protein thin down, alcohol, smoking and rest disturbance.[28]

Within the current think around an endeavor has been initiated to gage and contrast the level of C-reactive protein in sound controls and persistent periodontitis patients some time recently and after non-surgical periodontal treatment.

A critical diminish in CRP levels 3 months taking after the periodontal treatment was found in our think about which is in concomitance with the think about by D Aiuto *et al.* who watched a medium diminish in serum CRP of 0.5mg/l 6 months after periodontal therapy.[29] In differentiate to our think about Ide *et al.*[30] fizzled to watch a lessening in blood CRP levels following non-surgical periodontal therapy. Conceivable explanation for why CRP continues to rise after SRP is that SRP alone may not be adequate to control periodontal movement in all subjects, moreover SRP alone may not be able to kill profound situated miniaturized scale life forms. Evacuation of stores and smaller scale life forms in these regions is conceivable as it were with surgical mediation and /or antimicrobial specialists. Implies CRP levels were higher within the periodontitis patients compared to controls. Our ponder is comparable to that of Ebersole. However, Ebersole watched an awfully high-level CRP 9mg/dl in persistent periodontitis patients. This might be ascribed to more extreme cases of periodontitis in their think about.[31]

From the comes about of us ponder it can be concluded that a positive affiliation exists between the serum CRP levels and persistent periodontitis. The cytokines discharged beneath the condition of periodontitis are dependable for the generation of intense stage reactant like C receptive proteins. For periodontal treatment to have a vital effect on essential CRP it might take around 6 months. There's a plausibility of measurements reaction relationship between the degree of determination of the periodontal contamination and the lessening in systemic fiery markers.

Considers have recognized that periodontal treatment can be effective in diminishing CRP in as it were subset of patients, typically those with the highest serum CRP or scorching gist as determined by periodontal limiting factor at standard, those responding favorably to periodontal therapy, and those free of systemic diseases that can elevate CRP.

The size of expanded serum CRP as a result of periodontitis changes among the people. The result of periodontal treatment within the given people is hence altered by smoking, concurrent fiery conditions, standard serum CRP levels and adequacy of periodontal treatment. The result of the display considers reinforce the

ascertainment of the past considers demonstrating that periodontal infection is related with rise in serum CRP levels and non-surgical periodontal treatment such as SRP decreases their levels. Be that as it may, long term benefits of SRP diminishing the cardio vascular chance remains however to be documented by well controlled longitudinal ponders. Within the current consider systemic components known to extend CRP levels were avoided. Future considers in common populace with known chance figure for cardio vascular malady in periodontitis patients is fundamental since periodontitis may supplement to the total impact for expanding the chance.

CONCLUSION

Considerable prove budding within the final decade has shed light on the relationship connecting systemic health and verbal wellbeing. There's mounting prove that fiery instruments and nonstop contamination play a first portion in artherogenesis and cardio vascular infection. Atherogenesis could be a dynamic malady handle in which expansive to medium measured supply routes gotten to be blocked with fibro lipid injury. Inquire about has proposed that periodontal infection once stabilized attires an organic burden of endotoxin and inflammatory cytokines which assist escalating atherogenesis and thromboembolic procedures. C- responsive protein may be a strongly arrange reactant discharged by the body in response to emphatically harmed or provocative boosts and may be a preeminent reaction of the body to harm. The current considers envisioned to choose the effect of periodontitis and periodontal treatment upon the serum level of c- responsive protein. Interior they appear consider periodontal solid controls had lower brutal CRP levels as compared to patients with unremitting periodontitis. The brutal CRP levels diminished with noninvasive periodontal treatment. This productive influence cannot be generalized as our think around did not connect patients with extraordinary periodontal demolition. Inside the restrictions of the consider it can be concluded that periodontal treatment can be one of the focuses inside the maintain a strategic distance of contradicting cardiovascular occasions.

REFERENCE

1. **Pihlstrom B. L., Michalowicz B. S., Johnson N. W. 2005.** Periodontal diseases, *Lancet*. **19:**1-2 .
2. **Horne B. D., B. K. Yang, J. Ardans, G. Csako, L. M. Wahl. 2000.** Divergent effects of hormone therapy on serum markers of inflammation in post menopausal women with coronary artery disease on appropriate medical management, *J. Am Coll Cardiol.* **36 (6) :** 1774-80.
3. **Norman M. E., P. C. Baehni, C. C. Tsai, N. Stoller, W. P. McArthur, N. S. Taichman. 1979.** Studies of host responses during experimental gingivitis in humans, *J. Periodontol Res*. **14(5):** 361-69.
4. **M. Ide D, McPartlin P. Y., Coward M., Crook P., Lumb R. F. 2003.** Wilson Effect of treatment of chronic periodontitis on levels of serum markers of acute-phase inflammatory and vascular responses, *J. Clin Periodontol.* **30(4):**282 .
5. **D. Thompson, M. B. Pepys, S. P. Wood. 1997.** The physiologic structure of human CRP and its complex with phosphocholine Structure, *J Immunol*. **16 :** 77.
6. **Ebersole J. L. and Cappelli D. 2003.** Acute phase reactants in infections and inflammatory diseases, *Periodontology*. **23:** 19-49.
7. **Armitage G. C. 1999.** Development of a classification system for periodontal diseases and conditions, *Ann of periodontol.* **4(1):**1-6.
8. **Albandar J. M. & Rams T. E. 2000.** Global epidemiology of periodontal diseases : an overview. *Periodontol*. **29:**7-10.
9. **Tillett W. S. and Francis T. 1930.** Serological reactions in pneumonia with a non protein somatic fraction of pneumococcus, *J Exp Med*. **52:**561-71.
10. **Tushika Bansal & Anita Pandey. 2014.** CRP and its association with periodontal disease : A Brief Review. **8(7):** ZE21-ZE24.
11. **Langrand W. K., Vissr C. A., Hermans W. T. 1999.** C-reactive protein as cardiovascular risk factor.more than an epiphenomenon? , Circulation 100:96-102.
12. **Pepys M. B.1981.** C-reactive protein fifty years on , Lancet. **21:** 653-56.
13. **Mark B. Pepys and Gideon M. Hirschfield. 2003.** C-reactive protein :a critical update, *J. Clin Invest.* **11(12):** 1805-12.

14. **Gabay C., Kushner I. 1999.** Acute-phase proteins and other systemic responses to inflammation, *N Engl J Med* **340(6):**448–54.

15. **Isaac Suzart Gomes-Filho, Julita Maria Freitas Coelho, Simone Seixas da Cruz, Johelle Santana Passos, Camila Oliveira Teixeira de Freitas, Naiara Silva Aragão Farias, Ruany Amorim da Silva, Milena Novais Silva Pereira, Thiago Lopes Lima, Maurício Lima Barreto. 2011.** Chronic periodontitis and C-reactive proteins level. *J. Periodontol.* **82:**543-549.

16. **Ciubotaru I., Potempa L.A., Wander R. C. 2005.** Production of modified C-reactive protein in U937-derived macrophages. *Exp Biol Med*. **230(10):**762–70.

17. **R. G. Craig, J. K. Yip, M. K. So, R. J. Boylan, S. S. Socransky, A. D. Haffajee. 2003.** Relationship of Destructive Periodontal Disease to the Acute-Phase Response, *J Periodontol*. **74(7):**1007

18. **Volanakis J. E. 1982.** Complement activation by C-reactive protein complexes, *Ann NY Acad Sci.* **389 :**235-50.

19. **Soben Peter. 2013.** Essentials of public health dentistry, *5th edition Arya Medi.* 412-474.

20. **Loe H., Sillness J. 1963.** Periodontal disease in pregnancy. I. Prevalence and severity, *Acta Odontology Scand*. **21:** 533-551.

21. **Sillness J., Loe H. 1964.** Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition, *Acta Odontology Scand.* **22 :**121-135.

22. **Newmann M. G. Takei H., Carranza F. A., Klokkevold P. R.** Carranza's Clinical Periodontology, 10th ed U.S.A. WB. Saunders Company: 550-53 .

23. **Laiho K., H Mäenpää, H. Kautiainen, M. Kauppi, K. Kaarela, M. Lehto, E. Belt. 2001.** Rise in serum CRP after hip and knee artheroplasties in patients with rheumatoid arthritis, *Ann-Rheum Dis.* **3(60):** 275-7.

24. **Stephen K., Gordon Lowe, Mark B. Pepys, Simon G. Thompson, Emanuele Di Angelantonio, Rory Collins, John Danesh. 2010.** C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: an individual participant meta-analysis, *Lancet*. **375(9709):** 132-40.

25. **Wolfgang Koenig, M. Sund, M. Frohlich, H. G. Fischer, H. Lowel, A. Doring, W. L. Hutchison, M. B. Pepys. 1999.** CRP: a sensitive marker of inflammation predicts future risk of coronary heart disease in initially healthy middle aged men. *Circulation* **99(2):** 237-42.

26. **V. Pasceri, J. T. Willerson, E. T. Yeh. 2000.** Direct proinflammatory effect of C-reactive protein on human endothelial cells. *Circulation*. **102(18):**2165-8.

27. **Vanessa Machado, Joao Botelho, Claudia Escalda, Syed Basit Hussain, Shailly Luthra, Paulo Mascarenhas, Marco Orlandi, Jose Joao Mendes, Francesco D'Aiuto. 2021.** Serum C-Reactive Protein and Periodontitis: A Systematic Review and Meta-Analysis, Front. *Immunol.* **12 :** 7.

28. **Graziani F., Cei S., Tonetti M., Paolantonio M., Serio, R., Sammartino G., Gabriele M. and D'Aiuto F., 2010.** Systemic inflammation following non surgical and surgical periodontal therapy. *Journal of clinical periodontology,* **37(9):** 848-854.

29. **D' Aiuto F., Parkar M., Andreou G., Brett P. M., Ready D., Tonetti M. S. 2004.** Periodontitis and atherogenesis :casual association or simple coincidence? A pilot intervention study, *J Clin Periodontol*. **31:**402-11.

30. **Pedersen E.D. S R Stanke,S J Whitener, P T Sebastiani, B L Lamberts, D W Turner. 1995.** Salivary levels of alpha-2 macroglobulin, alpha-1 antitrypsin, CRP, Cathepsin G and elastase in humans with or without destructive periodontal disease, *Arch Oral Biol.* **40(12):** 1151-5.

31. **Slade G. D., S. Offenbacher, J. D. Beck, G. Heiss, J. S. Pankow. 2000.** Acute phase inflammatory response to periodontal disease in the US population, *J Dent Res.* **79:** 49-57.

***