Treatment of periodontal-endodonic lesions — a systematic review


Abstract
Background: The treatment of periodontal-endodonic lesions is challenging due to the involvement of both periodontal and endodontic tissues.
Objective: To evaluate the treatment options and outcomes of periodontal-endodonic lesions.
Material and Methods: A systematic literature search was performed for articles published by 12 May 2013 using electronic databases and hand search. Two reviewers conducted the study selection, data collection and validity assessment. The PRISMA criteria were applied. From 1087 titles identified by the search strategy, five studies and 18 case reports were included.
Results: Clinical studies and case reports were published from the years 1981 to 2012. A pronounced heterogeneity exists among studies regarding applied treatment protocols and quality of reporting. In all clinical studies, comprising 111 teeth, a non-surgical root canal treatment (RCT) was performed as initial treatment step. Non-surgical and/or a surgical periodontal therapy was applied in some studies without re-evaluation of the endodontic healing. Probing pocket depth reductions were reported in all included studies, comprising the data from 80 teeth at follow-up.
Conclusions: A sequential treatment with root canal treatment as a first treatment step appears to be reasonable. An adequate time for tissue healing is suggested prior to re-evaluation.

A communication between the periodontium and the dental pulp exists through different pathways including the apical foramina, lateral and accessory canals or exposed dentinal tubules (Seltzer et al. 1963, Rotstein & Simon 2006). These anatomical structures enable the development of diseases with concurrent periodontal and pulpal tissue involvement, e.g. so-called periodontal-endodonic lesions.

An endodontic lesion may elicit an inflammatory response in the adjacent periodontal tissue and thus can cause a retrograde periodontitis (Simring & Goldberg 1964). A periodontal lesion may provoke an extensive pulp pathosis (Langeland et al. 1974, Bergenholtz & Lindhe 1978). In addition, periodontal and endodontic diseases can develop independently. Teeth with a periodontal-endodonic lesion feature an inflamed or necrotic pulp tissue and increased probing pocket depths leading to great variability of possible diagnostic findings, e.g. altered response to pulp sensibility testing, multiple or singular increased periodontal probing depth, sinus tract, pus, radiological J-shaped lesion or infrabony defect (Harrington 1979, Abbott 1998). The retrospective identification of the primary cause of a periodontal-endodonic lesion, however, is not possible in most cases.

Several classification systems were applied to describe these lesions according to their pathogenesis or with respect to a suggested therapy sequence (Simon et al. 1972, Guldener 1975, Geurtsen et al. 1985, Dietrich et al. 2002). However, the...

The treatment of these lesions is a challenge and treatment planning needs to consider the involvement of the periodontal and pulpal compartment. A wide spectrum of different therapeutic options and/or therapeutic sequences including endodontic and periodontal treatment with nonsurgical and/or surgical procedures with or without local or systemic antibiotics has been described in case reports and clinical studies.

Review of Current Literature

Objective

The purposes of the present systematic review were to identify the treatment options and to evaluate the therapy with respect to the outcomes of combined periodontal-endodontic lesions in humans with respect to tooth loss and probing pocket depth (PPD) reduction.

The specific questions addressed in this systematic review were:

- Which treatments/treatment sequences were applied?
- In case the treatment was performed at different time points, what was the time interval for re-evaluation of the first treatment step?
- Which rates of tooth loss occurred after treatment?
- Does treatment of combined periodontal-endodontic lesions lead to a PPD reduction?

Material and Methods

Protocols

The present systematic review considers the PRISMA (Preferred Reporting Items for Systematic Review and Meta-analyses) criteria (Liberati et al. 2009, Moher et al. 2009) (Appendix S1). The research questions were adapted using the PICO (Patient, Intervention, Comparison, Outcomes) criteria (Miller & Forrest 2001).

Eligibility and ineligibility criteria

Publications were considered eligible for inclusion in this systematic review if they presented the following parameters: (1) the presence of a combined periodontal-endodontic lesion defined as the co-existence of a negative or altered pulp vitality test and PPD ≥ 6 mm on at least one tooth site in case reports or a mean probing pocket depth ≥ 6 mm in interventional studies and case series and (2) a follow-up after treatment of at least 6 months. No restrictions in terms of type of publication, i.e. case report, clinical study, review, language and time point of publication were applied. Review articles were screened for the presentation of case reports within the review. A publication considered ineligible for inclusion was hierarchically categorized (Appendix S4 and S5).

Outcome measures

The primary outcome measure was tooth loss after treatment of combined periodontal-endodontic lesions. Changes in PPD, probing attachment level (PAL), gingival recession (REC) and radiographic findings were evaluated as secondary outcome variables.

Information sources

The electronic databases MEDLINE by OVID and EMBASE and the grey literature (www.opengrey.eu) were searched for studies published prior to May 12, 2013. A hand search was performed on Journal of Clinical Periodontology, Journal of Periodontology, Journal of Endodontics and International Endodontic Journal from January 1990 to May 2013. Moreover, the references of publications examined for inclusion were thoroughly analysed to search for additional publications.

Results

Analysis of individual studies

A total of 1087 titles from the electronic databases were identified (Fig. 1). The titles and abstracts were screened by two reviewers (observed agreement = 90.16%, kappa = 0.773). The full texts of 186 publications (selected by at least one reviewer) were further analysed. Full text analysis led to exclusion of further 171 studies (Appendix S4 and S5). Finally, 15 publications from the electronic search satisfied the
inclusion criteria. Eight additional publications were identified by screening the references of studies evaluated for inclusion.

Description of characteristics, results, and quality assessment

The analysis for clinical studies and case reports was performed separately. The characteristics of included studies (Table 1) and case reports (Table 2, Appendix S6) and the assessment of risk of bias (Appendix S7) are summarized in tables.

Summary of characteristics (PICO – Population, Intervention/Comparison, Outcomes)

Population – number and characteristics of patients and tooth sites. The mean age of patients in two of the included publications amounted 44 and 51.2 years (Haueisen et al. 2000, Li et al. 2012). The patients’ age in two other publications ranged from 25 to 53 years (Hassan et al. 1986, Sun & Liu 2009), whereas it was not available in one case series (Ratka-Kruger et al. 2000). The proportion of females and males was provided in two publications including each 18 males and 12 females (Sun & Liu 2009, Li et al. 2012). Two studies stated exclusion criteria in terms of the presence of systemic diseases, increased mobility of teeth, tobacco use, pregnancy and the use of antibiotics during the observation period (Hassan et al. 1986, Li et al. 2012). Two case series stated the proportion of upper and lower teeth with a large majority of teeth in the mandibula (Haueisen et al. 2000, Ratka-Kruger et al. 2000).

The mean PPD at baseline ranged from 6.01 ± 0.31 mm to 14.29 ± 1.76 mm (Hassan et al. 1986, Li et al. 2012). The individual PPD values ranged from 9 to 17 mm (Hassan et al. 1986, Haueisen et al. 2000). Clinical symptoms in terms of pain, thermal sensitivity, gingival swelling, pus discharge from gingival margin, increased mobility and sensitivity to percussion were occasionally reported (Hassan et al. 2000, Ratka-Kruger et al. 2000, Sun & Liu 2009, Li et al. 2012). The appearance of a sinus tract was reported in some patients (Haueisen et al. 2000, Ratka-Kruger et al. 2000). In two case series, all included teeth demonstrated either a caries profunda treatment combined with a filling or a crown restoration (Haueisen et al. 2000, Ratka-Kruger et al. 2000). The presence of a restoration or a carious lesion was not reported in the remaining publications.

The radiographic findings were described as periapical, lateral and/or inter-radicular radiolucencies (Haueisen et al. 2000, Ratka-Kruger et al. 2000, Li et al. 2012). Moreover, Ratka-Kruger et al. (2000) specified the pattern of bone loss on radiographs as local and narrow, extending to the apex and frequently without any approximal bone loss.

In patients with combined periodontal-endodontic lesions:

Which treatments/treatment sequences were applied? Different treatments of combined periodontal-endodontic lesions, including periodontal and endodontic approaches with non-surgical and surgical methods were performed. In all publications, a non-surgical root canal treatment was performed as initial treatment step. A two-staged RCT with a temporary intracanal medication was applied in two case series. After re-evaluation no further endodontic or periodontal
<table>
<thead>
<tr>
<th>First author (year of publication)</th>
<th>Study design</th>
<th>Number of teeth and patients, age, gender, exclusion criteria</th>
<th>Tooth type (n)</th>
<th>Clinical manifestations (n)</th>
<th>Radiographic manifestations (n)</th>
<th>Additional diagnoses (n)</th>
<th>Treatment sequence</th>
<th>Follow-up</th>
<th>Calculated tooth loss (n)</th>
<th>PPD at follow-up</th>
<th>Additional outcomes</th>
<th>Number of drop-outs (patients) and reasons</th>
<th>Confounding factors considered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hassan et al. (1986)</td>
<td>Prospective case series</td>
<td>14 teeth 14 patients 25–40 yrs Inclusion criteria: systemic diseases, teeth excessively mobile requiring splinting</td>
<td>Maxillary and mandibular incisors (14)</td>
<td>Not specified</td>
<td>Mean PPD 14.29 ± 1.36 mm (range 12–17 mm)</td>
<td>No vitality (14)</td>
<td>SRP + Ocl – (2 w)</td>
<td>+6 mo</td>
<td>2</td>
<td>6.125 ± 4.076 mm (mean ± SD)</td>
<td>In 10 patients: 3.2–5.2 mm (range)</td>
<td>Mean attachment gain 8.16 ± 2.9 mm in 10 patients: attachment gain 7.9–14 mm in 2 patients: radiographic signs of partially bone fill, continuity of lamina dura</td>
<td>2, NFD</td>
</tr>
<tr>
<td>Haueisen et al. (2000)</td>
<td>Retrospective case series</td>
<td>10 teeth 51.2 yrs (mean)</td>
<td>Mandibular molars (10)</td>
<td>Narrow PPD 9–12 mm (10)</td>
<td>Pain (1)</td>
<td>Pain discharge from gingival margin (3)</td>
<td>FI (7)</td>
<td>Mobility grade I (2), grade II (2)</td>
<td>No vitality (7), unclear response to pulp test (3)</td>
<td>Sensitivity to percussion (2)</td>
<td>CP treatment and filling (2), crown or partial crown (8)</td>
<td>Fistula (4)</td>
<td>RCT1 – (CD, 3 w) – RCT2 (GP, S, LC)</td>
</tr>
<tr>
<td>Tooth Type/Number of Teeth and Patients, Age, Gender, Exclusion Criteria</td>
<td>Clinical Manifestations</td>
<td>Radiographic Manifestations</td>
<td>Additional Diagnosis</td>
<td>Treatment Sequence</td>
<td>Follow-up</td>
<td>Calculated Tooth Loss</td>
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<tr>
<td>Retrospective case series</td>
<td>Mean PPD 11.3 mm</td>
<td>Local narrow bone loss to apex, interradicular and/or periapical radiolucency without interproximal bone loss in molars, periapical radiolucency (&gt;0.5 of root length) with widened periodontal ligament space</td>
<td>Fistula RCT1 – RCT2 (CD, within 2 mo) – RCT2 (GP, S, LC)</td>
<td>+6 mo 5 3.1 mm (mean) No FI in 10 of 15 molars, radiographic signs of improvement up to complete regeneration in most teeth</td>
<td>5, NFD</td>
<td>+12 mo 11 2.2 mm (mean) No FI in 6 of 9 molars, radiographic signs of improvement up to complete regeneration in most teeth</td>
<td>11, NFD</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Sun &amp; Liu (2009)</td>
<td>Teeth with 6 mo follow-up (n = 29) Mean PPD 6.62 mm 0.230 mm Mean CAL 8.76 mm 0.209 mm Mean mobility 1.66 mm 0.134 mm Pain Swelling No vitality (37)</td>
<td>Bone loss – RCT1 – RCT2 (1 w) – Occlusal SRP – RCT2 (4 w) – PS (MWF, BA, D, AB amox/metro)</td>
<td>+6 mo 8 2.83 mm* Mean CAL 6.79 mm 0.182 mm* Mean mobility 1.38 mm 0.092 mm*</td>
<td>8, NFD Tooth type, age, experience of therapist</td>
<td>+12 mo 18 2.42 mm* Mean CAL 6.58 mm 0.268 mm* Mean mobility 1.26 mm 0.104 mm*</td>
<td>18, NFD</td>
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* Denotes significant difference
<table>
<thead>
<tr>
<th>First author</th>
<th>Study design</th>
<th>Number of teeth and patients</th>
<th>Tooth type</th>
<th>Clinical manifestations</th>
<th>Radiographic manifestations</th>
<th>Additional diagnosis</th>
<th>Treatment sequence</th>
<th>Follow-up</th>
<th>Calculated tooth loss</th>
<th>PPD at follow-up</th>
<th>Additional outcomes</th>
<th>Number of drop-outs (patients) and reasons</th>
<th>Confounding factors considered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Li et al.</td>
<td>Prospective randomized controlled trial</td>
<td>30 teeth 30 patients 44 yrs (mean)</td>
<td>18 male, 12 female</td>
<td>Exclusion criteria: systemic diseases, smoking, pregnancy, antibiotic intake during observation period</td>
<td>Test: anterior teeth (2), bicuspids (5), molars (9)</td>
<td>Control: anterior teeth (6), bicuspids (4), molars (7)</td>
<td>Control: RCT1 (RF, S, LC) + SRP Test: RCT1 (RF, diode laser irradiation, LC) + SRP (diode laser irradiation)</td>
<td>+3 mo 0</td>
<td>+6 mo 0</td>
<td>0</td>
<td>Tooth type, age, smoking, systemic diseases, experience of therapist</td>
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</tbody>
</table>

**Note:**
- PPD, probing pocket depth; PS, periodontal surgery; RCT, root canal treatment; RBF, reverse bevel flap; RF, rotary files; S, sealer; SRP, scaling and root planing; w, weeks; yrs, years.
- *p < 0.05 (difference between baseline and outcome).
- **p < 0.05 (difference between control and test group); the wording used in the table was adapted from the publications included.
- †Time related to last treatment step.

"xenograft; b1.5 W, continuous wave, one application (15 s); c1 W, continuous wave, one application daily (15 s) for 5 days; AB, systemic antibiotics; amox, amoxicillin; BG, bone graft; CAL, clinical attachment loss; CD, calcium hydroxide dressing; Cp, caries profunda; D, periodontal dressing; FI, furcation involvement; GP, gutta percha; LC, lateral condensation; M, membrane; mBI, modified bleeding index; metro, metronidazole; mo, months; MWF, modified Widman flap; NFD, not further described; Occl, occlusal adjustment; PAI, periapical index; PPD, probing pocket depth; PS, periodontal surgery; RCT, root canal treatment; RBF, reverse bevel flap; RF, rotary files; S, sealer; SRP, scaling and root planing; w, weeks; yrs, years.

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<table>
<thead>
<tr>
<th>First author (year of publication)</th>
<th>Gender</th>
<th>Age</th>
<th>Perio*</th>
<th>Smoking</th>
<th>Tooth type</th>
<th>PPD (mm) at baseline</th>
<th>Clinical manifestations</th>
<th>Radiographic manifestations</th>
<th>Additional diagnosis</th>
<th>Treatment sequence</th>
<th>Follow-up (mo)</th>
<th>PPD (mm) at follow-up</th>
<th>Additional outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moucachen et al. (1981)</td>
<td>M</td>
<td>50</td>
<td>NR</td>
<td>NR</td>
<td>38</td>
<td>12</td>
<td>NV, Per</td>
<td>A, L</td>
<td>–</td>
<td>Extraction</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Geurtsen et al. (1985)</td>
<td>F</td>
<td>17</td>
<td>NR</td>
<td>NR</td>
<td>46</td>
<td>13</td>
<td>NV, R</td>
<td>A, L, IR, wPLS</td>
<td>–</td>
<td>Extraction</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Stamatis &amp; Johnson (1994)</td>
<td>M</td>
<td>64</td>
<td>NR</td>
<td>NR</td>
<td>26</td>
<td>7</td>
<td>NV, Pu, M0, R</td>
<td>A, L, IR</td>
<td>–</td>
<td>RCT1 + RCT2</td>
<td>12</td>
<td>2-3</td>
<td>BF</td>
</tr>
<tr>
<td>Hauieisen et al. (1999)</td>
<td>M</td>
<td>59</td>
<td>NR</td>
<td>NR</td>
<td>36</td>
<td>12</td>
<td>UV, Pu, Per, R</td>
<td>A, L</td>
<td>–</td>
<td>RCT1 + RCT2</td>
<td>18</td>
<td>2</td>
<td>BF</td>
</tr>
<tr>
<td>Hauieisen et al. (1999)</td>
<td>M</td>
<td>56</td>
<td>NR</td>
<td>NR</td>
<td>46</td>
<td>12</td>
<td>UV, Pu, Per, R</td>
<td>A, L, IR</td>
<td>–</td>
<td>RCT1 + RCT2</td>
<td>12</td>
<td>2-3</td>
<td>BF</td>
</tr>
<tr>
<td>Ratka-Krüger et al. (1999)</td>
<td>M</td>
<td>56</td>
<td>NR</td>
<td>NR</td>
<td>47</td>
<td>12</td>
<td>NV, Pu, S, R, FI</td>
<td>A, L, IR</td>
<td>Ab, Fis</td>
<td>RCT1 + RCT2</td>
<td>6</td>
<td>3</td>
<td>BF</td>
</tr>
<tr>
<td>Zucchelli (2007)</td>
<td>F</td>
<td>39</td>
<td>P</td>
<td>NR</td>
<td>12</td>
<td>12</td>
<td>UV, M3, Rec, infraBDh</td>
<td>A, L</td>
<td>Fis</td>
<td>RCT1 + SRP + Spl –</td>
<td>36</td>
<td>3</td>
<td>BF, M0</td>
</tr>
<tr>
<td>Zehnder (2001)</td>
<td>M</td>
<td>28</td>
<td>AgP NR</td>
<td>NR</td>
<td>15</td>
<td>8</td>
<td>NV, Pu, Pu, Per, M3</td>
<td>L</td>
<td>Fis</td>
<td>RCT1 + SRP + RCT1 + SRP + Spl –</td>
<td>36</td>
<td>3</td>
<td>BF, M0</td>
</tr>
<tr>
<td>Zubery &amp; Machtei (1993)</td>
<td>F</td>
<td>23</td>
<td>AgP NR</td>
<td>NR</td>
<td>11</td>
<td>10</td>
<td>NV, Pa, S, M2, bBLh</td>
<td>A, L</td>
<td>Fis</td>
<td>RCT1 + SRP –</td>
<td>12</td>
<td>2</td>
<td>BF, M0-1</td>
</tr>
<tr>
<td>Walter et al. (2008)</td>
<td>M</td>
<td>53</td>
<td>ChP HS</td>
<td>NR</td>
<td>32</td>
<td>8i</td>
<td>NV, R, Fe, infraBDh</td>
<td>A, L</td>
<td>–</td>
<td>RCT1 + SRP + RCT1 + SRP + Spl –</td>
<td>6</td>
<td>5</td>
<td>BF, M1, AttG 3 mm</td>
</tr>
<tr>
<td>Yavuz et al. (2008)</td>
<td>M</td>
<td>25</td>
<td>NR</td>
<td>NR</td>
<td>22</td>
<td>7</td>
<td>NV, S, bBLh, infraBDh</td>
<td>A, L, AccR</td>
<td>Fis</td>
<td>RCT1 + SRP + Spl –</td>
<td>12</td>
<td>3-4</td>
<td>BF</td>
</tr>
<tr>
<td>Ghezzi et al. (2012)</td>
<td>M</td>
<td>38</td>
<td>NR</td>
<td>NR</td>
<td>21</td>
<td>9</td>
<td>NV, Pu, infraBDh, bBLh</td>
<td>L</td>
<td>–</td>
<td>RCT1 + SRP + Spl –</td>
<td>24</td>
<td>3</td>
<td>BF</td>
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</table>
therapy was deemed to be necessary in these case series (Haueisen et al. 2000, Ratka-Krugger et al. 2000). The remaining publications combined an endodontic with a periodontal treatment. Li et al. (2012) selected a non-surgical approach by combining a root canal treatment with the root debridement of involved teeth with (test group) or without (control group) diode laser irradiation. The laser was applied both during RCT and after initial periodontal treatment in a mode aiming in bacterial reduction. Two publications added periodontal surgery to a conventional non-surgical periodontal and endodontic treatment. Hassan et al. (1986) applied citric acid as conditioning agent to the root surfaces within an open flap debridement. Sun & Liu (2009) used a xenogenous bone graft material and adjuvant systemic antibiotics in addition to surgery.

In case the treatment was performed at different time points, what was the time interval for re-evaluation of the first treatment step? In two retrospective case series non-surgical endodontic treatment was performed as a first treatment step and the decision for any further therapy was left open and performed 6 months after re-evaluation of healing after RCT (Haueisen et al. 2000, Ratka-Krugger et al. 2000). In both case series, in all re-evaluated patients, no further therapy was deemed necessary since the initial RCT was classified as successful according to clinical and radiographic criteria.

Which rates of tooth loss occurred after treatment? No tooth loss was reported in either publications. The reported drop-out rate was two out of 14 (Hassan et al. 1986), 11 out of 20 (Ratka-Krugger et al. 2000) and 18 out of 37 (Sun & Liu 2009). No drop-outs were reported in the remaining two publications. As a result, the remaining two publications were reported in the remaining two publications. As a result, the remaining two publications were reported in the remaining two publications.
Does treatment of combined periodontal-endodontic lesions lead to a PPD reduction? Probing pocket depth reductions in terms of a decreased mean PPD or a smaller PPD range occurred from baseline to follow-up in all included publications for the teeth at follow-up. Data are reported after 6 months in all included publications. Three case series provided additional data after 12 months (Haueisen et al. 2000, Ratka-Krüger et al. 2000, Sun & Liu 2009).

The PPD after a follow-up of 6 months decreased from a mean PPD of 14.29 ± 1.76 to 6.125 ± 4.076 mm (Hassan et al. 1986). The individual values of the PPD ranged from 3.2 mm to 15.3 mm (Hassan et al. 1986).

In a randomized controlled trial, Li et al. (2012) reported a significantly different PPD reduction after a follow-up of 6 months from a mean PPD of 6.04 ± 0.37 to 5.64 ± 0.33 mm in the control group and from a mean PPD of 6.01 ± 0.31 to 4.34 ± 0.23 mm in the test group. The PPD decreased from a mean PPD of 11.3 mm to 3.1 mm after 6 months and to 2.2 mm after 12 months (Ratka-Krüger et al. 2000). After a follow-up of 6 months, the reported PPD range of 9 to 12 mm at baseline declined to 2 to 4 mm with no further changes up to 12 months (Haueisen et al. 2000).

A statistically significant reduction of the PPD after 6 months, i.e. from a mean PPD 6.62 ± 0.230 to 2.83 ± 0.149 mm and after 12 months, i.e. from mean PPD of 6.74 ± 0.285 to 2.42 ± 0.233 mm was noted by Sun & Liu (2009).

Discussion

Herein, we conducted a systematic review to evaluate the best available external evidence regarding the treatment options and outcomes of periodontal-endodontic lesions. A total of five studies, i.e. one randomized controlled clinical trial, two prospective and two retrospective case series, and 18 case reports were included from a systematic literature search. Due to an assumed scarcity of clinical studies, the initial purpose of this systematic review was to evaluate case reports supposed to be the best external evidence on this subject. As intervention studies and case series, however, were also found fulfilling the inclusion criteria (Fig. 1), the data were presented separately for clinical studies (Table 1) and case reports (Table 2, Appendix S6) according to the suspected hierarchy of best available external evidence.

A plethora of classifications for lesions with periodontal and endodontic tissue involvement has been proposed. Several authors suggested the differentiation between the primary cause(s) of the lesion, i.e. of periodontal or endodontic origin (Simon et al. 1972, Guldener 1975, Geurtsen et al. 1985). However, the correct identification of the disease onset is frequently impeded and remains of speculative nature in many cases, due to an incomplete dental history or critical results of a pulp sensitivity test, in particular in multi-rooted teeth. According to recent suggestions (Armitage 1999, Lang et al. 1999, Abbott & Salgado 2009), in this systematic review, only publications that described a well-defined feature of periodontal-endodontic lesions, i.e. teeth with a negative or an unclear response to a pulp sensibility test and an increased probing pocket depth, were considered (Appendix S4 and S5). Interestingly, three studies included neither used a classification system nor determined the disease onset probably reflecting the difficulties and limitations in differential diagnosis (Hassan et al. 1986, Sun & Liu 2009, Li et al. 2012).

The publications in this systematic review were published within a time frame from 1981 to 2012. As one finding from this review, nonsurgical RCT as a sole treatment approach was performed by some early publications whereas guided tissue regeneration (GTR) procedures were more frequently applied in recent publications (Stamas & Johnson 1994, Ghezzi et al. 2012). Time point of publication needs to be considered as a relevant parameter within a review, because dental treatment options and/or materials have changed dramatically over time (Hassan et al. 1986, Li et al. 2012, Rodriguez et al. 2013), probably leading to difficulties in comparison among the studies.

Although the English language is commonly accepted as the language of scientific research, relevant external evidence could also arise from studies in languages other than English (Morrison et al. 2012), in particular from members of the BRIC countries (Brazil, Russia, India, China) or findings from the early days of scientific periodontology (Schmidt et al. 2013). In addition, it was suggested that positive research findings are more likely to be published in an international English-language journal whereas negative results may be rather reported in a non English-language journal (Egger et al. 2003). The influence of language restrictions on the outcome of systematic reviews is uncertain in particular fields of medicine (Morrison et al. 2012). The exclusion of non English-language studies from a systematic review, however, may lead to a language bias. The literature search of the present systematic review was performed without any language restrictions. Two original studies in Chinese language have been translated. They fulfilled the inclusion criteria and were included comprising 67 teeth out of 111 analysed teeth (Sun & Liu 2009, Li et al. 2012).

Treatment planning includes the consideration of diagnostic and prognostic factors deemed to influence the treatment outcome of periodontal-endodontic lesions (Lang & Tonetti 2003, Kwok & Caton 2007). However, in addition to mode of endodontic and/or periodontal treatment, potential confounding factors, e.g. patient’s age, gender, tooth type, smoking, periodontal history and chronicity of lesions, were inconsistently considered in the included publications resulting in a high risk of bias (Appendix S7). Further research analysing the impact of these factors on the pathogenesis and the outcome of therapy of periodontal-endodontic lesions is required. No tooth loss was reported except for two case reports. In case series and case reports, success and/or survival rates may be subject to a publication bias as a treatment success.
may be more likely to be published. Therefore, treatment failures could be underrepresented. Several drop-outs occurred in three included publications (Hassan et al. 1986, Ratka-Krüger et al. 2000, Sun & Liu 2009). Such drop-out rates pose a threat to validity of any study (Walter et al. 2012a). In general, there are different possibilities to handle drop-outs including the so-called “worst case” analysis. As reasons for drop-outs were not provided, this method was applied in the present review and patients who dropped were assumed to may have lost their treated teeth (Myers 2000, Sutherland 2001, Wang et al. 2004). The tooth loss rate ranged from 0 to 27.9% resulting in a survival rate of 72.1 to 100%. The survival rate may be affected by the eligibility criteria, including the PPD range, applied in the randomized controlled clinical trial and in the case series. For example, the exclusion of hypermobile and multi-rooted teeth seems to be of prognostic relevance and may have implications on the treatment outcome (Hassan et al. 1986). The herein reported survival rate is within the previously noted range of survival rates. Schacher et al. found a survival rate of 61% after at least 5 years to retain teeth with treated periodontal-endodontic lesions (Schacher et al. 2007). In addition, a prospective clinical trial reported a favourable outcome of 77.5% in apicomarginal defects including periodontal-endodontic lesions (Kim et al. 2008). In contrast to the reported tooth loss and/or survival rate, the PPD reduction represents an alternative study end point, possibly representing a success rate from a strictly periodontal point of view. A PPD reduction was achieved in nearly all re-evaluated teeth, but there may be remaining deep probing depths (Hassan et al. 1986, Li et al. 2012). However, the PPD of the included teeth differed remarkably between the clinical studies (Hassan et al. 1986, Li et al. 2012). Keeping in mind, that a narrow localized deep PPD may represent a draining sinus tract of endodontic origin that healed after root canal treatment (Weiger et al. 1995, Abbott & Salgado 2009).

The data from clinical studies and case reports were analysed separately with respect to the suspected hierarchy of external evidence (Hujeel 2008). In general, the evidence levels refer to the study design suggesting randomized controlled clinical trials as higher available external evidence compared to other study designs (Hujeel 2008). For this reason, some authors feel there is a lack of direct evidence in case an (meta-analysis of) RCT’s is missing (Walter et al. 2012b, Schmidt et al. 2013, Walter & Friedmann 2013). In randomized controlled clinical trials, patients need to be assigned to a predefined protocol. This design may exclude the opportunity of alteration of a treatment plan, which may be indicated after evaluation of the tissue response after root canal treatment. Healing of lesions of endodontic origin depends on many modifying factors and may occur within a large time frame (European Society of Endodontology 2006, Ricucci et al. 2009, Walter et al. 2008). Prospective case series or cohort studies permit modifications of the treatment plan and sequence with respect to individual healing responses and findings at different time points. A (prospective) case series seems to be more reasonable to evaluate the treatment of periodontal-endodontic lesions at the current stage of knowledge. Four case series and 18 case reports were included in this review. In two case series and four case reports including five teeth, a non-surgical endodontic treatment was performed as initial treatment and the decision for further procedures were based on the tissue response at re-evaluation (Stamas & Johnson 1994, Haueisen et al. 1999, 2000, Ratka-Krüger et al. 1999, 2000, Walter et al. 2008). The remaining publications predefined the treatment sequence, i.e. a combined treatment consisting of non-surgical endodontic therapy with a non-surgical and/or a surgical periodontal therapy. Periodontal surgery consisted of an open flap debridement alone or in combination with regenerative or resective procedures.

Incomplete tissue healing after evaluation of the first treatment step may lead to the decision of further procedures (Dietrich et al. 2003). Healing of periapical lesions may take up to 4 years (European Society of Endodontology 2006) or longer (Ricucci et al. 2009). This process may be modified by several hereditary, environmental and behavioural risk factors, e.g. a smoking habit (Walter et al. 2008, 2012b, Rodriguez et al. 2013). Similarly, the improvement of periodontal conditions subsequent to scaling and root planing may take several months, with the deeper the initial PPD the longer the time for PPD reduction (Badersten et al. 1984). An observation time of at least 6 to 12 months before re-evaluation of the first treatment step was suggested by Zehnder (Zehnder 2001). The publications included in this review reported a follow-up time between six and 12 months. However, with respect to the individual range of the time required for periodontal tissue healing and the absence of radiographic changes in early stages of healing, the interpretation of treatment results after six to 12 months seems to be associated with some bias (Hirsch et al. 1989, Jansson et al. 1997, Kim et al. 2008). The results from included studies and case series may need to be regarded as short-term outcomes. Follow-up examinations after an observation period of several years would be desirable although challenging.

With respect to the uncertain treatment outcome of periodontal-endodontic lesions and the successful outcome of less invasive treatment approaches (Stamas & Johnson 1994, Haueisen et al. 1999, 2000, Ratka-Krüger et al. 1999, 2000), the concept of a sequential treatment with re-evaluation at different treatment stages may also be supported from an economic point of view. Financial costs and time efforts increase dramatically with more invasive treatment approaches (Walter et al. 2012c). On the other hand patient-centred outcomes, including quality of life, treatment time and costs, are of increasing importance within clinical trials to ensure an appropriate allocation of resources (Brägger 2005, Walter et al. 2012c).

Conclusions and Clinical Relevance
Taken together, this review may provide some evidence for root canal treatment as a first treatment step for the treatment of combined periodontal-endodontic lesions with increased PPD and a negative response of pulp sensibility testing. A reasonable time for healing of the endodontic lesion after root canal


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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. PRISMA 2009 checklist.

Appendix S2. Electronic search strategy for the databases MEDLINE and EMBASE.

Appendix S3. Scores for assessment of methodological and reporting quality.

Appendix S4. Studies and case series excluded based on full text analysis, and reason for exclusion.

Appendix S5. Case reports and reviews based on full text analysis, and reason for exclusion.

Appendix S6. Characteristics of included case reports in detail.

Appendix S7. Quality assessment of included studies.

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Clinical Relevance

Scientific rationale for the study: Different therapeutic approaches including endodontic and/or periodontal procedures have been proposed for the treatment of periodontal-endodontic lesions.

Principal findings: In this systematic review, a successful outcome in terms of probing pocket depth reduction of exclusive non-surgical endodontic therapy was identified for teeth with a negative response to pulp sensibility testing combined with increased probing pocket depth.

Practical implications: The findings from this systematic review may support an initial root canal treatment for the treatment of this kind of periodontal-endodontic lesions. A reasonable time in advance of consideration of further procedures is suggested.