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ORIGINAL RESEARCH

Non surgical periodontal treatment in patients with gingivitis and moderate periodontitis. Biochemical and microbiological response

Tratamiento periodontal no quirúrgico en pacientes con gingivitis y periodontitis moderada. Respuesta bioquímica y microbiológica

Susana Macín-Cabrera,[*,§] Mariano Sanz-Alonso,[§] Laura Castrillón-Rivera,[||] Alejandro Palma-Ramos,[||] Norma Noguez-Méndez,[¶] Carlos Quirino-Barreda,[¶] Alejandro Rubio-Martínez[**]

ABSTRACT

Objective: To ascertain inflammatory response through interleukin 1β presence and identify pathogenic microorganisms as possible immunological and microbiological markers in diagnosis and treatment non-surgical periodontal in patients with gingivitis and moderate chronic periodontitis in a sample of Mexican population. **Material and methods:** In the present prospective cohort study, 18 patients with signs of gingivitis and 17 patients with moderate chronic periodontitis were selected. Samples of subgingival biofilm and of crevicular gingival fluid were collected. Interleukin 1β was

RESUMEN

Objetivo: *C*onocer la respuesta inflamatoria a través de la presencia de interleucina 1β e identificar microorganismos patógenos como posibles marcadores inmunológicos y microbiológicos en el diagnóstico y tratamiento periodontal no quirúrgico en sujetos con gingivitis y periodontitis crónica moderada en población mexicana. **Material y métodos:** En este estudio prospectivo de cohortes, se seleccionaron 18 pacientes con signos clínicos de gingivitis y 17 pacientes con periodontitis crónica moderada, se recolectaron las muestras de biopelícula subgingival y de fluido gingival crevicular.

Abbreviations:

Units

Base 10 logarithm	= log
Microliters	= μL
Milliliters	= mL
Pictograms	= pg
Percentage	= %
Revolutions per minute	= rpm
Seconds	= sec

Acronyms

APC	= Antigen presenting cells.
BANA	= Nα-Benzol-DL-Arginineβ-Naphtilamide.
BMPs	= Bone morphogenic proteins.
Ep	= Gingival epitelial cells.
CGF	= Crevicular gingival fluid.
IFNγ	= Gamma interferon.
IL	= Interleukin.
IL-1α	= Interleukin 1 alpha.
IL-1β	= Interleukin 1 beta.
IL-1AcP	= Accessory protein of receptor to IL-1.
IL-Rα	= Antagonist receptor.
IL-IRI	= Type I receptor to IL-1.
IL-RII	= Type II receptor to interleukin 1.
iNOS	= Nitric oxide oxidase.
LB	= B lymphocyte.
LT	= T lymphocyte.
LPS	= Bacterial lipopolysaccharide.
MCH	= Main histocompatibility complex.
MMPs	= Matrix metalloproteinases.
NK	= Natural leukocytes of «natural killers».
PAMPs	= Pathogen-associated molecular patterns.
PGE2	= Prostaglandin E_2.
PGI2	= Prostaglandin I_2.
PMN	= Polymorphonuclear leukocytes.
RSP	= Root scaling and planing.
TCR	= T cell receptors.
TH0	= Cooperative T lymphocyte.
TH1	= Cooperative T lymphocyte 1.
TH2	= Cooperative T lymphocyte 2.
TIMPs	= Inhibitor factors of matrix metalloproteinases.
TNFα	= Alpha tumoral necrosis factor.
TNFβ	= Beta tumoral necrosis factor.
CFU	= Colony forming units.
VCAM	= Vascular cell adhesive molecule.

[*] Health Care Department, Metropolitan Autonomous University, Xochimilco Campus, Mexico City.
[§] School of Dentistry, Complutense University, Madrid.
[||] Biological Systems Department, Immunology Laboratory, Metropolitan Autonomous University, Xochimilco Campus, Mexico City.
[¶] Biological Systems Department, Pharmacy Laboratory, Metropolitan Autonomous University, Xochimilco Campus, Mexico City.
[**] Department of Chemistry, Polymers Laboratory, Technological Institute of Queretaro, Mexico.

quantified during the pre-treatment, post-treatment and maintenance phases of the non- surgical periodontal treatment. Continuous variables were analyzed with the Student test, as well as categorical variables which were analyzed with the Turkey-Kramer test. For independent groups the Pearson test was used. **Results:** Microbiological response variables showed that *Porphyromonas gingivalis Prevotella intermedia, Fusobacterium nucleatum*, *Aggregatibacter actinomycetemcomitans* significantly decreased in subjects with gingivitis, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Fusobacterium nucleatum*, *Aggregatibacter actinomycetemcomitans* and *Actinomyces ssp*. decreased in cases. Biochemical response variables showed significant decrease in IL-1β concentration and total count in individuals with moderate chronic periodontitis in treatment maintenance phase. The same result applied to clinical response variables. **Conclusions:** There is a decrease in Interleukine 1β levels with decrease in microflora. Interleukin 1β are sensitive markers for diagnosis of periodontal disease and assessment of its severity.

Se cuantificó la interleucina 1β durante las fases pretratamiento, postratamiento y de mantenimiento del tratamiento periodontal no quirúrgico. **Resultados:** Las variables de respuesta microbiológica mostraron que *Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans* disminuyeron significativamente en individuos con gingivitis. Así como *Porphyromonas gingivalis, Tannerella forsythia, Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans y Actinomyces sp*. en periodontitis crónica moderada. Las variables de respuesta bioquímica mostraron una disminución significativa en la concentración y cuenta total de interleucina 1β en los individuos con periodontitis crónica moderada en la fase de mantenimiento del tratamiento así como de las variables de respuesta clínica. **Conclusión:** Hay reducción de los niveles de interleucina 1β con la disminución de la microflora. Los niveles de interleucina 1β son marcadores sensibles para el diagnóstico y severidad de la enfermedad periodontal.

Key words: Non surgical periodontal treatment, Mexican population, gingivitis, moderate periodontitis.
Palabras clave: Tratamiento periodontal no quirúrgico, población mexicana, gingivitis, periodontitis crónica moderada.

INTRODUCTION

Within the scope of periodontal disease two entities are distinctive, and they possess clearly defined phenotypes: gingivitis (G) and periodontitis (P). These conditions can be clinically observed through chronic inflammatory processes, although in one case (periodontitis) this process evolves and destroys the periodontal attachment apparatus, and in the other case (gingivitis) the inflammatory process is maintained with no evolution towards destruction.[1-6] This inflammatory and immune response is determined by the presence of periodontal pathogens which are Gram negative anaerobic bacteria involved in the subgingival biofilm[1,7-11] such as *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans* (Ac), *Tannerella forsythia* (Tf), *Prevotella intermedia* (Pi), *Fusobacterium nucleatum* (Fn), *Parvimonas micra* (Pm), *Campylobacter rectus* (Cr) and *Actinomyces ssp.*[11-16] as well by biological modifying factors such as: systemic factors, genetic factors[17,18] and behavioral factors such as oral hygiene, smoking habits and stress.[19] In periodontal attachment tissue, inflammatory response comes forth with great amounts of neutrophil polymorphonuclear leukocytes (PMNs) and macrophages with phagocytosis and destructive functions in the sites of bacterial interaction with tissue surface, inciting the presence of inflammatory infiltrate, the activation of the immune system, complement cascade and the cytokine production system.[10,20,21] Most of the substances released by inflammatory and immune cells concentrate in an exudate, characteristic of inflammatory processes observed in G and moderate chronic periodontitis (MCP). This substance (fluid) is called crevicular gingival fluid (CGF). In this fluid it is possible to identify the following pro-inflammatory cytokines: 1-beta interleukin (IL-1β) as well as alpha tumor necrosis factor (TNF-α). Both cytokines are mediators of the inflammatory process because they modulate the extracellular component of bone and connective tissue. In periodontal disease they show high CGF levels, therefore they can have diagnostic interest in G and MCG, since they can be associated to the active phase of these conditions.[22-25]

The aim of the present study was to contribute to the knowledge of microbial flora in this sample of Mexican population and assess links between microbial flora and immunological response in G and its progression to MCP. A prospective cohorts study was conducted to endeavor to find differential aspects with respect to etiological factors and host response. For this purpose quantification of IL-1β in the CGF was used as immunological marker and to examine the effect of NSPT at the pre-treatment, post-treatment and maintenance phases.

MATERIAL AND METHODS

Patient selection

18 patients were selected with clinical and radiographic diagnoses of G (evidence of gingival inflammation, volume increase of the gums, redness and haemorrhage when probing, without loss of epithelial attachment) and 17 patients with MCP (loss of insertion in three or more sites in all quadrants, with pocket depth of 5-7 mm in three or more sites, radiographic evidence of bone loss at a half of the root length in three or more sites of all quadrants, bleeding