VOLUME-8, ISSUE-2, FEBRUARY-2019 • PRINT ISSN No 2277 - 8160

Original Research Paper

Dental Science

EFFECT OF NON-SURGICAL PERIODONTAL THERAPY ON PLASMA LEVELS OF FIBRINOGEN IN PATIENTS WITH CHRONIC GENERALIZED PERIODONTITIS - A PILOT STUDY

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ABSTRACT

Background:
Emerging evidence from epidemiologic studies indicates that periodontal infection is associated with a moderate systemic inflammatory response . Fibrinogen is a marker of inflammation, and their systemic levels have been associated with coronary heart disease. This pilot study was carried for the assessment of whether non-surgical periodontal treatment is associated with changes in plasma fibrinogen levels in chronic generalized periodontitis.

Methods:
Five systemically healthy subjects with chronic generalized periodontitis participated in this one-month pilot study. Periodontal disease was measured through the clinical parameters including bleeding on probing (BOP), probing depth (PD) and clinical attachment loss. All subjects received non-surgical periodontal therapy that included scaling, root planning, oral hygiene instruction and chlorhexidine mouthwash. Systemic levels of inflammatory marker(Fibrinogen) were measured prior at baseline and 1 month after non surgical periodontal therapy. The collected blood samples were sent to the laboratory for estimation of plasma fibrinogen through STA analyser using commercially available STA fibrinogen test kit.

Results:
The baseline of plasma fibrinogen level in the selected sample was 408+/-150.7 mg/dl. After treatment, the plasma fibrinogen levels reduced to 271+/-25.2 mg/dl. Together with the positive changes in parameters of periodontal disease, Non-surgical periodontal therapy resulted in a significant reduction ($P < 0.05$) in the circulating levels of fibrinogen after one month of treatment.

Conclusions:
In this study, Non-surgical periodontal treatment resulted in significant decrease in plasma fibrinogen levels.

KEYWORDS : Non-Surgical Periodontal Therapy, Fibrinogen, Chronic Periodontitis.

INTRODUCTION

Periodontitis is a common, chronic infection of the supporting tissues of the teeth. Periodontitis has been proposed as having an etiological or modulating role in cardiovascular disease, diabetes, respiratory diseases and adverse pregnancy outcomes [1]. This could occur via the host immunoinflammatory mediators which are elicited in response to bacterial toxin or local tissue response to cytokines.

The initiation and progression of gingivitis and periodontitis may be affected by certain systemic conditions. On the other side of the relationship between systemic health and oral health has also been demonstrated. This means that there may be potential effects of periodontal disease on a wide range of organ systems. Emerging evidence from epidemiologic studies indicates that periodontal infection is associated with a moderate systemic inflammatory response [2,3,4].

In more severe forms of the disease the ulcerated epithelial lining of the periodontal pocket may constitute a substantial surface area through, which lipopolysaccharide and other bacteria derived antigenic structures challenge the immune system, triggering the synthesis of several proinflammatory cytokines.

One of the important cytokines, interleukin-6 (IL-6) named as messenger cytokine can travel from local inflamed tissue to the liver where it initiates a change in the program of protein synthesis from the housekeeping protein albumin to other proteins, characteristic of the acute phase response, including fibrinogen and haptoglobin [5].

Patient suffering from severe periodontitis have increased local production of inflammatory cytokines (IL-7, TNF, and IL-6) and moderate systemic inflammatory response, which is defined by raised concentration of CRP, haptoglobin, fibrinogen and moderate luecocytosis.

Fibrinogen (Factor I) is a soluble, 340 kDa plasma glycoprotein that is converted by thrombin into fibrin during blood clot formation. Fibrinogen is synthesized in the liver by the hepatocytes. The concentration of fibrinogen in the blood plasma is 1.5-5.0 g/day. Fibrinogen is the major coagulation factor in the plasma. Low plasma fibrinogen concentrations are therefore associated with an increased risk of bleeding due to impaired primary and secondary haemostasis. Fibrinogen is a classical positive acute-phase reactant protein and is an independent predictor of chronic generalized periodontitis [6].

Chronic generalized periodontitis is shown to raise inflammatory markers such as fibrinogen in the blood. Based on cross sectional and intervention studies, periodontitis has been linked to raised level of fibrinogen, however the mechanism responsible for this association is obscure.

This pilot study was conducted to investigate the association between periodontal disease and plasma fibrinogen by comparing

the levels of fibrinogen before and after non-surgical periodontal therapy.

MATERIAL AND METHODS

Study design and experimental population

Participants were selected from the patients referred for the treatment of periodontitis in the Division of Periodontology from Department of Dental Surgery, AFMC, Pune. Five systemically healthy subjects with generalized chronic periodontitis in the age range of 19–65 years; with ≥ 2 mm clinical attachment loss were included in this pilot study after obtaining the informed consents. Periodontal parameters and inflammatory markers [plasma fibrinogen] was evaluated at baseline and one month after delivery of intensive non-surgical periodontal therapy. Patients with known history of current smoking, smoking within the last 6 months, alcohol consumption or drug abuse, diabetes, or any other acute/chronic systemic diseases, subjects taking antibiotics or anti-inflammatory drugs previous six months or treatment with any medication affecting the serum level of inflammatory makers such as hormone replacement and steroids, statins, immunosuppressants and anti-coagulants.; and pregnancy/lactation were excluded. Ethical approval and permission for conducting of the study were obtained from ethical committee of the college.

Data Collection:

All relevant data including demographic factors, social history, medical and dental history was recorded and a full mouth periodontal examination was performed in each subject at baseline and one month after the non-surgical periodontal therapy. Florida probe system was used to obtain the clinical measurements.

1. Bleeding on probing
2. Probing pocket depth
3. Clinical attachment level

Sample Collection:

The Plasma samples (10ml of blood) were collected from all the selected patients from the antecubital vein using a vacutainer and standard aseptic precautions at baseline (Fig 1). Blood was collected in tubes with clot activator, 3.2% sodium citrate, or EDTA, for fibrinogen. Sample for fibrinogen was centrifuged and stored (-20C) until analysis. Fibrinogen (mg/l) was analyzed using a commercially available kit. All the selected patients underwent non-surgical periodontal therapy and were given similar homecare instructions which also included the use of 0.2% chlorhexidine mouthwash. The serum samples after non-surgical periodontal intervention were collected after 1 month and the levels of plasma fibrinogen were assessed.

Analysis:

The fibrinogen was estimated through STA analyser using commercially available STA- fibrinogen test kit (Fig 2).

Statistical analysis

All the clinical parameters and the grades of plasma fibrinogen levels (obtained by STA analyser of blood samples of periodontally compromised patients before and after non surgical periodontal therapy) were tabulated and compared [Table 1]. The same data for CAL has been visually represented in the form of a graph [Graph 1]. The comparisons between the parameters were also analyzed and statistical significance assessed.

RESULT

The periodontal parameters recorded showed statistically significant improvement after non-surgical periodontal therapy. The baselines of plasma fibrinogen in the selected were 408+/-150.7 mg/dl. After treatment, the plasma fibrinogen level reduced to 271+/-25.2 mg/dl. This was statistically highly significant (P <0.05) using Bon ferroni probability tests [Table 2]. The same data has been visually represented in the form of a graph [Graph 2]. The treatment had an effect on the plasma fibrinogen level for all the selected subjects.

DISCUSSION

The present study aimed at examining the effect of non-surgical periodontal therapy on the level of systemic inflammatory indicators, which also play an important role as risk markers for periodontal disease. We showed that eradication or suppression of periodontal pathogens not only from the periodontal pockets but also from all their intra-oral habitats (mucous membranes, tongue and saliva) by applying full mouth disinfection in a relatively short time period [7], was associated with a significant decrease in plasma fibrinogen levels, otherwise healthy individuals affected by chronic generalized periodontitis.

In a recent case-control study, Vidal et al [8] measured the serum level of CRP, IL-6 and fibrinogen after non-surgical periodontal therapy which affected by chronic generalized periodontitis. They reported significant decrease in plasma variables in 3 months after intervention in addition to the improvement of periodontal clinical results they had reached.

Accumulating evidence has shown that treatment of chronic generalized periodontitis is associated with subsequent changes in the level of serum inflammatory markers [9]. However, Behle et al [10] reported a negative or no impact of periodontal therapy on the levels of serum inflammatory markers.

CONCLUSION

In the present study, non-surgical periodontal therapy resulted in significant decrease in plasma fibrinogen levels. Due to the small sample size, this study results obtained cannot be extrapolated on to a large population, for which multicentric longitudinal studies are recommended with large sample sizes.

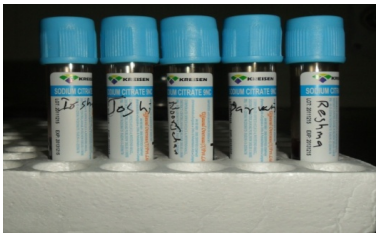
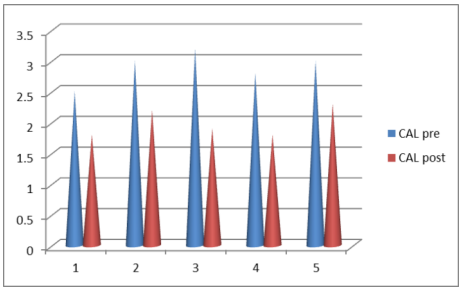


Fig 1

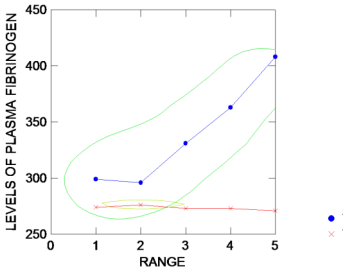
Fig 1: Vacutainer containing blood

Fig 2: STA analyser



Graph 1 : CAL

GRAPHICAL REPRESENTATION



Graph 2 : Plasma fibrinogen level

Table 1: Clinical Parameters recorded of all the subjects

Subjects	Bleeding on probing (avg)		Probing Depth (mm)		CAL (mm)		Plasma fibrinogen(mg/dl)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
A	46 %	26%	4.5	3.8	2.5	1.8	299	274
B	50%	25%	5	4.2	3	2.2	296	276
C	60%	28%	5.2	3.9	3.2	1.9	331	273
D	55%	30%	4.8	3.8	2.8	1.8	363	273
E	50%	26%	5	4.3	3	2.3	408	271

Table 2: Statistical analysis:

Clinical parameters	Standard Deviation	t -Value	p -Value	dunn sidak	
Bleeding on probing	4.324	13.031	< 0.05	< 0.03	significant
Probing depth	0.255	7.94	< 0.05	< 0.02	significant
CAL (Bon ferroni)	0.255	7.984	< 0.05	< 0.01	Significant
Plasma fibrinogen	48.68	3.023	<0.03	<0.04	significant

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