Orthodontic treatment simultaneous to or after periodontal cause-related treatment in periodontitis susceptible patients. Part I: Clinical outcome. A randomized clinical trial

Eglė Zasčiurinskienė1,2 | Nomeda Basevičienė3 | Rune Lindsten2,4 | Christer Slotte5,6,* | Henrik Jansson2,5 | Krister Bjerklin4

1Department of Orthodontics, Medical Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania
2Centre For Oral Health, School of Health and Welfare, Jönköping University, Jönköping, Sweden
3Department of Dental and Oral Pathology, Medical Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania
4Department of Orthodontics, Institute for Postgraduate Dental Education, Jönköping, Sweden
5Department of Periodontology, Institute for Postgraduate Dental Education, Jönköping, Sweden
6Department of Biomaterials, Institute for Clinical Sciences, Sahlgrenska Academy, University of Gothenburg, Göteborg, Sweden

Correspondence
Eglė Zasčiurinskienė, Department of Orthodontics, Lithuanian University of Health Sciences, Kaunas, Lithuania.
Email: eglezas@gmail.com

Abstract

Aim: To compare two treatment strategies regarding the effect of orthodontic treatment on periodontal status in patients with plaque-induced periodontitis.

Subjects and Methods: This was a randomized clinical trial. Fifty periodontal patients were randomly assigned to the test or control groups according to periodontal treatment timing. All patients received supra- and subgingival debridement following baseline examination. Control group patients received cause-related periodontal treatment before the start of orthodontic treatment and which was performed simultaneous to orthodontic treatment for the test group patients.

Results: No difference between the test and control groups was found regarding change of clinical attachment level (CAL) after periodontal-orthodontic treatment. Fewer sites with initial pocket depth (PD) of 4–6 mm healed after periodontal–orthodontic treatment in the test group (20.5%, IQR = 11.9%) in comparison with controls (30.4%, IQR = 27.1%) (p = .03). Anterior teeth [OR 2.5] and teeth in male patients [OR 1.6] had a greater chance for PD improvement ≥2 mm. Total periodontal–orthodontic treatment duration was significantly longer for the control group (p < .01).

Conclusions: Both groups showed a gain of CAL and a reduction in sites with PD ≥ 4 mm. Orthodontic treatment, simultaneously to the periodontal treatment, could be used in the routine treatment of patients with plaque-induced periodontitis.

KEYWORDS
clinical attachment level, gingival recession, orthodontic treatment, periodontal treatment, periodontitis, pocket depth

1 INTRODUCTION

Periodontitis is described as a result of an imbalance of the oral microbiota, in the dento-gingival area, and the host response leading to inflammation and destruction of the periodontium in susceptible individuals (Berglundh & Donati, 2005; Flemmig, 1999). To reduce microorganisms, both non-surgical and surgical therapies have been successfully performed (Badersten, Nilveus, & Egelberg, 1981, 1984; Becker et al., 2001; Deas, Moritz, Sagun, Gruwell, & Powell, 2016; Isidor & Karring, 1986; Kaldahl, Kalkwarf, Patil, Dyer, & Bates, 1988; Ramfjord et al., 1987; Sanz, Alonso, Carasol, Herrera, & Sanz, 2012).

As a consequence of plaque-induced periodontitis, patients with increased numbers of teeth showing attachment loss also have...
pathological migration of anterior teeth, decreased posterior bite height and worsened smile aesthetics (Brunsvold, 2005; Johal & Ide, 1999). The latter is the most common reason for patients seeking treatment.

Some clinical studies have shown that orthodontic treatment may be effective in intruding and replacing the migrated maxillary incisors (Artun & Urbye, 1988; Cardaropoli, Re, Corrente, & Abundo, 2001, 2004; Corrente, Abundo, Re, Cardaropoli, & Cardaropoli, 2003; Melsen, 2001). The use of orthodontic tooth movement after conventional periodontal therapy in periodontally involved patients has been described in a number of case reports in the past years (Agarwal et al., 2014; Fukunaga, Kuroda, Kurosaka, & Takano-Yamamoto, 2006; Janson, Janson, & Murillo-Goizueta, 2011; Nakamura, Gomi, Oikawa, Tokiwa, & Sekiya, 2013; Pinho, Neves, & Alves, 2012). However, the effect of orthodontic treatment on periodontal tissues, the risks and benefits of orthodontic tooth movement in the patients with periodontal pathology is controversial and lacking sufficient scientific evidence (Zasciurinskiene, Lindsten, Slotte, & Bjerklin, 2016). Therefore, it is not clear if it could be used as a routine treatment protocol in periodontally involved dentitions.

Tooth movement in reduced, but healthy periodontium may improve attachment level, and conversely, in active periodontal cases, orthodontic treatment may cause further progression of periodontal destruction (Ericsson, Thilander, Lindhe, & Okamoto, 1977; Melsen, 2001; Melsen, Agerbaek, Eriksen, & Terp, 1988 Wennstrom, Lindhe, Sinclair, & Thilander, 1987). These studies led to the knowledge that periodontal treatment should be performed first.

The aim of the present randomized clinical trial was to compare two treatment strategies regarding the effect of orthodontic tooth movement on periodontal status in patients with plaque-induced periodontitis. The secondary aim was to evaluate differences in treatment duration.

The null hypothesis tested was that no statistically significant difference could be demonstrated between the test and control groups assessing clinical attachment level (CAL) change after combined periodontal-orthodontic treatment.

**TABLE 1** Inclusion and exclusion criteria

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>• &gt;25 years of age;</td>
<td>• any systemic disease (e.g. diabetes) or medication that would influence treatment;</td>
</tr>
<tr>
<td>• good general health status;</td>
<td>• periodontal treatment in the previous 2 years;</td>
</tr>
<tr>
<td>• a minimum of six anterior teeth present;</td>
<td>• pregnant or lactating women;</td>
</tr>
<tr>
<td>• periodontitis experience and marginal bone loss at ≥3 teeth and more than 1/3 of the root length (assessed by panoramic radiographs);</td>
<td>• smokers (&gt;5 cigarettes/day);</td>
</tr>
<tr>
<td>• ≥3 teeth with bleeding on probing (BoP), pocket depth (PD) ≥4 mm and loss of clinical attachment level (CAL) ≥4 mm (assessed by clinical examination);</td>
<td>• failure to comply to oral hygiene instructions provided or/and to keep to regular study appointments.</td>
</tr>
<tr>
<td>• malocclusion that needs orthodontic treatment.</td>
<td></td>
</tr>
</tbody>
</table>
2.2 | Trial design

This was a randomized, controlled, parallel, open-label clinical trial. The CONSORT (consolidated standards of reporting trials) guidelines for clinical trials were followed (Moher et al., 2012; Pandis, 2013).

2.2.1 | Randomization and allocation concealment

After consultation by orthodontist, selected patients were offered the treatment with fixed orthodontic appliances. The patients who consented to undergo orthodontic treatment were randomly assigned into either the intervention test group (periodontal treatment simultaneous to orthodontic treatment) or the control group (periodontal treatment before orthodontic treatment) (Figure 1). Randomization was performed immediately after baseline examination according to a computer-generated randomization list. All patients were numbered consecutively. Clinicians and patients were not blinded due to the design of this research. Patients who dropped out of the study (n = 2) were replaced by additional patients, but no later than January 2015 (Figure 1).

All trial patients received oral hygiene instructions (OHI) and professional oral hygiene (POH) (Figure 2).

Test group: Twenty-five patients started orthodontic treatment with conventional straight-wire mechanics directly after POH. Non-surgical therapy and subsequent periodontal surgery in sites showing probing depth (PD) ≥6 mm that bled on probing were performed after orthodontic alignment and leveling phases had finished.

Control group: Twenty-five patients received cause-related non-surgical therapy with subsequent periodontal surgery in sites showing PD ≥6 mm that bled on probing, before orthodontic treatment. Orthodontic treatment was applied as in the test group (Figure 2).

Changes in periodontal status were compared between the groups.

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**FIGURE 1** Flow chart of trial patients
2.3 | Periodontal treatment (PT)

2.3.1 | Professional oral hygiene

POH treatment including the removal of supra- and subgingival calculus by ultrasonic instrumentation supplemented with hand instruments was performed for every study patient following baseline examination. The result was assessed after 2 weeks. In cases with BoP > 30%, POH was repeated.

2.3.2 | Cause-related periodontal treatment

Non-surgical therapy was performed under local anaesthesia at different time points (Figure 2). Treatment was scheduled in four weekly

POH – professional oral hygiene; BoP – bleeding on probing; PD – pocket depth.

FIGURE 2 Flow chart of trial procedures
visits. Patients had to rinse with a 0.12% chlorhexidine solution twice daily during this phase.

### 2.3.3 | Periodontal surgery

Modified Widman flap surgery was scheduled and performed in sites showing PD ≥ 6 mm that bled on probing, following SRP at the 3-month follow-up visit. All patients were prescribed mouth rinsing with a 0.12% chlorhexidine solution twice daily during the surgical phase and for two weeks following surgery (American Academy of Periodontology 2011). No antibiotics were prescribed for the additional effect of periodontal treatment. However, antibiotics in Lithuania are easily accessible and often used to treat other diseases as in many parts of the world.

The application of active orthodontic forces for the test group was postponed 2–4 weeks after periodontal surgery.

### 2.3.4 | Supportive periodontal treatment

OHI (including interproximal brushing) was performed continuously throughout all treatments for both groups (Figure 2). POH was continuously maintained at 3–6 month intervals by a dental hygienist during OT.

### 2.4 | Orthodontic treatment (OT)

Orthodontic treatment with straight-wire appliance was performed in the Orthodontic Department of LUHS and two private clinics by one of the authors (EZ).

Good oral hygiene with a full-mouth plaque score <25% was assured before the start of OT (Lang & Tonetti, 2003).

After baseline examination and POH, the decision was made about the teeth that needed to be moved in each patient. This depended on the number of teeth present, the severity of malocclusion of each patient, planned/existing neighbouring implants and/or prosthetic restorations.

Self-ligating brackets with MBT (McLaughlin, Bennett, Trevisi), prescription (3M Unitek Orthodontic Products; Monrovia, CA, USA) and 0.022-inch slot were used. Aesthetic brackets (Clarity™ SL) were used for maxillary anterior segment to fulfill the aesthetic requirements of the trial patients according to individual needs. Initial levelling and alignment were performed using round, nitinol heat-activated archwires (3M Unitek Orthodontic Products; Monrovia, CA, USA). The inter-dental enamel reduction technique was used in crowded cases where needed. Intrusion and retraction of maxillary and/or mandibular anterior teeth were provided in flared incisor cases. Space closure was performed using rectangular or round stainless steel wires. Anchorage, where needed, was ensured with microscrews or temporary crowns on implants.

Following active OT, the fixed appliances were removed and a fixed (multistrand wire) orthodontic retainer for maxillary and mandibular anterior teeth was applied for all patients and Hawley or vacuum-pressed retainers were provided for the night time for unlimited period of time.

### 2.5 | Clinical examination

Standardized clinical measurements at all time points for every trial patient were performed by the same investigator (EZ). Measurements were recorded on forms designed for the trial. The measurements were performed at all sites around each tooth with a manual periodontal probe (Hu-Friedy PCP-UNC 15, Chicago, IL, USA). Only the deepest measurement was recorded for each of the four sites: mesial (M), buccal (B), distal (D) and lingual (L). To decrease measurement bias of the clinical parameters, every clinical examination was recorded on a separate record form at every time point. Periodontal assessments before and after OT were performed only for teeth that were orthodontically moved.

**T0: Baseline examination.** All participants were given a baseline examination including the following measurements:

- Visible plaque index (VPI): counted by the presence of visible dental plaque along the gingival margin at four sites (M-B-D-L) of each tooth and expressed as a percentage of examined sites within each subject (Ainamo & Bay, 1975).
- Bleeding on probing (BoP): measured after probing to the base of the pocket.
- Pocket depth (PD): measured from the gingival margin to the bottom of the pocket at four sites (M-B-D-L) and recorded to the nearest millimetre.
- Gingival recession (REC): measured as a distance from the cemento-enamel junction (CEJ) to the gingival margin at four sites (M-B-D-L) and recorded to the nearest millimetre.
- Clinical attachment level (CAL): measured as a distance from the bottom of the pocket to cement–enamel junction and recorded in millimetres at four sites (M-B-D-L).

**T1: Pre-orthodontic examination.** All patients were examined before OT start with all and the same measurements: VPI, BoP, PD, REC and CAL.

**T2: Post-orthodontic examination (study end point).** All patients were examined the day of bracket debond: VPI, BoP, PD, REC and CAL.

Changes in periodontal status were compared between time points T0, T1, T2 in both groups.

### 2.5.1 | Examination during OT

Every 3 to 6 months: BoP, PD.

### 2.6 | Radiographic examination

The enrolled study patients were examined with panoramic radiographs before PT for diagnostic purpose—evaluation of alveolar bone status.
2.7 | Outcomes

Gain in CAL was chosen as the primary response variable to judge the effectiveness of the treatment (Page & DeRouen, 1992; Savage, Eaton, Moles, & Needleman, 2009). Secondary outcomes included (i) PD reduction; (ii) REC development, (iii) treatment duration.

2.7.1 | Data management

Data analyses were based on the patient as the unit of measure. CAL gain was evaluated by the millimetre change. Measurement analysis included sites that had CAL ≥ 4 mm and expressed as median value of the assessed registrations within each subject in test and control groups at time points corresponding to T0 and T2.

CAL and PD changes in patient level were also evaluated in the subgroup analysis. Sites were classified as follows: <4 mm, 4–6 mm and ≥6 mm. Sites that had changed CAL and PD class during treatment phases T0–T1, T1–T2 and T0–T2 were analysed.

Analysis was performed also at tooth level for PD change from T0 to T2. Only sites PD ≥ 4 mm were included. PD change was measured for every tooth number according to FDI World Dental Federation system. PD change T0–T2 was grouped (<2 and ≥2 mm) according to median value of 2 mm. Teeth were also grouped as anterior (central incisors, lateral incisors, canines) and posterior (premolars, molars). Two age groups (≤35 and >35 years) were also created (Demmer & Papapanou, 2010). PD change of ≥ 2 mm as dependent variable was used in multivariate binary logistic regression analysis model.

2.8 | Statistical analysis

2.8.1 | Sample size

The CAL difference of 1.0 mm was considered to be of clinical importance between the test and control groups. To be able to detect a clinically meaningful difference in mean CAL of 1.0 mm between groups, standard deviation of 1.0 mm, with a power of 90% and an alpha-level at 0.05, 22 patients were needed in each group. To compensate for dropouts, twenty-five patients were recruited in each group.

2.8.2 | Statistical method

Statistical data analysis was performed using SPSS 20.0 program package (SPSS Inc, Chicago, IL).

Every data set was tested for normality by Shapiro–Wilk test. Statistical analyses to compare two independent variables were conducted using Student’s t test or Mann–Whitney U test.

Wilcoxon signed-ranks test was used for dependent variables.

Chi-square test was used to compare the proportions of categorical variables between the groups.

To determine correlation between variables, Spearman’s coefficient was used.

Logistic regression analysis was performed to determine odds ratio (OR) predictive values.

For the assessment of reproducibility of measurements made by two investigators, the interclass correlation coefficient (ICC) and “consistency type” were used. Intra-class reproducibility (EZ) was calculated with “absolute agreement” type.

In all analyses, a p value < .05 was considered significant.

2.8.3 | Calibration

Calibration of the investigator (EZ) was performed twice (with 1-year interval) during the experimental period by recording measurements of same half mouth of each of three patients by periodontologist (NB) and orthodontist (EZ). Examiners were blinded to each other. CAL at four sites of 30 teeth was examined. A total of 120 repeated measurements were evaluated.

The repeated measurements were made twice at the same visit with 30-min intervals for intra-class reproducibility for the author EZ.

The analysis showed high interclass agreement (0.95; 95% CI: 0.92, 0.96; p = .0001) and intra-class agreement (0.93; 95% CI: 0.87, 0.95; p = .0001).

3 | RESULTS

3.1 | Participant flow

The present trial was conducted between 2010 and 2016.

The flow chart of trial patients and the reasons for exclusion are presented in Figure 1.

In total 50 selected patients, 25 in the test and 25 in the control group started periodontal–orthodontic treatment. After two withdrawn and two replaced patients, 50 patients completed the trial and were finally evaluated. Reasons for dropout are shown in the flow diagram (Figure 1).

3.2 | Baseline data

The baseline demographic and clinical data for both groups are shown in Table 2. There were no significant differences between the test and control treatment groups. One patient in each group smoked ≤ 5 cigarettes per day. Both of them stopped smoking during the treatment.

Majority of included patients showed reduced posterior height of the occlusion with proclined anterior teeth (80%). Most of them had spacing in the upper and/or lower teeth and/or over-eruption of incisors. Nineteen (38%) patients had flared maxillary incisors. Dental mal- occlusions according to angle classification are described in Table 2.

3.3 | Adverse events

One maxillary central incisor developed severe apical root resorption more than 1/3 of the root. Due to reduced bone level and extensive apical root resorption, this tooth was deemed for extraction after OT.

One maxillary central incisor developed severe external root resorption with extension to the pulp. This tooth was vital, resulted in CAL gain with PD < 4 mm and was left for follow-up.
3.3.1 | CAL change after periodontal–orthodontic treatment (T0–T2)

No difference in CAL change was found between test and control patients. Twenty-two (88%) patients in every group resulted in CAL gain. The values are presented in Table 3.

3.3.2 | T0–T1: Pre-orthodontic treatment phase

No difference in the median % of CAL change was found between test and control groups. The overview of calculated values for comparison is seen in Table 4.

There was a significant difference in mean duration of the initial (pre-orthodontic) treatment phase between the test and control groups (Table 5).

3.3.3 | T1–T2: Orthodontic treatment phase

Mean 20.6 (95% CI: 18.19; 22.93, range 7–28) teeth in the test group and 20.84 (18.47; 23.21, range 9–28) were incorporated in orthodontic appliances (p = .8). Mean OT time did not differ between the groups (Table 5).

No difference in CAL change was observed between the test and control groups after OT. The median % changes of sites for CAL, PD and REC are seen in Table 4.

3.3.4 | T0–T2: Whole periodontal–orthodontic treatment

Total periodontal–orthodontic treatment duration was significantly longer for the control group with mean time difference found 4.05 ± 1.59 months (Table 5).

A significant difference between the groups was found in median % of sites that changed to the lower disease group of PD (Table 4). This difference was observed in sites with baseline PD of 4–6 mm, which after combined treatment became <4 mm (Table 5).

Tooth level analysis of PD change T0–T2 showed no significant difference between different teeth and was mean 2.72 (SD 1.25) mm. Univariate binary logistic regression analysis revealed statistically significant association between PD improvement ≥2 mm and tooth group (anterior/posterior) [OR 2.6; 95% CI: 2.03, 3.211, p = .001], sex [OR 1.7; 95% CI: 1.29–2.12, p = .001], age group (≤35/>35 years) [OR 1.4; 95% CI: 1.03–1.78, p = .03]. Analysis showed multicol-linearity between sex and age group (r = .327, p < .001). Multivariate
binary logistic regression analysis (overall percentage of the model 72.8%) revealed higher odds ratio for PPD improvement by ≥2 mm for anterior teeth [OR 2.5; 95% CI: 1.97–3.11, \( p = .0001 \)] and teeth in male patients [OR 1.6; 95% CI: 1.18–1.97, \( p = .001 \)].

As no difference in CAL change (Tables 3 and 4) after combined periodontal–orthodontic treatment was found between test and control patients, the null hypothesis was accepted.

### Table 3: Comparison of clinical attachment level (CAL) change between test and control group patients before (T0) and after (T2) periodontal–orthodontic treatment

<table>
<thead>
<tr>
<th>Variable</th>
<th>Test patients n = 25</th>
<th>Control patients n = 25</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAL (mm) (sites ≥4 mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td>5.28 (5.07, 5.49)</td>
<td>5.24 (5.02, 5.47)</td>
<td>4.81 (4.59, 5.04)</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>5.18 (4.85; 5.64)</td>
<td>5.15 (4.84; 5.58)</td>
<td>4.70 (4.37; 5.05)</td>
</tr>
<tr>
<td>Min–Max</td>
<td>4.61–6.39</td>
<td>4.51–6.75</td>
<td>4.17–7.17</td>
</tr>
</tbody>
</table>

| CAL CHANGE (mm) (sites ≥4 mm) | | | |
| Mean (95% CI) | 0.44 (0.25, 0.62) | 0.38 (0.18, 0.58) | .59* |
| Median (IQR) | 0.40 (0.20, 0.59) | 0.49 (0.12, 0.62) | .59* |
| Min–Max | −0.47 to 1.84 | −1.34 to 1.02 | .70* |

| CAL GAIN (mm) (sites ≥4 mm) | | | |
| Mean (95% CI) | 0.51 (0.38; 0.64) | 0.52 (0.35; 0.70) | .50* |
| Median (IQR) | 0.50 (0.32; 0.66) | 0.42 (0.27; 0.67) | .50* |
| Min–Max | 0.08–1.2 | 0.08–1.84 | .70* |

CAL, clinical attachment level; IQR, interquartile range.
*Mann–Whitney U test.
**Wilcoxon signed-ranks test.

The change of sites with PD ≥4 mm was observed in both groups in every treatment phase (Table 4). At the end (T0–T2), there were more sites that changed PD class (healed) in the control than in the test group (Tables 4 and 6). Classified PD analysis showed that this difference was mainly in sites with initial PD of 4–6 mm (Table 6). This could probably be explained by more sites with PD 4–6 mm at T0 in the control group. The method we used to classify pockets could also influence the result that PD of 4–6 mm remained in the same class. The pockets that initially were 6 mm could heal to 4 mm, but we could not see that as they remained in the limits of the same class. However, deep periodontal pockets (>6 mm) at T0 showed similar improvement in both groups (Table 6). Nearly half of them healed to the “healthy” status (<4 mm). In previous studies that were performed on periodontal patients, PD reduction was also observed (Cardaropoli et al., 2001; Corrente et al., 2003; Han, 2015; Re, Corrente, Abundo, & Cardaropoli, 2000).

The multivariate binary logistic regression analysis showed greater chance for PD improvement by ≥2 mm in anterior teeth and teeth in male patients. As mentioned before, 80% of patients had baseline proclination of anterior teeth. So, anterior teeth were intruded and retruded and this could influence difference in soft tissue response (Figure 3).

Gingival recession is a common consequence of periodontal and orthodontic treatment (Becker et al., 2001; Lindhe, Socransky, Nyman, & Westfelt, 1987; Renkema, Fudalej, Renkema, Kiekens, & Katsaros, 2013). In the present trial, more sites resulted in REC in the control group at T0–T1, and part of them improved during OT (Table 4). This could happen due to orthodontic movements, as shown in earlier

### 4 | DISCUSSION

The present trial shows the overall favourable results of periodontal–orthodontic treatment of patients with plaque-induced periodontitis.

To the best of our knowledge, the present study is the first RCT, which is designed to analyse two different timings of PT in combination with OT in patients with plaque-induced periodontitis. Previously published clinical studies (Cardaropoli et al., 2001; Corrente et al., 2003; Melsen, 2001; Melsen, Agerbaek, & Markenstam, 1989; Re, Cardaropoli, Abundo, & Corrente, 2004) evaluated OT effect on attachment apparatus, when surgical PT was completed 1–2 weeks before OT.

#### 4.1 | Main findings

The main clinical finding of the present trial was CAL gain observed in both groups, in spite of the timing of the PT (Tables 3 and 4). CAL gain was also reported in a study by Corrente et al. (2003), where intrusion was used to replace migrated maxillary incisors.
studies (Cardaropoli et al., 2001; Melsen, 2001; Melsen et al., 1989; Re et al., 2004). For the test group, fewer sites developed REC during the initial PT. But the changes went in the opposite direction during OT with an increase in sites with recession development. This could be related to the PT being performed simultaneous to OT for this group. After T0–T2, there was no significant difference in median % of sites with REC development between the groups (Table 4).

Mean OT duration was similar for both groups and is in agreement with the findings of a recent review article, where the average comprehensive OT time was 20 months (Tsichlaki, Chin, Pandis, & Fleming, 2016). Based on the results of the present trial, total treatment time was significantly longer for the control group patients (Table 5). This difference for the control group was due to the longer PT phase before OT. If the treatment duration is important for the patient, based on the findings of this trial, final PT may be postponed and performed during OT.

The cooperation between one orthodontist (EZ) and ten periodontists was needed to perform the present trial. This can be a limitation but also a strength because it was possible to carry out the trial in a reasonable time (6 years), and we were not dependent on a single periodontologist.

Due to the absence of studies with comprehensive OT in patients with plaque-induced periodontitis, especially RCTs, we were not able to compare our findings.

### 5 | CONCLUSIONS

Both groups showed gain of clinical attachment level (CAL) and reduction in sites with probing depth (PD) ≥4 mm. No difference in REC development between the two groups was observed. Total

### TABLE 4

<table>
<thead>
<tr>
<th>Group</th>
<th>Initial periodontal treatment T0–T1</th>
<th>Orthodontic treatment T1–T2</th>
<th>Total treatment T0–T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAL gain</td>
<td>Median (Q1–Q3) % sites</td>
<td>Median (Q1–Q3) % sites</td>
<td>Median (Q1–Q3) % sites</td>
</tr>
<tr>
<td>Control</td>
<td>6.3 (2.5–14.8)</td>
<td>14.1 (9.7–21.7)</td>
<td>25.0 (18.1–30.7)</td>
</tr>
<tr>
<td>Test</td>
<td>4.0 (0–10.6)</td>
<td>17.0 (14.2–21.2)</td>
<td>23.2 (15.3–28.0)</td>
</tr>
<tr>
<td>CAL no change</td>
<td>Control</td>
<td>93.4 (85.2–96.8)</td>
<td>83.3 (74.9–89.3)</td>
</tr>
<tr>
<td>Test</td>
<td>96.1 (89.4–100)</td>
<td>82.1 (78.9–84.3)</td>
<td>76.9 (71.1–82.9)</td>
</tr>
<tr>
<td>CAL loss</td>
<td>Control</td>
<td>0</td>
<td>1.0 (0–2.8)</td>
</tr>
<tr>
<td>Test</td>
<td>0</td>
<td>0 (0–1.7)</td>
<td>0 (0–1.3)</td>
</tr>
<tr>
<td>PD reduced</td>
<td>Control</td>
<td>31.0 (14.8–45.6)</td>
<td>7.5 (4.3–14.6)</td>
</tr>
<tr>
<td>Test</td>
<td>8.3 (1.6–23.2)</td>
<td>13.4 (9.8–18.7)</td>
<td>25.0 (16.7–36.5)</td>
</tr>
<tr>
<td>PD no change</td>
<td>Control</td>
<td>69.0 (54.4–85.3)</td>
<td>86.6 (83.5–94.1)</td>
</tr>
<tr>
<td>Test</td>
<td>91.7 (76.8–98.5)</td>
<td>85.9 (81.4–89.2)</td>
<td>75.0 (63.5–83.3)</td>
</tr>
<tr>
<td>PD increased</td>
<td>Control</td>
<td>0</td>
<td>1.4 (0–2.8)</td>
</tr>
<tr>
<td>Test</td>
<td>0</td>
<td>0 (0–0.6)</td>
<td>0</td>
</tr>
<tr>
<td>REC development</td>
<td>Control</td>
<td>-27.7 (-40.1 to -5.1)</td>
<td>4.2 (-2.5 to 10.8)</td>
</tr>
<tr>
<td>Test</td>
<td>-4.2 (-13.8 to 0)</td>
<td>-3.6 (-7.4 to 4.4)</td>
<td>-10.9 (-20.5 to -1.3)</td>
</tr>
<tr>
<td>PLAQUE reduction</td>
<td>Control</td>
<td>54.2 (41.0–65.5)</td>
<td>2.6 (-1.2 to 9.4)</td>
</tr>
<tr>
<td>Test</td>
<td>51.4 (41.6–62.2)</td>
<td>4.2 (0–10.1)</td>
<td>57.1 (45.3–68.7)</td>
</tr>
<tr>
<td>BoP reduction</td>
<td>Control</td>
<td>73.7 (62.1–82.2)</td>
<td>4.8 (-1.9 to 12.1)</td>
</tr>
<tr>
<td>Test</td>
<td>60.9 (46.1–76.8)</td>
<td>11.1 (6.4–16.7)</td>
<td>81.0 (66.7–86.0)</td>
</tr>
</tbody>
</table>

CAL, clinical attachment level; PD, pocket depth; REC, gingival recession; BoP, bleeding on probing; Q1, the first quartile; Q3, the third quartile; control group n = 25, test group n = 25; p value by Mann–Whitney U test.

### TABLE 5

<table>
<thead>
<tr>
<th>Group</th>
<th>Initial periodontal treatment T0–T1 (months)</th>
<th>Orthodontic treatment T1–T2 (months)</th>
<th>Total treatment T0–T2 (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SE</td>
<td>95% CI</td>
<td>p value</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Test</td>
<td>1.38 ± 0.18</td>
<td>1.02 1.74</td>
<td>.0001</td>
</tr>
<tr>
<td>Control</td>
<td>4.56 ± 0.35</td>
<td>3.84 5.28</td>
<td>.08</td>
</tr>
</tbody>
</table>

Cl, confidence interval; p value by Student t test.
TABLE 6  Distribution of different pocket depth (PD) classes (<4 mm, 4–6 mm, >6 mm) in control (n = 25) and test (n = 25) patients at registrations before (T0) and after (T2) combined periodontal–orthodontic treatment. Also change of PD class from T0 to T2

<table>
<thead>
<tr>
<th>PD (mm)</th>
<th>Group</th>
<th>T0 Median (Q1–Q3)</th>
<th>T0 % Sites</th>
<th>T2 Median (Q1–Q3)</th>
<th>T2 % Sites</th>
<th>T0–T2 PD level change (mm)</th>
<th>Group</th>
<th>T0 Median (Q1–Q3)</th>
<th>T0 % Sites</th>
<th>T2 Median (Q1–Q3)</th>
<th>T2 % Sites</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;4</td>
<td>Control</td>
<td>51.8 (40.6–66.5)</td>
<td>90.6 (84.1–95.7)</td>
<td>90.6 (84.1–95.7)</td>
<td>&lt;4 and &lt;4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Control</td>
<td>51.8 (40.6–66.5)</td>
<td>.13</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Test</td>
<td>69.4 (54.7–76.4)</td>
<td>93.8 (87.7–95.3)</td>
<td>93.8 (87.7–95.3)</td>
<td>&lt;4 and 4–6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Test</td>
<td>62.5 (50.5–74.0)</td>
<td>.14</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>4–6</td>
<td>Control</td>
<td>40.9 (26.8–50.0)</td>
<td>9.4 (4.3–15)</td>
<td>9.4 (4.3–15)</td>
<td>4–6 and &lt;4&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Control</td>
<td>30.4 (18.8–45.9)</td>
<td>.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Test</td>
<td>26.4 (19.8–33.7)</td>
<td>6.0 (3.9–11.5)</td>
<td>6.0 (3.9–11.5)</td>
<td>4–6 and 4–6&lt;sup&gt;d&lt;/sup&gt;</td>
<td>Test</td>
<td>20.5 (13.6–25.5)</td>
<td>.29</td>
<td></td>
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<tr>
<td>&gt;6</td>
<td>Control</td>
<td>4.0 (1.0–8.7)</td>
<td>0</td>
<td>0</td>
<td>&gt;6 and &lt;4&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Control</td>
<td>1.3 (0–5.0)</td>
<td>.52</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Test</td>
<td>3.6 (1.1–6.7)</td>
<td>0</td>
<td>0</td>
<td>&gt;6 and 4–6&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Test</td>
<td>1.1 (0–2.9)</td>
<td>.69</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt;6 and &gt;6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Control</td>
<td>1.2 (0–6.1)</td>
<td>.69</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&gt;6 and &gt;6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Test</td>
<td>2.0 (0–3.1)</td>
<td>.69</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PD, pocket depth; Q1, the first quartile; Q3, the third quartile; control group n = 25; test group n = 25; T0, baseline; T2, after total treatment.
<sup>a</sup>No change.
<sup>b</sup>Increase.
<sup>c</sup>Decrease.
<sup>*</sup>P value by Mann–Whitney U test.

FIGURE 3  Treatment progress of Angle II-1 patient with plaque-induced periodontitis baseline (a), after periodontal treatment (b), during orthodontic treatment (c, d) and after treatment (e) with straight-wire mechanics and microimplant used for anchorage. Periapical radiograph at baseline (f)
periodontal–orthodontic treatment time was significantly longer for the control group, where the final periodontal treatment was performed before the orthodontic treatment. Based on the results of this trial, we conclude that orthodontic treatment, simultaneously to the periodontal treatment, could be used in the routine treatment of patients with plaque-induced periodontitis.

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CONFLICT OF INTEREST
No external funding—except support from the authors’ institution—was available for this study. We declare that we have no conflict of interest in this study.

ORCID
Eglė Zasčiurinskienė http://orcid.org/0000-0001-8364-0510

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