ORIGINAL RESEARCH

Comparison of gingival crevicular fluid periostin levels in healthy, chronic periodontitis, and aggressive periodontitis

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ABSTRACT

Background: Periostin (POSTN), a matricellular protein released by fibroblasts, is an important regulator of bone development and connective tissue integrity in both healthy and diseased conditions. The goal of the current study was to measure the levels of POSTN in gingival crevicular fluid (GCF) in patients with aggressive and chronic periodontitis and compare them to healthy controls. **Materials and Methods**: A total of 90 people were recruited and divided into three groups: the healthy group, the two forms of periodontitis (chronic and aggressive), each with 30 participants. The GCF fluid samples were taken using a microcapillary pipette. With the aid of the enzyme-linked immunosorbent test, the POSTN levels were calculated. **Results**: The mean levels of total POSTN in GCF fluid (in pg/µl) were 182.41, 79.87, and 49.28 for the healthy, CP, aggressive periodontitis groups, respectively. There was a statistically significant difference between the groups with $P < 0.05$. Furthermore, there were statistically significant differences when compared among the groups with $P < 0.05$. When all three groups were examined together, there were negative correlations between GCF POSTN levels and clinical parameters. **Conclusion**: It was concluded that the levels of POSTN in GCF could be contemplated as a dependable marker in periodontal disease diagnosis and its activity.

Keywords: Aggressive periodontitis, chronic periodontitis, gingival crevicular fluid, healing, periostin

INTRODUCTION

Periodontal disease involves the interaction of the biofilm and host immune-inflammatory response, affects the integrity of the periodontal structures, resulting in destruction of connective tissue and resorption of alveolar bone.[1] Gingivitis is characterized by inflammation that is confined to the gingiva, without loss of periodontal attachment apparatus. The host's immune inflammatory response is markedly different in individuals who develop periodontitis compared to individuals who never progress beyond gingivitis. The components of gingival crevicular fluid (GCF) have commonly been considered to find out the individuals with periodontal disease activity.[2]

Periostin is an 811 amino acid protein, originally identified in murine osteoblasts. Periostin showed structural similarity with fasciclin-1, which is an insect axonal guidance protein and it is also termed as osteoblast specific factor-2.[3]Periostin derived its name, because of its expression in periodontal ligament and periosteum.[4] It is an extracellular matrix protein, has a role in connective tissue integrity and cell migration and adhesion. Another important role of periostin is wound repair, causes interaction of type I collagen with fibronectin, thereby helps in remodelling the periodontium.[5]Periostin is secreted by connective tissues rich in collagen, releases periostin when they are subjected to mechanical stresses.[6]Periostin is also associated with tooth eruption processes promoting adhesion and migration of various cell types, leading to formation of mineralized tissues of the tooth and periodontium.[7]Periostin is entrapped between the cytoplasmic processes of periodontal fibroblasts, cementoblasts and also the surrounding collagen fibrils.[8] Hence periostin may be used as a periodontal regeneration marker and the GCF periostin levels have been found to reduce with the increase of the severity of periodontal disease.[9]

This study was conducted to evaluate the comparison of gingival crevicular fluid periostin levels in healthy, chronic periodontitis, and aggressive periodontitis.

MATERIAL AND METHODS

The study included 90 subjects in all. Following the examination of the individuals clinically and radiographically, they were divided into three groups as follows: the healthy group, CP group, and AgP group (30 in each group). The informed consent was signed by all individuals after describing the need for the study.Group-1 comprised of healthy controls with periodontal probing depth <3 mm, no bleeding on probing and no evidence of bone loss radiographically; GROUP-II-generalized (or) localized mild, moderate, or severe CP patients (According to Armitage criteria); GROUP-III-localized (or) generalized AgP patients (According to Lang's criteria).

The subjects who were pregnant, lactating mothers, current smoker or former smoker, underwent periodontal therapies during the past 6 months, with any drug intake during the past 3 months, with any systemic condition that could compromise POSTN levels such as diabetes mellitus, cancer, and ventricular hypertrophy were not included in the study.

RESULTS

Table 1: age and gender wise distribution of subjects.

Clinical parameters	Group	Number of subjects	Mean	Standard Deviation
Age	Healthy	30	26.45	4.36
	Chronic periodontitis	30	45.57	8.63
	Aggressive periodontitis	30	29.54	9.01
	Total	90	32.78	11.02
		Males	**Females**	**Total**
Gender	Healthy	12	18	30
	Chronic periodontitis	13	17	30
	Aggressive periodontitis	21	09	30
	Total	46	44	90

When the mean age was compared among groups, there was a statistically significant difference among healthy and CP groups ($P < 0.05$), among CP and AgP groups ($P < 0.05$) and there was no statistically significant difference when age was compared among healthy and AgP groups ($P = 0.610$).The clinical parameters that were examined such as mean Plaque Index scores, mSBI percentage, CAL were statistically significant between the groups. When these clinical parameters were compared among groups, there was a statistically significant difference among healthy and CP groups, among healthy and AgP groups and not statistically significant among CP and AgP groups.

The mean levels of total POSTN in GCF were 182.41 pg/µl, 79.87 pg/µl and 49.28 pg/µl for the healthy, CP, AgP groups, respectively. There was a statistically significant difference between the groups. The mean levels of total POSTN in GCF were significantly lower in the CP and AgP groups than in the healthy controls. The amount of POSTN in GCF decreased by 56% in CP group and by 73% in AgP group when compared to the healthy group. There was also statistically significant difference among the groups.

When all clinical groups were observed together, there were negative correlations between POSTN levels in GCF and age, mSBI, plaque index, CAL, that is, the POSTN levels in GCF were inversely related to age, mSBI, plaque index, CAL. It was found that the negative correlation between the POSTN levels in GCF and age ($r = −0.303$; $P = 0.061$) was not statistically significant and the negative correlations between the POSTN levels in GCF and mSBI, plaque index, CAL ($r = −0.788$, $r = −0.655$, $r = −0.691$, respectively; $P < 0.01$) were statistically significant (two-tailed).

Furthermore, in the healthy group, negative correlations were found between the POSTN levels in GCF and age ($r = −0.302$; $P = 0.317$), and the POSTN levels in GCF and Plaque Index ($r = −0.146$; $P = 0.635$), but the correlations were not statistically significant. The correlations between the POSTN levels in GCF and mSBI and the POSTN levels in GCF and CAL were not found.

In CP group, negative correlations were found between the POSTN levels in GCF and mSBI ($r = −0.204$; $P = 0.504$), and the POSTN levels in GCF and Plaque Index ($r = −0.373$; $P = 0.209$), but the correlations were not statistically significant. The correlation between the POSTN levels in GCF and age ($r = 0.333$; $P = 0.267$) and the POSTN levels in GCF and CAL ($r = 0.370$; $P = 0.214$) were positive and not statistically significant.

In AgP group, a negative correlation was found between the POSTN levels in GCF and age ($r = −0.280$; $P = 0.354$) which was not statistically significant. However, the correlations between the POSTN levels in GCF and mSBI ($r = 0.499$; $P = 0.082$), the POSTN levels in GCF and Plaque Index ($r = 0.228$; $P = 0.454$), and the POSTN levels in GCF and CAL ($r = 0.427$; $P = 0.145$) were found to be positive and not statistically significant.

DISCUSSION

Periodontitis is initiated by tooth-associated microorganisms organized as a biofilm, and evokes a host inflammatory response. Reversible inflammatory changes occur in gingivitis, but non-reversible destruction happens in periodontitis and if not treated leads to tooth loss.[10]Periostin being a multifaceted protein, has been involved in repair and regenerative processes of various tissues. It is also a matricellular modulator and it is considered as a biomarker for various diseases.[11] The 90 and 87-kDa isoforms are produced by neuroectodermal-derived fibroblasts.[12] Once it is secreted, periostin has an affinity to bind to molecules such as tenascin-C, collagen and BMP-1.[13] This property helps in the extracellular matrix maturation and increases strength of the tissue.[14]Periostin also possesses potent mitogenic properties and binds with the cell membrane via integrins.[15]Periostin shows greater specificity, among the proteins expressed in periodontal ligament. The purpose of the current study was to investigate GCF and serum periostin levels in individuals with clinically healthy periodontium, gingivitis and chronic periodontitis patients.

Hence, this study was conducted to evaluate the comparison of gingival crevicular fluid periostin levels in healthy, chronic periodontitis, and aggressive periodontitis.

In this study, When the mean age was compared among groups, there was a statistically significant difference among healthy and CP groups ($P < 0.05$), among CP and AgP groups ($P < 0.05$) and there was no statistically significant difference when age was compared among healthy and AgP groups ($P = 0.610$). The clinical parameters that were examined such as mean Plaque Index scores, mSBI percentage, CAL were statistically significant between the groups. When these clinical parameters were compared among groups, there was a statistically significant difference among healthy and CP groups, among healthy and AgP groups and not statistically significant among CP and AgP groups.

The mean levels of total POSTN in GCF were 182.41 pg/µl, 79.87 pg/µl and 49.28 pg/µl for the healthy, CP, AgP groups, respectively. There was a statistically significant difference between the groups. The mean levels of total POSTN in GCF were significantly lower in the CP and AgP groups than in the healthy controls. The amount of POSTN in GCF decreased by 56% in CP group and by 73% in AgP group when compared to the healthy group. There was also statistically significant difference among the groups.

When all clinical groups were observed together, there were negative correlations between POSTN levels in GCF and age, mSBI, plaque index, CAL, that is, the POSTN levels in GCF were inversely related to age, mSBI, plaque index, CAL. It was found that the negative correlation between the POSTN levels in GCF and age ($r = −0.303$; $P = 0.061$) was not statistically significant and the negative correlations between the POSTN levels in GCF and mSBI, plaque index, CAL ($r = −0.788$, $r = −0.655$, $r = −0.691$, respectively; $P < 0.01$) were statistically significant (two-tailed).

Furthermore, in the healthy group, negative correlations were found between the POSTN levels in GCF and age ($r = −0.302$; $P = 0.317$), and the POSTN levels in GCF and Plaque Index ($r = −0.146$; $P = 0.635$), but the correlations were not statistically significant. The correlations between the POSTN levels in GCF and mSBI and the POSTN levels in GCF and CAL were not found.

In CP group, negative correlations were found between the POSTN levels in GCF and mSBI ($r = −0.204$; $P = 0.504$), and the POSTN levels in GCF and Plaque Index ($r = −0.373$; $P = 0.209$), but the correlations were not statistically significant. The correlation between the POSTN levels in GCF and age ($r = 0.333$; $P = 0.267$) and the POSTN levels in GCF and CAL ($r = 0.370$; $P = 0.214$) were positive and not statistically significant.

In AgP group, a negative correlation was found between the POSTN levels in GCF and age ($r = −0.280$; $P = 0.354$) which was not statistically significant. However, the correlations between the POSTN levels in GCF and mSBI ($r = 0.499$; $P = 0.082$), the POSTN levels in GCF and Plaque Index ($r = 0.228$; $P = 0.454$), and the POSTN levels in GCF and CAL ($r = 0.427$; $P = 0.145$) were found to be positive and not statistically significant.

When the mSBI was compared among groups, there were statistically significant differences among healthy and CP groups, among healthy and AgP groups and there was no statistically significant difference among CP and AgP groups. Similar to the present study, no significant difference in full-mouth bleeding on probing score was observed between CP (55.35%) and AgP groups (58.99%) ($P > 0.05$) in Aral et al.'s study.[16]

In Padial-Molina *et al.*'s study, it was found that the GCF POSTN levels (in pg/µl) elevated with time and a correlation was found with the healing process in periodontitis and healthy controls. The analysis revealed that levels of POSTN before the surgery (367.85) were statistically significantly lower than those at the 2nd day (1496.14) ($P < 0.001$) and at the 14th day (836.80) ($P < 0.024$). It is found in the Padial-Molina *et al.*'s study that GCF POSTN levels were lower in diseased condition than in healthy, similar to the present study and increased during healing after treatment.The[17] repair and regeneration of the tissues of periodontium are affected directly by the reduced levels of POSTN.[18,19]

CONCLUSION

It was concluded that the levels of POSTN in GCF could be contemplated as a dependable marker in periodontal disease diagnosis and its activity.

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