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Changes in antimicrobial susceptibility profile and prevalence of quinolone low-sensitive strains in subgingival plaque from acute periodontal lesions after systemic administration of sitafloxacin

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ABSTRACT

This study aimed to assess changes in antimicrobial susceptibilities of subgingival bacteria in acute periodontal lesions following systemic administration of a new-generation fluoroquinolone, sitafloxacin and to monitor the occurrence and fate of quinolone low-sensitive strains. Patients with acute phase of chronic periodontitis were subjected to microbiological assessment of their subgingival plaque samples at baseline (A1). Sitafloxacin was then administered systemically (100 mg/day for 5 days). The microbiological examinations were repeated one week after administration (A2). Susceptibilities of clinical isolates from acute sites to various antimicrobials were determined using broth and agar dilution methods. At A2, subgingival bacteria with low sensitivity to levofloxacin were identified in four patients, and they were subjected to a follow-up microbiological examination at on the average 12 months after sitafloxacin administration (A3). The patients received initial and supportive periodontal therapy during the period A2 to A3. From the examined subgingival sites, 8 and 19 clinical isolates were obtained at A2 and A3, respectively. Some Streptococcus strains isolated at A2 were found to be resistant to levofloxacin (MIC 16–64 μg/ml), azithromycin (MIC 2–>128 μg/ml) or clarithromycin (MIC 1–>32 μg/ml). At A3, isolated streptococci were highly susceptible to levofloxacin (MIC 0.5–2 μg/ml), while those resistant to azithromycin or clarithromycin were still isolated. It is suggested that the presence of the quinolone low-sensitive strains in initially acute lesions after sitafloxacin administration was transient, and they do not persist in the subgingival milieu during the periodontal therapy.

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1. Introduction

In acute phase of periodontitis, an expressed breakdown of periodontal tissue occurs during a limited period of time, with pronounced clinical symptoms [1,2]. For the treatment of acute periodontal lesions, antimicrobial therapy is implemented as an adjunct treatment modality to subgingival debridement and/or abscess drainage. For this purpose, different types of antimicrobial agents are being used, and β-lactam antibiotics are currently the first choice in Japan because of their broad-spectrum antimicrobial effects. β-lactam antibiotics, however, were shown to possess low effect against some of the periodontal pathogens [3]. Furthermore, it was recently reported that β-lactamase-positive subgingival bacteria were detected in more than one-half of subjects with severe chronic periodontitis [4].

In recent years, azithromycin has been used as an adjunctive agent in the management of periodontitis [5]. While the use of azithromycin can be an effective adjunctive treatment modality, a concern has been raised regarding the increased incidence of bacterial resistance to macrolides including azithromycin [5,6]. The use of metronidazole alone or in combination with amoxicillin may be one of the most potent antimicrobial therapies for periodontitis including aggressive periodontitis [7]. However, the use of metronidazole is not indicated for the treatment of periodontitis in Japan.

Sitafloxacin is a new-generation oral fluoroquinolone with

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broad-spectrum antibacterial activity against Gram-positive and Gram-negative aerobes and anaerobes [8,9]. Since 2008, this drug has been used clinically in Japan for a number of conditions including pneumonia, cystitis, pyelonephritis and oral infections [9,10]. It was demonstrated that sitafloxacin possesses *in vitro* high antimicrobial activity against periodontal pathogens [3]. Sitafloxacin has also been shown to effectively suppress the major periodontal pathogens such as *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia* in elderly patients receiving supportive periodontal therapy [11]. In a previous study, we reported that systemic administration of sitafloxacin exhibited potent antimicrobial activity against majority of subgingival bacteria isolated from acute periodontal lesions [12]. Currently information is limited regarding the longitudinal change in susceptibility profile of periodontal bacteria following antimicrobial therapy in patients with periodontitis.

In the present study, we aimed to monitor the occurrence and fate of quinolone low-sensitive strains in acute periodontal lesions following systemic administration of sitafloxacin.

2. Materials and methods

2.1. Participants

This study is a follow-up of a previously reported study in which we assessed the effects of systemic administration of sitafloxacin on subgingival microbial profiles of acute periodontal lesions [12]. Study participants were consecutively recruited from patients who visited the Conservative Dentistry, Tokyo Dental College Chiba Hospital (Chiba, Japan) or Dentistry and Oral Surgery, Keio University Hospital (Tokyo, Japan) during the period of March 2012 through May 2013. Patients were asked to participate if they were diagnosed with acute phase of chronic periodontitis [13]. Criteria for the acute phase included at least two of the following clinical signs or symptoms; pain, swelling, redness or a feeling of warmth in the periodontal lesion [12]. Exclusion criteria included uncontrolled systemic diseases, history of allergic reaction to antimicrobial agents, history of antimicrobial or anti-inflammatory therapy in the previous 6 months, <30 years of age, pregnant or lactating women [12]. In case of pronounced periodontal abscess that might require drainage through external incision or tooth extraction, such patient was excluded.

All participants provided a written informed consent. Ethical approvals for this study were obtained from institutional review boards of Tokyo Dental College (No. 322, 448) and Keio University School of Medicine (No. 2011-239), and the study was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000.

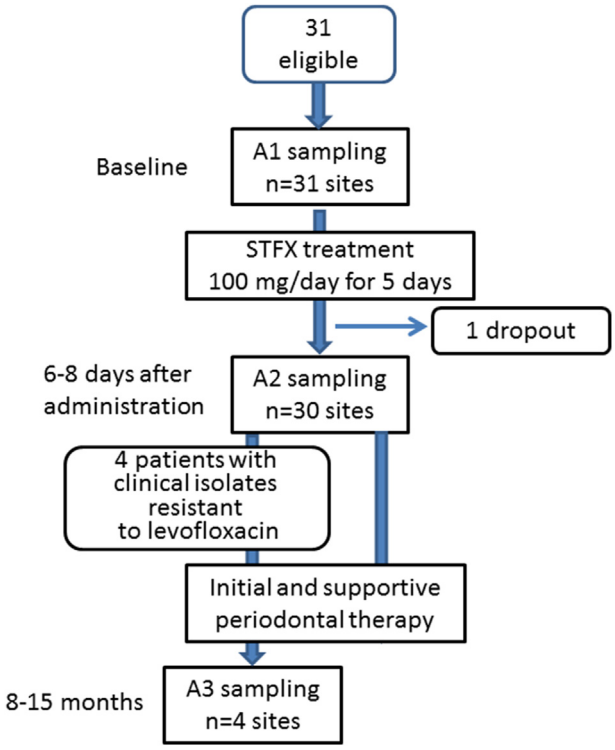

Fig. 1. Study flow chart. A1; baseline, A2; one week after sitafloxacin administration, A3; follow-up examination. STFX; sitafloxacin.

2.2. Clinical examinations and subgingival plaque sampling

The study flow chart is shown in Fig. 1. Periodontal parameters including probing depth (PD), bleeding on probing (BOP) and tooth mobility (TM) were recorded as described previously [12]. Briefly, PD was measured using a Williams probe with an approximate force of 0.25 N and rounding to the nearest millimeter. BOP was recorded as the presence or absence of bleeding after measurement of PD.

For subgingival plaque sampling, one acute site in each patient was chosen as the experimental site, and one chronic site as the control. In the present study, data from acute sites were subjected to analysis. At baseline (A1), subgingival plaque samples were collected by inserting two sterile paper points (Absorbent Paper Points, size 040, Zipperer, Munich, Germany) for 10 s into the deepest area of the pocket accessible after carefully removing the supragingival plaque with sterilized cotton pellets. One paper point was transferred into a sampling tube for the PCR-based analysis. The other paper point was placed into a transport tube (Seed Tube, Eiken Chemical, Tokyo) for the microbiological assessment by culture method.

2.3. Intervention

After sampling, gentle subgingival irrigation with sterile saline solution was performed. No mechanical subgingival debridement was provided at this time. Thereafter, systemic sitafloxacin (Gracevit®, Daiichi Sankyo, Tokyo) was administered (100 mg/day for 5 days). Clinical and microbiological examinations of acute sites were repeated at 6–8 days after the sitafloxacin administration (designated A2). A total of 30 participants complied with dose regimen and completed the A2 examination, as described previously [12]. Then all study participants received non-surgical therapy comprised mainly of oral hygiene instruction and scaling and root planing (SRP) (as initial and subsequent supportive periodontal therapy). For those who harbored subgingival bacteria with low sensitivity to levofloxacin at A2, further follow-up sampling was performed (designated A3). The same sites (initially acute sites at A1) were used for the sampling throughout the study.

2.4. Microbiological assessment and antimicrobial susceptibility testing

Each patient contributed one acute site for microbiological sampling, and all samples collected (n = 30) were processed for the following assessment. For the detection of periodontal pathogens (*Aggregatibacter actinomycetemcomitans*, *P. gingivalis*, *T. forsythia* and *T. denticola*) by PCR-Invader method [14,15], the subgingival plaque samples were sent to a commercial laboratory (BML, Tokyo)

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