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SALIVARY PAF LEVELS IN EARLY ONSET AND ADULT PERIODONTITIS PATIENTS THROUGHOUT INITIAL PERIODONTAL THERAPY

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INTRODUCTION

Periodontal diseases reflect a constellation of inflammatory mediators which act individually or synergistically to promte disease progression.[1]

Bacteria or their products and components are the driving force behind the observed tissue destruction. Substances from periodontopathic bacteria initiate and drive the inflammatory response and their continued presence is essential for maintenance of the inflammation. Nevertheless, endogenous molecules mediate the inflammatory process and play a major role in its ampification and perpetruation and in the ensuing tissue destruction.[2]

Cellular response to inflammation involves the formation and accumulation of bioactive mediators. Platelet activting factor (PAF) is among the most potent of these mediators, as it leads to cell damage through several mechanisms.[3]

PAF is a family of structurally related, acetylated phospholopids capable of inducing marked pro-inflammatory responses.[4,5] Although originally named for its ability to cause aggregation and histamine release from rabbit platelet,[6] PAF has since been documented to promote a wide range of phlogistic processes which are initiated via specific PAF receptors on various cells and tissues. These processes include the stimulation of diverse targets and effects, such as polymorphonuclear leukocyte (PMN) activation (e.g. chemotaxis, aggregation, lysosomal enzyme release, arachidonic acid metabolism, and superoxide production), monocyte macrophage aggregation and phagocytosis, eosinophil activation, increased vascular permeability, vasoconstriction, and smooth muscle contraction.[4,5,7]

PAF is rapidly synthesized by various inflammatory cells after activation by either immunologically or nonimmunologically triggered signals.[8] Interestingly, PAF is produced by a variety of activated inflammatory cells including many of the same cells which it targets, such as PMN, vascular endothelial cells, monocytes, eosinophils, basopils, platelets and lymphocytes. Thus the pleiotropic effects of these acetylated phospholipids develop as a result of paracrine

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and autocrine stimulation of the inflammatory process.[1]

The presence of PAF in normal human mixed saliva was first reported in 1981 by Cox et al.[9] Pure parotid saliva apparently has no detectable PAF activity, which suggests that PAF in mixed saliva originates from a source other than this salivary gland.[9] Moreover, edentulous, healthy subjects have undetectable or significantly decreased levels of salivary PAF.[10] In combination these results suggest that PAF in mixed saliva may be derived from periodontal tissues.[8]

Subsequent investigations indicate that the gingival crevice appears to be the source of PAF in normal human mixed saliva.[10] Consistent with these observations, the presence of PAF in gingival tissues and crevicular fluid has been associated with clinical signs of periodontal inflammation.[11,12,13]

Salivary PAF levels in periodontitis patients have been correlated with the extent of periodontal disease.[8] Similarly, the levels of PAF in saliva from patients with refractory periodontitis were elevated in comparison to patients who had responded to conventional periodontal therapy and maintenance.[14] Thus a number of separate studies provide the basis for suggestion that PAF a proinflammatory phospholipid autacoid may be involved in periodontal tissue injury and disease.

The crosssectional studies outlined above indicate that the levels of PAF is saliva are correlated with the extent of periodontal disease. However, longitudinal studies to assess the effect of periodontal treatment on salivary PAF in periodontitis patients have been insufficient. The purpose of this study is thus to evaluate salivary PAF levels throughout initial peiodontal treatment in patients with early onset and adult periodontitis in relation to clinical parameters of the diseases.

MATERIALS AND METHODS

Human subjects:

The subjects of this study were divided into two groups, as follows:

Group I (subdivided into):

Study Group I: Ten early onset periodontitis patients with radiographic evidence of alveolar bone loss, of ages ranging from 19 to 29 years.

Control Group I: Ten healthy control subjects matching their study group in age and sex, enjoying clinically healthy gingiva and no radiographic evidence of bone loss.

Group II (subdivided into):

Study Group II: Ten chronic adult periodontitis patients diagnosed through clinical and radiographic examinations of ages ranging from 35 to 50 years.

Control Group II: Ten healthy control subjects with clinically healthy gingiva and no radiographic evidence of bone loss, matching their study group in age and sex.

The medical and dental history of each subject were reviewed to exclude those suffering from systemic illness. Patients having been subjected to antibiotics or

periodontal treatment (except plaque control) in the past three months were excluded from this study.

Clinical evaluation:

A single examiner performed comprehensive clinical periodontal examination at the initial appointment, at the middle of the study, and after complete scaling and root planing.

The following data was collected for each patient.

1. Plaque index: defined as the percentage of surfaces with plaque.
2. Probing depth to the nearest mm, defined as the distance from the gingival margin to the base of the probeable pocket.
3. Clinical attachment level to the nearest mm, defined as the distance from the cementoenamel junction to the base of the probeable pocket. Both probing depth and attachment level were recorded at six locations per tooth, four proximal (mesiobuccal, mesiolingual, distobuccal, and distolingual), midfacial, and midlingual.
4. The Eastman interdental bleeding index (EIBI) was utilized as a measure of gingival inflammation.[15] A wooden interdental cleaner was inserted between all teeth from the facial aspect and the papilla was depressed one to two mm. The tip of the cleaner was slightly angled (coronally) in order to prevent undue trauma to the gingiva. The cleaner was inserted and withdrawn into each interdental space four times, bleeding within 15 seconds was considered a positive response. The EIBI was calculated as the percentage of sites with bleeding in comparison to the total sites examined.

Treatment:

Treatment of each subject consisted of:

1. Detailed home care instructions emphasizing tooth brush and interdental cleaner.
2. Supragingival scaling with scalers and curettes and a rubber cup polish.
3. Subgingival scaling and root planing with local anaesthesia.

All subjects were treated using ultrasonic and hand instruments. Scaling and root planing was considered complete when clinical assessment using a probe and explorer revealed all teeth to be free of plaque and calculus and roots to be smooth and hard. Therapy was generally accomplished over four weeks.

Collection of saliva samples:

At the beginning of every appointment, one ml of unstimulated mixed saliva was collected directly into a 16 X 100 mm disposable glass tube containing 1.25 ml of chloroform and 2.5 ml of methanol. Thus the final ratio of chloroform to methanol to saliva was 1:2:8 V/V/V. Tubes were immediately capped and shaken vigorously to assure complete mixing prior to storage at 4°C.

Participants were instructed to refrain from smoking, performing oral

hygiene measures, rinsing, eating, drinking or taking anything by mouth for one hour before saliva sample collection.

Phospholipid extraction:

Phospholipid in saliva samples were extracted as described by McManus et al in detail.[10] One hour following extraction at room temperature, samples were centrifuged (1000 xg, 10 minutes at room temperature). The supernatant was decanted into a 16 X 100 mm disposable glass tube containing chloroform and water (to achieve a final ratio of chloroform to methanol to water-saliva of 1:1:9 V/V/V, salivary lipids, including PAF were present in the lower, chloroformrich (phased chloroform) layer which was stored at -20°C until phospholipid fractionation by thin layer chromatography.

PAF determination by high performance thin layer chromatography (HPTLC):

Two basic techniques can be used for quantitative thin layer chromatography (TLC). In the first technique the substances to be determined are assayed directly on the layer. In the second, the substances are removed from the layer and assayed, generally spectorphotometrically. When substances are assayed directly on the layer, there are no transfer or extraction errors, and the procedures are quite simple. Unfortunately, the quantization methods that one must use are not especially accurate, and overall errors vary between 5 and 10%. Spots on a TLC can be quantized spectroscopically by transmission or reflectance. In the transmission mode, the layer is passed through a beam of light, and the transmitted energy is measured by the Camag TLC scanner or densiometer (Wilmington, NC, USA).

The chloroformic layer was dried under nitrogen and then dissolved in 1 ml chloroform and finally cleaned up by Sep-Pak columns (Waters, Milford, MA, USA) and 3 ml of acetonechloroform (2:3) was added to sep-pak column. The eluent was dried under reduced pressure at room temperature. The eluted extract was then quantitatively transferred with chloroform into a small vial and evaporated until dryness under nitrogen.

The residue was redissolved in 100µl chloroform and spotted on precoated TLC and HPTLC (Merck, Germany) using chloroform: methanol: water (65:53:6 V/V/V) as the developing solvent. Authentic (1-O-Hexadecyl-2-acetyl-sn-glycero-3-phosphocho-line; PAF (C16); 1-O-palmityl-2-acetyl-sn-glycero-3-phosphocholine (CALBIOCHEM. La Jolla, Ca, USA), was dissolved in chloroform to produce a concentration range of 2.5 to 50 fmoles and stored at -20°C.*

Salivary PAF was measured by comparing it to the known quantity of authentic PAF. PAF concentrations in

*N.B: Fmole = 10^{-15} mole and pmole = 10^{-12} mole.

TLC fractions of unknown samples was calculated directly from the PAF C_{16} standard curve and expressed pmole equivalent of PAF C_{16}/ml saliva. Total PAF for a given saliva sample represented the sum of PAF activity detected in all TLC fractions for that sample.

Statistical analysis of the data:

Data are presented as the mean ± standard deviation (SD) of the mean. Descriptive statistics were used to measure central tendency and variability. Student T-tests were performed to compare the different groups. Paired T-test were used to evaluate the change of parameters within each group.

Partial correlation coefficients were calculated to determine the correlation between two variables dueing the various appointments (initial, pre and, post scaling and root planing). P values ≤ 0.05 were considered significant.

RESULTS

Table (1) shows the mean and standard deviation (SD) of the clinical assessments of early onset periodontitis patients throughout the study. The initial periodontal therapy had an obvious effect on the mean plaque index and the mean EIBI, which significantly decreased throughout the study. On the other hand, analysis of the clinical attachment level (AL) did not reveal any significant changes as a result of therapy.

Table (2) shows the clinical assessments of adult periodontitis patients throughout the study. Again, there is a significant improvement in all clinical paramaters (except AL) throughout the study.

Table (3) shows the mean, SD, and t-test of salivary PAF levels in early onset periodontitis patients as compared to controls throughout the study. There is a statistically significant difference between the study group and their controls.

Table (4) shows the mean, SD, and t-test of salivary PAF levels in adult periodontitis patients as compared to controls. A marked difference between the study group and their control is present before and at the middle of treatment, but no significant difference exists between them after treatment.

Table (5) shows the mean, SD, and t-test of salivary PAF levels in early onset periodontitis patients as compared to adult periodontitis throughout the study. There is no significant difference between the two groups.

Table (6) shows the effect of periodontal treatment on salivary PAF levels in early onset periodontitis patients. A significant decrease of salivary PAF levels is observed in the middle of the treatment relative to before treatment, with t=3.74. A significant difference also exists between the middle of the treatment and post-treatment, with t = 3.67. Finally, a t value of 9.2 shows a significant difference between pre- and post-treatment.

Table (7) shows the effect of periodontal treatment on salivary PAF levels in adult periodontitis patients. Again, there is a significant decrease of salivary PAF throughout initial treatment (t = 2.84 between before and in the middle of treatment, t = 3.2 between middle and post-treatment, and t = 5.36 between pre- and post-treatment).

Table (8) shows the relationship between changes in salivary PAF levels and changes in clinical assessments of early onset periodontitis patients throughout the study, using parital correlation coefficient. Statistical analysis indicates a positive correlation between changes in the salivary PAF levels of this group and their clinical parameters (plaque index and EIBI). On the other hand, there is no correlation between these changes in PAF levels and mean pocket depth and mean attachment level.

Table (9) shows the relationship between changes in salivary PAF levels and changes in clinical assessments of adult periodontitis patients throughout the study, using parital correlation coefficient. Again, positive correlation between changes in the salivary PAF levels of this group and their clinical parameters (plaque index and EIBI), while there is no correlation between these changes in PAF levels and mean pocket depth and mean attachment level.

Table (1): Clinical assessment of early onset periodontitis patients throughout the study.

Parameters	Pre-treatment		Mid-t_t		Post-t_t	
	M	S.D	M	S.D	M	S.D
Pl.I (%)	40.57	37.37	15.4	14.38	12.1	7.87
EIBI (%)	35.2	40.86	4.71	4.07	0.81	1.38
MPD	3.27	0.51	2.31	0.34	2.7	0.5
P.D > 4ml(%)	28.4	9.52	18.53	6.02	8.53	3.42
MAL	3.59	0.52	3.58	0.53	3.58	0.52

Pl.I: Plaque index *EIBI: Eastman interdental bleeding index*
MP.D: Mean probing depth *MAL: Mean attachment level*

Table (2): Clinical assessment of adult periodontitis patients throughout the study.

Parameters	Pre-t_t		Mid-t_t		Post-t_t	
	M	S.D	M	S.D	M	S.D
Pl.I (%)	66.5	21.45	45.9	20.19	16.4	12.02
EIBI (%)	58.89	31.67	23.7	20.09	6.14	12.46
MPD	2.83	0.5	2.42	0.56	1.39	0.43
P.D > 4ml(%)	19.1	12.06	8.27	8.07	4.48	4.17
MAL	3.23	0.54	3.25	0.55	3.24	0.54

Table (3): Salivary PAF level in early onset periodontitis as compared to controls throughout the study.

Appointment	Early onset periodontitis n = 10		Control n = 10		t	P
	Mean	S.D	Mean	S.D		
Pre-t_1	23.18	1.81	15.90	2.06	9.34	S
Mid-t_1	20.64	1.8	15.90	2.06	4.13	S
Post-t_1	17.62	2.05	15.90	2.06	2.76	S

Table (4): Salivary PAF level in adult periodontitis as compared to controls throughout the study.

Appointment	Early onset periodontitis n = 10		Control n = 10		t	P
	Mean	S.D	Mean	S.D		
Pre-t_1	22.47	3.22	13.33	2.48	7.12	S
Mid-t_1	19.38	2.08	13.33	2.48	5.19	S
Post-t_1	14.79	4.45	13.33	2.48	0.91	N.S.

S: Significant
N.S.: Non significant

Table (5): Salivary PAF level from early onset periodontitis as compared to adult periodontitis throughout the study.

Appointment	Early onset periodontitis n = 10		Control n = 10		t	P
	Mean	S.D	Mean	S.D		
Pre-t_1	22.47	3.22	23.18	1.81	0.61	N.S
Mid-t_1	19.38	2.08	20.64	1.8	1.45	N.S
Post-t_1	14.79	4.45	17.62	2.05	1.83	N.S

Table (6): The effect of periodontal treatment on salivary PAF levels in early onset periodontitis patients throughout the study.

Appointment	PAF		t	P
	Mean	S.D.		
Pre-t	23.18	1.81		
			3.74	S
Mid-t	20.64	1.8		
			3.67	S
Post-t	17.62	2.05	9.2	S

Table (7): The effect of periodontal treatment on salivary PAF levels in adult periodontitis patients throughout the study.

Appointment	PAF		t	P
	Mean	S.D.		
Pre-t	22.47	3.22		
			2.84	S
Mid-t	19.38	2.08		
			3.2	S
Post-t	14.79	4.45	5.36	S

Table (8): Relationship of changes in salivary PAF levels to changes in clinical assessments of early onset periodontitis patients throughout the study.

PAF vs clinical indices	r	P
PAF vs PL.I	0.58	S
PAF vs EIBI	0.72	S
PAF vs MPD	0.22	N.S
PAF vs AL	0.02	N.S

Vs: Versus R: Partial correlation coefficient

Table (9): Relationship of changes in salivary PAF levels to changes in clinical assessments of adult periodontitis patients throughout the study.

PAF vs clinical indices	R	P
PAF vs PL.I	0.61	S
PAF vs EIBI	0.73	S
PAF vs MPD	0.11	N.S
PAF vs AL	0.25	N.S

DISCUSSION

PAF participates in normal cell functions as well as pathology. The dual nature of this bioactive phospholipid is similar to that displayed by glutamate, interleukin-1 and nitrous oxide. Synthesis of these molecules occurs in cells in response to normal physiological stimuli, but when their concentrations increase above certain levels they can be harmful.[3] This is in agreement with our findings, which show increased PAF levels in unstimulated mixed saliva from early onset and adult periodontitis patients as compared to their controls. These results coincide with those of Garito et al[8] who reported that PAF levels in saliva progressively increased from the healthiest group to the most severaly affected group suffering from periodontal disease. These results support the hypothesis that this pro-inflammatory phospholipid autacoid may participate in the pathogenesis of periodontal tissue injury and disease.

The current study indicates a significant positive correlation between salivary PAF levels and measures of periodontal inflammation represented by the number of bleeding sites in early onset and adult periodontitis patients. The incremental decrease in salivary PAF levels during initial periodontal therapy occurred in parallel with improvements in clinical status. Similarly Marks et al.[1] observed a relationship between salivary PAF levels and clinical estimates of inflammation in adult periodontitis patients. These findings may reflect the density & types of inflammatory cells within periodontal tissues which could serve as a source of PAF. Previous histological studies have revealed a number of potential cellular sources of PAF in inflammed oral tissues obtained during the course of periodontal therapy. Caton et al,[16,17] concluded that oral hygiene in combination with a single episode of subgingival scaling significantly reduced the density of PMN, lymphocytes, macrophages and monocytes at interproximal sites, which in turn stopped bleeding. Thus PAF may be involved in inflammatory events during the course of both early onset and adult periodontitis. On the other hand, a relationship between changes in PAF levels and those in mean probing

depth and attachment level in both our study groups is insignificant. This may be because these established clinical parameters reflect the cumulative history of periodontal disease but do not necessarily reflect current disease activity.[18] This also explains our finding that no significant difference exists between salivary PAF level in early onset periodontitis patients and those in adult periodontitis patients. In addition to salivary PAF, several host factors (cytokines, enzymes, etc.) have been implicated in the pathogenesis of periodontitis.

As explained above, the results of the study show a significant improvement in the clinical conditions of early onset periodontitis patients correlating to a reduction in salivary PAF levels. Despite this improvement, there is still a marked difference between the patients' salivary PAF levels after scaling root planing and those of their controls. In contrast, there is no significant difference between the salivary PAF levels of adult periodontitis patients after complete scaling and root planing and PAF levels of their controls. This may be explained by the faster rate of disease progression in younger individuals (early onset periodontitis patients). Moreover local mechanical therapy often fails to completely eliminate, for instance, Actinobacillus actinomycetemcomitans from early onset periodontitis cases.[19]

PAF stimulates the production of other diverse inflammatory mediators and conversely, many of these mediators initiate the biosynthesis of PAF.[3,4,6] Moreover, PAF may act either directly or synergistically with other inflammatory mediators to induce phlogistic events including the recruitment and activation of inflammatory cells such as the PMN.[20] Furthermore, PAF enhances IL-I production by monocytes[21] and stimulates osteoclasts,[22,23] effects which could contribute to localized bone resorption in chronic periodontal disease. PAF also inhibits, IL-2 production by T lymphocytes[24] and regulates B lymphocyte functions.[25] Thus PAF produced by activated inflammatory cells within the periodontium may participate in tissue injury through various means, either by initiating inflammation or by combining other host derived mediators in synergistic effects.

Human plasma PAF acetylhydrolase is a novel extracellular phospholipase A_2 that is physiologically and pathologically important because it abolishes the diverse, potent activites of PAF and oxidized phospholipids. Tjoelker et al[26] reported that the administration of supplemental, exogenous. PAF acetylhydrolase suppresses inflammation. Thus, in diseases in which there is a deficiency of enzyme activity, PAF or related compounds might accumulate and supplemental PAF acetylhydrolase may reverse the pathological response. Further studies could therefore investigate whether the approach is clinically viable, particularly when

treatment does not begin until the inflammatory response is underway.

Several specific PAF antagonists have also been developed.[27] The utility of these agents in the treatment of periodontal disease can be envisioned. Addionally, more longitudinal studies are important to further elucidate the role of PAF in periodontal disease and to assess whether salivary PAF may be useful in identifying patients at risk of further periodontal destruction.

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مستويات ب-١-ف اللعاب في مرضى التهابات النسيج الدعامي السني المبكر والبالغ أثناء العلاج المبدئي لهؤلاء المرضى

لقد أجرى هذا البحث لدراسة مستويات ب-١-ف اللعابى لمرضى التهابات النسيج الدعامى السنى المبكر والبالغ وعلاقتها بالقياسات الإكلينيكية للمرضى اثناء العلاج المبدئى لهؤلاء المرضى وقد قسمت مجموعات البحث إلى:

مجموعة الدراسة الأولى:

وتتكون من عشرة مرضى مصابين بالالتهاب المبكر لنسيج الدعامى السنى وفقدان فى العظام الدعامية للأسنان الذى تأكد من فحص الأشعة والذين تتراوح أعمارهم من تسعة عشر إلى تسعة وعشرون عاماً.

مجموعةالقياس الأولى:

وتتكون من عشرة اشخاص مماثلين فى السن والجنس لنظرائهم فى مجموعة الدراسة الأولى ومتمتعين بالصحة اللثوية والداعية للأسنان.

مجموعة الدراسة الثانية:

وهى تشمل عشرة مرضى مصابين بالتهاب فى النسيج الدعامى السنى البالغ أعمارهم ما بين ٣٥-٥٠ سنة شخصوا عن طريق الكشف الإكلينيكى وبالاشعة.

مجموعة القياس الثانية:

وتضم عشرة أشخاص مماثلين فى السن والجنس لنظائرهم فى مجموعة الدراسة الثانية وهم أصحاء ولايوجد عندهم أى امراض فى اللثة أو النسيج الدعامى السنى.

وقد وجد من نتائج هذا البحث زيادة مستويات ب-١-ف اللعابى فى مرضى التهابات النسيج الدعامى السنى المبكر والبالغ مقارنة بمجموعات القياس مما يؤكد دور ب-١-ف أمراض النسيج الدعامى للأسنان.

ايضاً وجد البحث علاقة موجبة واضحة بين مستويات ب-١-ف اللعابى فى هؤلاء المرضى وقياسات الالتهابات فى النسيج الدعامى للأسنان وان النقص المتدرج فى مستويات ب-١-ف اثناء العلاج المبدئى لهؤلاء المرضى المرضى موازية للتحسن الإكلينيكى لحالاتهم.