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Comparative Evaluation of Salivary Visfatin Concentration in Patients with Chronic Periodontitis and Aggressive Periodontitis

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Abstract

Introduction: Periodontal disease is a local inflammatory reaction that affects and destroy supporting tissues of the teeth, leading to bleeding, resorption of alveolar bone and loss of teeth attachment. The use of biomarker- based disease diagnostics in identification of active periodontal disease from plaque biofilms, Gingival Crevicular Fluid, saliva and serum is one of the recent advancements in the field of periodontal diagnostics. **Aim and Objectives**: This clinical study aimed to evaluate and compare unstimulated salivary Visfatin concentration in patients with chronic periodontitis and aggressive periodontitis.

Material and methods: The study was conducted on seventy-five (75) selected subjects in the age group of 25 -55 years divided into three groups. The clinical Parameters i.e. Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL) were recorded. The salivary Visfatin levels were evaluated in the unstimulated saliva sample. **Results:** The study showed that salivary Visfatin level is associated with periodontal inflammation and altered host response. Therefore, the salivary Visfatin can be considered as an important biomarker in evaluation of periodontal health and disease.

Keywords: saliva, visfatin, inflammation, aggressive periodontitis

Introduction

Periodontal disease is a multifactorial inflammatory disease in which cytokines including IL–6 and TNF- α,

chemokines, arachidonic acid metabolites and inflammatory mediators including proteolytic enzymes are released as a result of the activities of the tissue defense systems against the microbiological agents. These released molecules indirectly cause soft tissue degradation and bone resorption in the host tissue.[1]

Traditionally used clinical diagnostic parameters like bleeding on probing pocket depth, bone loss, gingival inflammation and plaque index criteria are often insufficient for determining active disease sites for monitoring quantitative response to therapy or for measuring the degree of susceptibility to future disease progression.[2] They have several limitations like requirement of highly trained clinician, use of expensive radiographic equipment, more procedure time and intensive labour. With the evolution of biomarkers like matrix metalloprotienases (MMPs), Collagenase-2 (MMP-8), Osteopontin, Fibronectin, Platelet derived growth factor and Visfatin, these limitations have been overcome.

Biomarkers are synthesized in periodontal tissues and different body tissues, one of which is adipose tissue. Adipose tissue synthesise many inflammatory factors, including adiponectin, resistin, leptin, cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) and Visfatin.[3] Visfatin is considered an inflammatory adipokine that is available in inflammatory cells and inflammatory conditions. For example, the expression of Visfatin increases in acute and chronic inflammatory conditions like rheumatoid arthritis, sepsis, acute lung injury, inflammation, inflammatory bowel disease and psoriasis and plays an important role in the persistence of inflammation through its ability to inhibit apoptosis of neutrophils.[4] Various biological media like saliva, GCF and serum are used to determine biomarkers in periodontal health and disease.[5]

Saliva is used as a diagnostic tool to evaluate various biomarkers associated with periodontal disease.[6] In terms of periodontal diagnosis, whole saliva has elements that reflect the activity of all periodontal sites and therefore provide an indication of disease status in the mouth as a whole, in contrast to the site-specific GCF analysis. Recent studies have shown that Visfatin acts as a biomarker in saliva and also is used to detect the periodontal disease activity.[7] Thus, Visfatin with its multitude of inflammatory functions is an ideal candidate biomarker for investigation in periodontitis.

The aim of the present study was to assess the concentrations of human salivary Visfatin in saliva and to do comparative evaluation of salivary Visfatin in chronic periodontitis and aggressive periodontitis patients.

Materials and Methods

A total of 75 subjects, 25 patients with generalized chronic periodontitis (CP), 25 patients with generalized aggressive periodontitis (AP) and 25 individuals with clinically healthy periodontium in the age group of 18-55years were selected using from the outpatient department of Periodontics & Implantology, Guru Nanak Dev Dental College and Research institute, Sunam, Punjab. The study was approved by the ethical committee of the institute and written informed consent was obtained from all the subjects.

Inclusion Criteria

- Systemically healthy individuals
- A minimum of 18 teeth should be present
- Age: 18 to 55 years
- Patients with BMI < 25kg/m2
- Patients with generalized Chronic periodontitis were diagnosed by clinical attachment loss ≥3mm and pocket depth (PD)≥5 mm in at least more than 30% sites involved with consistent amount of microbial deposits.

- Patients with generalized Aggressive periodontitis were diagnosed by generalized interproximal attachment loss ≥3mm affecting at least three permanent teeth other than 1st molars and incisors with inconsistent amount of microbial deposits.
- In individuals with plaque and gingival index of 0-1 were selected.

Exclusion Criteria

- History of any periodontal therapy 6 months prior to study
- Smokers and tobacco chewers
- Liver or kidney dysfunction
- Salivary gland dysfunction
- Any systemic disease
- Patients on any medications
- Pregnant or lactating mothers

Armamentarium used included mouth mirror, UNC 15 Probe, Explorer, Tweezer, Gloves, Mouth mask, UV sterilized cups, Eppendorf tubes, ELISA kit (Sincere Biotech Human Visfatin ELISA kit), Centrifugation machine. Clinical parameters were assessed using Plaque Index, Gingival Index, Periodontal Probing Depth and Clinical Attachment Loss.[8,9,10,11]

Probing pocket depth was measured from the crest of the marginal gingiva to the base of the pocket using UNC-15 graduated periodontal probe at the selected site. Clinical Attachment Loss was calculated from a fixed reference point cementoenamel junction (CEJ), and it was computed by calculating the distance from the CEJ to the base of the pocket.

Study Design

A case-control cross-sectional study was carried out to compare salivary Visfatin concentration between clinically periodontally healthy individuals, Chronic periodontitis patients and Aggressive periodontitis patients.

Total 75 subjects were clinically evaluated and were divided into three groups.

- GROUP I (CONTROL) (N=25) – Patients with healthy periodontium.
- GROUP II (N=25) - Patients with generalized Chronic periodontitis.
- GROUP III (N=25) - Patients with generalized Aggressive periodontitis.

Clinically measurements were performed at four points per tooth using a probe and recorded in a chart. Patients in the test group had a diagnosis of generalized Chronic Periodontitis and Aggressive periodontitis. This diagnosis was established from clinical parameters, including plaque index (PI), gingival index (GI) Probing pocket depth (PPD) and clinical attachment level (CAL).

Method for Saliva Sample Collection

Saliva sample were collected according to circadian rhythm. Patients were refrained from eating or rinsing 60 minutes before the collection of saliva sample. The whole saliva was collected into UV sterilized cup by letting the saliva accumulate in the mouth and then spitting out into the cup. About 5 ml of unstimulated saliva was collected from each patient and samples were immediately placed on cryobox maintained at -20°C and taken to lab where it was stored at -80° C until further determinations were done. Samples were thawed and were analysed within 6 months of collection.

Visfatin Analyses

Each saliva sample (5ml) was pipetted into a clean microcap tube and clarified by centrifugation at 10,000 x g for 5 minutes. The supernatant was transferred to clean microcap tubes and used immediately for an enzyme–linked immunosorbent assay (ELISA). Concentration of Visfatin were determined using an ELISA kit, according to the manufacturer's instructions. The ELISA was performed by using a commercial ELISA kit, SINCERE

BIOTECH Human Visfatin (VF) ELISA kit (PHOTOGRAPH NO. 4) which was specific for human salivary Visfatin. The result of Visfatin assay were expressed as nanograms per milliliter for concentrations. The data thus collected was computed and put to further statistical analysis using SPSS 20 software.

Statistical Analysis

Mean, standard deviation and confidential interval of data were evaluated using descriptive statistics. Statistical parameters were set at standard deviation (SD) of 15, 5% risk and 80% power. Chi square test was used to compare the gender distribution amongst three groups. One way ANOVA and POSTHOC Tukey's test was used to compare age, mean plaque score, mean gingival index score, mean probing pocket depth, mean clinical attachment level and Visfatin levels between three groups. Pearson's correlation test was used to correlate the Visfatin levels with age, mean plaque score, mean gingival index score, mean probing pocket depth, and mean clinical attachment level in all the three groups. For statistical analysis SPSS 20 software was used.

Results

Graph 1 shows that among 75 subjects there were total of 43 females and 32 males. Of these in group I, there were 16 females and 9 males; in group II there were 13 females and 12 males; in group III there were 14 females and 11 males.

When the mean plaques scores were compared statistically within the three groups using ANOVA, the difference was found to be statistically significant (Table 1). Table 2 shows the statistical evaluation & comparison of mean gingival index was done using ANOVA. From statistical analysis the difference in mean gingival index among all the groups was statistically significant (P<0.001). Graph 2 shows the distribution of mean periodontal probing depth in the three groups in the study showing highest values in group C while lowest values in group A. Applying post-HOC Tukey's test on the values, inter group comparison of mean periodontal probing depth showed significant statistical differences (Table 3). Table 4 shows that difference between the three groups was found to be statistically significant when the salivary Visfatin levels were compared using ANOVA. Table 5 shows that on applying post-HOC Tukey's test on values, the difference was found to be statistically significant. Correlation of Visfatin levels with other clinical parameters did not yield statistically significant results in the study.

Graph 1:

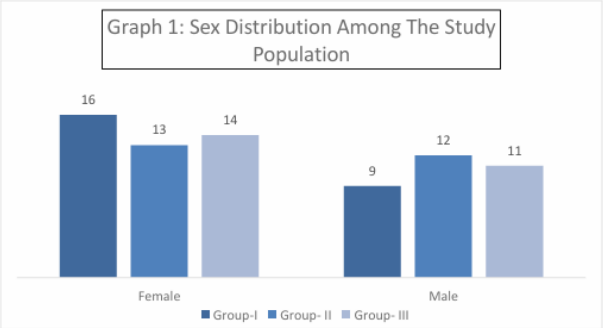

Table 1: Comparison of Mean Plaque Scores within all groups

	GROUPS	N	Mean	Std. Deviation	Welch Statistics/F (ANOVA)	P VALUE
MEAN PLAQUE SCORE	GROUP- I	25	0.1896	0.141759	160.319	<0.001 *HS
	GROUP- II	25	1.758	0.439858		
	GROUP- III	25	1.0412	0.550986		

*HS- Highly Significant

Table 2: Comparison of mean Gingival Index Score within all groups

	GROUPS	N	Mean	Std. Deviation	Welch Statistics/F (ANOVA)	P VALUE
MEAN GINGIVAL INDEX SCORE	GROUP- I	25	0.1048	0.222788	266.891	<0.001 *HS
	GROUP- II	25	2.1584	0.428356		
	GROUP- III	25	1.4348	0.427679		

*HS- Highly Significant

Graph 2: Distribution of Mean PPD among three groups in study.

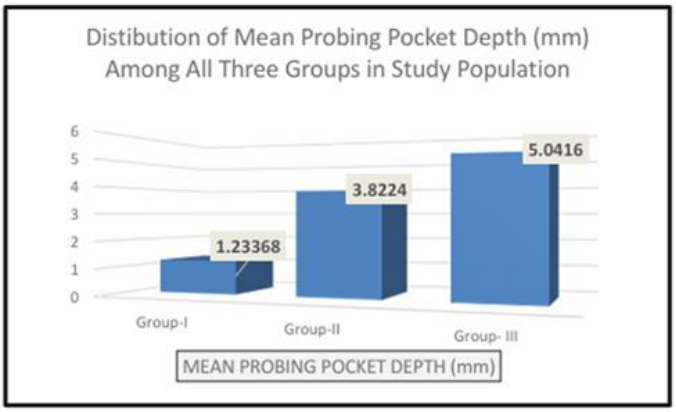

Table 3: Inter-group comparison of mean probing pocket depth (mm) between all three groups

Dependent Variable	Variable – I	Variable-J	Mean Difference (I-J)	Std. Error	P VALUE
MEAN PROBING POCKET DEPTH (mm)	GROUP - I	GROUP- II	-2.5887200	0.307845	<0.001 *HS
		GROUP - III	-3.8079200	0.307845	<0.001 *HS
	GROUP- II	GROUP- III	-1.2192000	0.307845	0.001 *HS

*HS- Highly Significant

Table 4: Comparison of salivary Visfatin level (ng/ml) between all three groups

	GROUPS	N	Mean	Std. Deviation	Welch Statistics/F (ANOVA)	P VALUE
VISFATIN LEVEL (mg/ml)	GROUP-I	25	22.04832	3.458296	209.22	<0.001 *HS
	GROUP-II	25	32.70616	0.892706		
	GROUP-III	25	34.59176	1.908517		

*HS- Highly Significant

Table 5: Inter-group comparison of salivary Visfatin level (ng/ml) between all three groups.

Dependent Variable	Variable-I	Variable-J	Mean Difference (I-J)	Std. Error	P VALUE
VISFATIN LEVEL (ng/ml)	GROUP-I	GROUP-II	-10.6578400	0.661295	<0.001 *HS
		GROUP-III	-12.5434400	0.661295	<0.001 *HS
	GROUP-II	GROUP-III	-1.8856000	0.661295	0.015 *S

*HS- Highly Significant

*S- Significant

Discussion

Visfatin, also known as pre-B-cell colony enhancing factor/nicotinamide phosphoribosyltransferase (NAMPT), is a adipokine released from adipose tissue, macrophages and leukocytes.[5] It is an inflammatory mediator that induces the synthesis of proinflammatory cytokines such as TNF-α, IL-6 and acts as a chemotactic factor. It induces the expression and activity of matrix metalloproteinase (MMP) and conversely, down regulates the inhibitors of the MMP's in monocytes and endothelial cells. Any imbalance between these MMP's and their inhibitors play a key role in the progression of periodontitis.[12] Visfatin can be of diagnostic and therapeutic target for periodontal tissues.[13] Ozcan et al (2015) proved positive correlation of periodontal pathogens like porphyromonas gingivalis, prevotella intermedia and EBV with salivary Visfatin level.[1] Cytokine changes in oral diseases can be detected in various fluids like serum, GCF and saliva. It was indicated that Visfatin is identifiable and measurable in saliva. In comparison to GCF, the collection of saliva is a convenient procedure and the technique is non invasive to diagnose periodontal disease. Abolfazli N et al in 2015 also concluded that the changes in salivary Visfatin levels were more prominent than serum levels.[14] Hence, saliva was used for bio marker analysis in the current study.

Visfatin stimulates the production of C-C motif chemokine ligand 2 and matrix metalloproteinase-1 in the periodontal ligament cells which leads to inflammation of periodontium and destruction of connective tissue and this could be the reason behind increased value of GI, PI, PPD and CAL gradually from Group-I to Group-II and Group-III in this study. Ozcan et al (2015) also evaluated the correlation between clinical parameters & salivary Visfatin and concluded that salivary Visfatin levels were

positively related to PI and GI.[1] In periodontitis patients, P.gingivalis increases in number and it affects many cytokine release such as TNF-α ,IL-1β and IL-6 which upregulates the release of Visfatin indirectly. A positive correlation is found between clinical attachment level and salivary Visfatin concentrations in Chronic and aggressive periodontitis patients as Visfatin synthesis is modulated by microbial infection.

On comparison of probing pocket depth and clinical attachment level between the groups, it was found that in Aggressive periodontitis (Group III) both the parameters were significantly higher as compared to other groups. Similar findings were found in study by Kumar V et al 2019.[15]

The reason of increased probing pocket depth and clinical attachment level is due to increase severity of disease in aggressive form as a result of altered immune response and increased number of microbial pathogens. The findings of Pradeep et al, Wu Y et al & Ozcan et al also noticed that clinical periodontal parameters correlated positively with Visfatin levels.[1,16,17]

The result of this study indicates that concentrations of Visfatin in saliva increased proportionately with the severity of the disease. In addition, the proportionate increase in the Visfatin levels from healthy gingiva to periodontitis in saliva confirms that Visfatin was actively secreted by predominant cells of periodontal disease activity such as polymorphonuclear leukocytes and neutrophils. This result is in accordance with a study done by Ozcan et al (2015) and in which the salivary Visfatin levels were found higher in periodontitis patients as compared to subjects with healthy gingiva.[1] The results obtained in the current study were in concurrence with past studies authenticating the association of salivary Visfatin and periodontal disease.

Limitations

Since it was a cross-sectional study, it could not determine disease activity. Secondly, the subjects in chronic periodontitis group were not divided into sub group of mild, moderate and severe forms which can lead to a bias during comparison of severe chronic periodontitis with aggressive periodontitis as per described in American Academy of Periodontology (AAP) classification of 2017. Thirdly, the subjects of each group were not matched in age which leads to a confounding bias. Fourth, we did not have information about the levels of Visfatin after the treatment of periodontitis such information would be useful to understand the role of Visfatin in pathogenesis of periodontal diseases. So, further long term and interventional studies are required to assess the efficacy of salivary Visfatin as a bio-marker for early detection of periodontal disease and prevention of its progression.

Conclusions

The following conclusions can be drawn:

- Clinically, periodontal inflammation can be positively correlated with the progression of periodontal disease with maximum inflammation found in generalized chronic periodontitis patients.
- Maximum probing depth and clinical attachment loss with inconsistent plaque and inflammation was found in patients with aggressive periodontitis.
- Salivary Visfatin level was found to be highest in patients with aggressive periodontitis followed by chronic periodontitis and least in clinically periodontally healthy individuals.
- A positive correlation was found between periodontal destruction and Visfatin levels in aggressive periodontitis patients but intergroup difference was found to be non- significant.

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