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Plasma polyunsaturated fatty acids and periodontal recovery in Taiwanese with periodontitis: A significant relationship

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ABSTRACT

Background: Plasma levels of polyunsaturated fatty acids (PUFAs) are different before and after periodontal treatment. Asians and Westerners have significantly different baseline levels of plasma PUFAs. However, no Asian study has reported the effects of nonsurgical treatment on the correlation between periodontal condition and plasma levels of PUFAs. We analyzed whether recovery from periodontitis was correlated with the elevation of plasma fatty acids 3 months after the nonsurgical intervention and with no recommended supplements.

Design: Thirty-five Taiwanese patients with periodontitis were recruited. Probing pocket depths (PPDs) and clinical attachment levels (CALs) were measured at baseline and 3 months after the nonsurgical treatment. Plasma levels of fatty acids were determined using gas chromatography. Differences and correlations between plasma fatty acid composition and periodontitis severity at baseline and 3 months after treatment were determined.

Results: Twenty-six patients completed the study. At the baseline, PPDs were negatively correlated with plasma n-3 PUFAs ($r = -0.52$, $p < 0.01$), but at 3 months post intervention, periodontitis severity had declined and the weight percentages of n-3 PUFAs, DPA, and DHA were significantly ($p = 0.019$, 0.005, and 0.037, respectively) higher. The recovery percentages of CALs were positively and significantly correlated with plasma ΔPUFAs and the percentage of Δn-3 PUFAs in ΔPUFAs ($r = 0.42$ and 0.45, respectively; $p < 0.05$ for both).

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Abbreviations: AA, arachidonic acid; BHT, butylated hydroxytoluene; CALs, clinical attachment levels; CRP, C-reactive protein; DCM, dichloromethane; DHA, docosahexaenoic acid; DPA, docosapentaenoic acid; EPA, eicosapentaenoic acid; FAMEs, fatty acid methyl esters; IL, interleukin; MUFAs, monounsaturated fatty acids; n-3 PUFAs, n-3 polyunsaturated fatty acids; NSAIDs, nonsteroidal anti-inflammatory drugs; PPDs, probing pocket depths; PUFAs, polyunsaturated fatty acids; SFAs, saturated fatty acids; TNF, tumour necrosis factor.

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Conclusions: We conclude that a higher weight percentage of n-3 PUFAs in total PUFAs was related to the recovery of CALs 3 months after the nonsurgical periodontal treatment. However, no such relationship was found for PPDs.

1. Introduction

Periodontitis, an inflammatory disease of the tissue that surrounds and supports the teeth, is caused by a host immune-inflammatory response to infection by periodontal pathogens.[1] Increased circulating inflammatory mediators such as interleukin (IL)-1β, tumour necrosis factor (TNF)-α, IL-6, and C-reactive protein (CRP) are correlated with the severity of periodontitis.[2–4] Reducing serum levels of proinflammatory cytokines and eicosanoids, such as prostaglandin E_2 (PGE_2) and leukotriene B_4 (LTB_4), and using anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), can improve the outcomes of the traditional periodontal treatment.[5,6]

Fatty acids, both polyunsaturated fatty acids (PUFAs) and saturated fatty acids (SFAs), contribute to the regulation of proinflammatory and anti-inflammatory cascades.[7,8] An experimental study on gingivitis in humans indicated that drug uptake with n-3 PUFAs for 1 week significantly reduced the gingival index.[9] Another experimental study[10] in rats indicated that no effective prevention of alveolar bone resorption was found after treatment with n-3 fatty acids. Furthermore, clinical trials[11,12] showed that lowering the plasma levels of inflammatory metabolites of n-6 PUFAs inhibited the progression of the periodontal disease. The blood levels of PUFAs, especially n-3 PUFAs, have been proposed as the precursors of important metabolites that indicate the inflammatory status in the human body.[7] Elevated levels of plasma n-3 PUFAs were inversely correlated with the plasma levels of inflammatory markers.[13–15] Requirand et al.[16] reported that plasma levels of PUFAs, especially n-3 PUFAs, were significantly lower in patients with periodontitis. A standard first-line therapy for periodontitis is nonsurgical treatment. If this therapy is successful, the patient's periodontal condition will improve. However, the effects of the nonsurgical treatment on the correlation between changes in plasma levels of the compositions of PUFAs and the improvement in the patient's periodontal condition need more studies to verify the relationships.

To the best of our knowledge, there are only four cross-sectional studies[16–19] and one randomized clinical study[20] that report the difference in the plasma levels of long-chain fatty acids between participants with and without periodontitis, periodontitis with and without nonsurgical treatment, and drug uptake with and without n-3 PUFAs in periodontitis. The results of these studies are not comparable, and their conclusions are inconsistent. The differences in the changes of plasma fatty acid compositions, especially n-3 PUFAs, before and after the nonsurgical treatment without prescribed food supplements, remain unclear.

A recent study[21] showed that the baseline data of circulating fatty acid compositions were significantly different between Asians and Westerners. However, no study specifically related to Asians has explored the effects of nonsurgical treatment on the correlation between the severity of periodontitis and the plasma levels of PUFAs. We analyzed whether the recovery from periodontitis is correlated with the elevation of plasma fatty acids 3 months after the nonsurgical intervention and without prescribing recommended supplements.

2. Materials and methods

2.1. Patients and study design

Between March 2005 and March 2007, 35 patients diagnosed with periodontitis and without any other chronic or systemic diseases were recruited. All diagnoses were based on radiography and clinical periodontal examinations by two independent periodontists in the Department of Periodontology, Kaohsiung Medical University Hospital, Taiwan. The reliability of the data supplied by these two periodontists was verified using Cronbach's α (0.92). This study was approved by the Institutional Review Board of Kaohsiung Medical University Hospital (KMUH-IRB-940180), and all study participants signed ''informed consent'' before the data collection.

A standardized structured questionnaire was used to collect demographic data, medical and dental histories, and self-reported smoking habits. Patients with $<$20 remaining teeth, a history of cancer, systemic disorders; those who were currently lactating, pregnant, or planning a pregnancy; those who had undergone subgingival periodontal debridement or periodontal surgery; and those who had taken antibiotics, anti-inflammatory drugs, or dietary fatty acid supplements within the 3 months before the start of the study were excluded.

At baseline and 3 months after the periodontal treatment, the same periodontist gave all participants a full-mouth periodontal examination that assessed bleeding on probing, probing pocket depths (PPDs), and clinical attachment levels (CALs) in order to evaluate the damage caused by periodontitis at six sites per tooth: mesio-buccal, disto-buccal, buccal, mesio-lingual, disto-lingual, and lingual. No dietary recommendations or systemic antibiotics were prescribed during the period of this study.

2.2. Periodontal treatment

After the initial assessment, periodontal treatment was scheduled. The nonsurgical periodontal treatment involved oral hygiene instruction, full-mouth scaling, and subgingival root planing, with both ultrasonic scalers and hand instruments, after a local anaesthetic had been administered. The