The Saudi Dental Journal (2014) **26**, 171–175

King Saud University

The Saudi Dental Journal

www.ksu.edu.sa
www.sciencedirect.com

ORIGINAL ARTICLE

Influence of phase I periodontal therapy on levels of matrix metalloproteinase 1 and tissue inhibitor of metalloproteinase 1

Pallavi S. Ghodpage [a,1], Rajashri A. Kolte [a,*], Abhay P. Kolte [a,2], Madhur Gupta [b,3]

[a] *Department of Periodontics, VSPM Dental College and Research Centre, Digdoh Hills, Hingna Road, Nagpur 440019, India*
[b] *Department of Biochemistry, NKP Salve Institute of Medical Sciences, Digdoh Hills, Hingna Road, Nagpur 440019, India*

Received 25 February 2014; revised 9 April 2014; accepted 6 May 2014
Available online 15 July 2014

KEYWORDS

MMP-1;
TIMP-1;
Chronic periodontitis;
Gingival crevicular fluid

Abstract *Background:* Matrix metalloproteinase-1 (MMP-1) is a member of a family of enzymes that can degrade most extracellular matrix macromolecules. Extracellularly, MMPs are controlled by tissue inhibitors of metalloproteinases (TIMPs) and by mechanisms of pro-MMP activation. Levels of MMPs and TIMPs change during healing, inflammation, and normal tissue turnover. Herein we aimed to evaluate the levels of MMP-1 and TIMP-1 in gingival crevicular fluid (GCF) from periodontally healthy patients (control group) and chronic periodontitis patients before and after phase 1 therapy.

Methods: In this study we examined 30 patients who had chronic periodontitis with probing depth sites $\geqslant 5$ mm and a clinical attachment level (CAL) $\geqslant 5$ mm. We included 30 periodontally healthy patients as a control. Clinical measurements such as plaque (PI) and gingival (GI) indices, papillary bleeding index (PBI), probing depths (PD), and CAL were recorded both before treatment (BT) and after phase I periodontal treatment (AT). Assays for MMP-1 and TIMP-1 were performed with an enzyme-linked immunosorbent assay (ELISA) method.

Results: All clinical parameters were significantly reduced at the post-therapy visit. MMP-1 levels were significantly higher in patients BT than the controls; however, the patients AT were not statistically different than the controls. TIMP-1 levels in patients BT were significantly lower than

* Corresponding author. Tel.: +91 9011071468.
E-mail addresses: pghodpage@gmail.com (P.S. Ghodpage), drrajashrikolte@gmail.com (R.A. Kolte), drabhaypkolte@gmail.com (A.P. Kolte), drmadhur20@rediffmail.com (M. Gupta).
[1] Tel.: +91 9730755718.
[2] Tel.: +91 9011071467.
[3] Tel.: +91 9422782291.

Peer review under responsibility of King Saud University.

Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.sdentj.2014.05.003

in the controls and significantly lower than patients AT. We observed a significant positive correlation between GCF volume and MMP-1 levels. Furthermore, TIMP-1 levels were significantly negatively correlated with both GCF volume and all clinical parameters.

Conclusions: We observed that as the extent of periodontal destruction increases, MMP-1 concentration increases and TIMP-1 concentration decreases in GCF. When chronic periodontitis patients were treated by scaling and root planing (SRP), the average MMP-1 concentrations decreased and TIMP-1 concentrations increased in GCF.

1. Introduction

Periodontitis is an infectious disease characterized by periodontal attachment loss, bone destruction, and eventually tooth loss (Socransky and Haffajee, 1992). The tissue destruction appears to result from a complex interaction between bacteria and the host immune system and a complex network of cytokines, prostaglandins, reactive oxygen species, and proteolytic enzymes. Matrix metalloproteinases (MMPs) are a genetically distinct but structurally related family of host-derived proteolytic enzymes involved in the physiological and pathological degradation of extracellular matrix and basement membrane proteins (Birkedal-Hansen, 1993). To date, 24 different MMPs have been cloned and 23 of those are found in humans.

Fibrillar collagens are resistant to most proteinases and have the potential to initiate collagenolysis during periodontal inflammation. MMP-1, or interstitial collagenase, is one of the key proteolytic enzymes that degrade fibrillar collagens, especially types I and III, which are the predominant types of interstitial collagens in gingiva. MMP-1 is expressed by fibroblasts, endothelial cells, macrophages, hepatocytes, chondrocytes, osteoblasts, tumor cells, and migrating epidermal keratinocytes, but not neutrophils (Birkedal-Hansen, 1993). Tissue inhibitors of metalloproteinases (TIMPs) regulate the proteolytic activity of MMPs. For example, TIMP-1 effectively inhibits the activity of interstitial collagenase, such as MMP-1 (Shibata et al., 1991). TIMPs are expressed by many cells, including fibroblasts, keratinocytes, monocytes, macrophages, endothelial cells, and osteoblasts. There are four identified types of TIMPs, TIMP-1, -2, -3, and -4, which are widely distributed in oral and body fluids. The correct balance of MMPs and TIMPs is critical to degrade the connective tissue matrix under both physiological and pathological conditions. Thus, it is important to study the relationship between MMPs and TIMPs, as they reflect the status of periodontal diseases. The present study aimed to study MMP-1 and TIMP-1 levels in healthy patients and chronic periodontitis patients. Further, we examined the effect of phase 1 therapy on MMP-1 and TIMP-1 levels.

2. Materials and methods

2.1. Study population

A total of 60 subjects between 25 and 55 years of age were selected from patients visiting the Department of Periodontics of Vidya Shikshan Prasarak Mandal Dental College and Research Centre, Nagpur, India. These patients were assessed clinically and biochemically. All protocols were cleared by the Institutional Ethics Committee of our institute. We designed a survey to allow systematic recording of information and observations. The survey included a detailed case history, clinical examination, periodontal indices, and the patient's written consent. The study comprised of two groups: group I, a control group of 30 periodontally healthy subjects, and group II, a test group of 30 patients with chronic periodontitis who were assessed before therapy (BT) and after phase 1 therapy (AT) – It included oral hygiene instructions and scaling and root planing. Patients were considered healthy if they exhibited a probing depth < 3 mm and there was no clinical attachment loss (CAL). The average age of this group was 35.13 ± 7.14. Patients were diagnosed with chronic periodontitis if they exhibited a probing pocket depth $\geqslant 5$ mm and CAL $\geqslant 5$ mm at multiple sites. The average age of this group was 29.6 ± 4.84 years. All subjects were generally in good health and none had received periodontal treatment or medication during the past 6 months. No participants had a history of systemic conditions such as heart disease, diabetes, or other disorders that could influence the course of periodontal disease. Patients were not on any medication that could affect the manifestations of periodontal disease such as antibiotics, or their use in the 6 months prior, phenytoin, cyclosporine, anti-inflammatory drugs, or calcium channel blockers. Postmenopausal women and smokers were excluded from the study.

The clinical evaluation of patients was based on the following indices: plaque index (PI) (Silness & Loe, 1964), gingival index (GI) (Loe & Silness, 1963), probing depths (PD), CAL, and papillary bleeding index (Muhlemann, 1977). All parameters, which were recorded for the complete dentition, were measured using a Williams probe calibrated in mm. The most severely affected upper anterior sextant (maxillary incisors and the canine teeth) was chosen for collection of gingival crevicular fluid (GCF). GCF sampling and clinical index scores were recorded at baseline before treatment and 6 weeks after phase I periodontal therapy. All patients underwent therapy including oral hygiene instruction, scaling, root planning, and gingival curettage under local anesthesia.

2.2. GCF sampling and processing

All of the patients were informed about the procedure and were seated comfortably in the dental chair. Prior to GCF collection, the supragingival soft deposits were removed without causing trauma to the gingival crevice. If hemorrhage was evident after this procedure the GCF was not collected from that site. GCF samples were collected from 13 to 23 regions because the regions were easy to isolate and salivary contamination could be avoided. A microcapillary pipette was used to collect