CASE REPORT

Step-wise treatment of two periodontal-endodontic lesions in a heavy smoker

C. Walter, G. Krastl & R. Weiger

Department of Periodontology, Endodontology and Cariology, University of Basel, Basel, Switzerland

Abstract

Walter C, Krastl G, Weiger R. Step-wise treatment of two periodontal-endodontic lesions in a heavy smoker. *International Endodontic Journal*, **41**, 1015–1023, 2008.

Aim To report a clinical case of two advanced periodontal-endodontic lesions with a focus on treatment issues related to tobacco use.

Summary A 53-year-old Caucasian male was referred to the School of Dentistry, Basel, Switzerland, for periodontal treatment. The major diagnoses were chronic (smoker) periodontitis and advanced combined periodontal-endodontic lesions on the mandibular left lateral incisor and right incisor. Conventional root canal treatment was performed, and subsequently led to reduced radiolucencies around the affected roots after 14 months. The remaining osseous defect was augmented by guided tissue regeneration using bovine bone substitute and resorbable membrane. The follow-up revealed a stable situation from clinical (probing depth 2–4 mm) and radiological points of view 32 months after initiation of treatment. Treatment considerations related to tobacco use are discussed.

Key learning points

• After conventional root canal treatment, osseous healing should occur before further complementary therapy is taken into account.

• Issues related to tobacco use have to be considered before treatment is initiated.

Keywords: periodontal-endodontic lesion, regenerative periodontal surgery, root canal treatment, smoking, tobacco use, wound healing.

Received 11 February 2008; accepted 9 June 2008

Introduction

Periodontal-endodontic lesions may be primarily of either endodontic or periodontal origin, or caused by simultaneous development of both diseases (Simon *et al.* 1972). However,

Correspondence: Clemens Walter, Department of Periodontology, Endodontology and Cariology, University of Basel, Hebelstrasse 3, 4056 Basel, Switzerland (Tel.: +41 61 2672623; fax: +41 61 2672659; e-mail: clemens.walter@unibas.ch).

the aetiology is often difficult to assess in individual cases. In addition to specific treatment considerations, the periodontal-endodontic lesion category was included in the classification of periodontal diseases and conditions in 1999 because of its explicit aetiology (Armitage 2000). Patient history, current status of the entire dentition, and sensibility testing of the affected teeth/tooth are essential for deciding how to treat the disease. Apart from correct diagnosis and treatment sequence, chronicity of the lesion is also an important factor for clinical success (John *et al.* 2004). Root canal treatment is recommended as the initial step in cases with negative results to pulp sensibility testing according to a rare study on periodontal-endodontic treatment (Vakalis *et al.* 2005). Adequate root canal treatment induces healing of lesions in this tissue. Complete healing is often possible, presumably due to an intact periodontal ligament on the root surface (Solomon *et al.* 1995). Furthermore, periodontal conservative or surgical treatment should only be initiated after radiological evaluation of the healing process. The timing of the healing process depends largely on various hereditary, environmental or behavioural risk factors.

Tobacco use alters the turnover of cells during periodontal tissue repair and the immune response of the affected host. This modifiable habit is considered a major risk factor for periodontal diseases and a potential risk factor for endodontic diseases (Tonetti 1998, Duncan & Pitt Ford 2006). The detrimental effect of tobacco use depends on the number of cigarettes a patient currently smokes (Tomar & Asma 2000) as well as the intensity, duration of smoking, and the time since smoking cessation (Dietrich & Hoffmann 2004). Furthermore, the outcome of various dental treatment procedures is less favourable in patients who use tobacco, i.e. smoke cigarettes (Tonetti 1998, García *et al.* 2007). This clinical report describes the step-wise treatment of two periodontal-endodontic lesions in a heavy smoker.

Case

A 53-year-old Caucasian male was referred to the Department of Periodontology, Endodontology and Cariology at the University of Basel, Switzerland for periodontal treatment. The patient was in good general health with fair oral hygiene (Fig. 1). Evaluation of his smoking history revealed that he was a heavy-smoker with a consumption of 20–30 cigarettes per day for 34 years, i.e. approximately 40 pack-years. The maxillary right first molar, the mandibular left second molar, the central incisor, and the mandibular right second molar were missing due to periodontal destruction, i.e. increased mobility and periodontal abscesses. The clinical examination demonstrated increased periodontal



Figure 1 Initial intra-oral view of the patient.



Figure 2 Initial periapical radiographs of the mandibular cuspids and incisors.

probing depths up to 9 mm, furcation involvement, and increased tooth mobility, as well as bleeding on gentle periodontal probing of approximately half of the dentition. Some of the restorations were insufficient. All teeth except the mandibular left lateral incisor and the right central incisor responded to pulp sensibility testing with CO₂. For reliable results, pulp testing was performed lingually on the exposed metal. Radiological examination revealed generalized horizontal bone loss and periapical radiolucency on the lateral and central incisor in question (Fig. 2).

The diagnoses were chronic (smoker) periodontitis and a combined periodontalendodontic lesion on both the left lateral incisor and the right central incisor of the mandible. Prevention of further tooth loss was the main concern of the patient.

Treatment

An individual oral hygiene programme including the use of interdental brushes and the modified Bass technique was initiated. The patient was informed about the impact of his smoking habit on periodontal and endodontic diseases. Smoking cessation was recommended and assistance offered.

The conservative, nonsurgical periodontal treatment – scaling and root planing – was performed on all affected teeth within two appointments, using hand and ultrasonic instruments under local anaesthesia. Both the mandibular left lateral and the right central incisor were excluded from subgingival instrumentation and were only scaled supragingivally. Root canal treatment was initiated on both teeth. Caries was excavated after local anaesthesia, the removal of the fixed partial denture, and placement of rubber dam. The root canals, two in the mandibular right central incisor and one in the mandibular left lateral incisor, were identified using an operating microscope (OPMI® pico; Carl Zeiss AG, Jena, Germany).

The canals were instrumented up to an apical size 40 with NiTi rotary instrumentes (ProTaper[™]; Maillefer, Ballaigues, Switzerland) and an apical stop was prepared using NiTi hand files (Nitiflex[™]; Maillefer). Working length determination was performed using an apex locator (Raypex4; VDW, Munich, Germany) and radiography. Calcium hydroxide slurry was packed into the root canals and the access cavities were sealed with Cavit[™] (3M ESPE, Seefeld, Germany). A chairside fixed partial denture was constructed and fitted.

Twenty-five days later, the root canals were filled with the Thermafil[™] System (Maillefer) and sealer (Apexit[™]; Vivadent, Liechtenstein). The coronal 3 mm of the root fillings were removed and the teeth were restored with composite resin. The radiograph taken after completion of root canal treatment revealed a surplus of filling material on the mandibular right central incisor.



Figure 3 Periapical radiographs obtained 8 months after root canal treatment.

Clinical healing was uneventful. The re-evaluation of the periodontal conditions after 3 months revealed clinically acceptable probing depths, indicating 'closed periodontal pockets' on most of the involved periodontium (Tomasi *et al.* 2006). Reconstructive procedures were initiated, a fixed partial denture was positioned to replace the maxillary right first molar, and restorations were completed using composite materials.

Radiographs revealed signs of osseous tissue regeneration on the mandibular left lateral incisor and on the right central incisor. Radiographs obtained after 8 (Fig. 3) and 14 months (Fig. 4) showed that the appearance of the mandibular left lateral incisor remained unchanged. Probing depths measured from mesio-buccal to mesio-lingual on the mandibular left lateral incisor after 14 months were recorded as 5-5-4-3-2-8 mm and



Figure 4 Periapical radiograph obtained 14 months after root canal treatment.



Figure 5 Follow up of the surgical action: (a) preoperative view, (b) the defects after debridement, (c) inserted bovine bone substitute, (d) covered by a resorbable membrane, (e) primary wound closure with monofil polyethylene sutures, (f) intraoral view after 9 months.

1-2-1-2-2 mm on the right central incisor. Periodontal regenerative treatment was initiated on the left lateral incisor.

The patient was informed about the treatment possibilities, the timeframe and the risks of the treatment in patients with a history of smoking. Regeneration of the defect by guided tissue regeneration (GTR) with a bovine bone substitute and a resorbable membrane was performed (Fig. 5a-f).

After rinsing with chlorhexidine, local anaesthesia was administered labially and lingually. A full-thickness flap was raised from the mesial aspect of the mandibular left cuspid to the distal aspect of the right lateral incisor without making vertical releasing incisions. After thorough debridement, a three-wall bony defect and a mesial fenestration defect were visualized on the left lateral incisor and the right central incisor, respectively. The bony defects were filled with a bovine bone substitute (BioOss, Geistlich, Schlieren, Switzerland) and covered with a resorbable membrane (Bio Gide, Geistlich, Schlieren, Switzerland).

After slight mobilization of the labial flap in a coronal direction, the membrane was completely submerged and the wound was closed using monofil polyethylene sutures (Prolene, Ethicon, Johnson and Johnson, Somerville, NJ, USA). Clindamycin was prescribed for 8 days. The initial healing was uneventful and the sutures were removed 6 days post-surgery.



Figure 6 Intraoral view 9 months after surgical treatment.

Regular follow-ups revealed no visible changes 8 months post-surgery. Therefore, adjustment of the pontic design was initiated in order to achieve an optimal cervical contour as well as an emergence profile of the pontic teeth. Several modifications resulted in a satisfactory outcome. However, the patient opposed to any further changes, such as technological improvement of the prosthesis. After 2 years of treatment, the fixed prosthesis was fitted (Fig. 6). Finally the patient was introduced to a 3-month supportive periodontal therapy programme.

The follow-up revealed a stable condition from a clinical and radiological point of view 32 months after initiation of treatment (Fig. 7). Probing depth measurements from mesiobuccal to mesio-lingual on the mandibular left lateral incisor were recorded as 4-2-2-2-4 mm and 1-1-3-3-2-2 mm on the right central incisor.

Discussion

There is a large body of evidence showing the detrimental effects of tobacco use on the periodontal tissue and on dental implants. In recent years, several studies have been carried out to determine the relationship between tobacco use and endodontic diseases or treatment outcomes, including wound healing after periapical surgery (García *et al.* 2007) and the prevalence of apical periodontitis amongst smokers (Kirkevang & Wenzel 2003, Bergström *et al.* 2004, Segura-Egea *et al.* 2008). The results, however, were inconclusive. A recent review (Duncan & Pitt Ford 2006) summarized the current knowledge in the field and emphasized the need for clinical studies on tobacco use and its association with endodontic diseases and endodontic treatment considerations. Therefore, the observations and treatment suggestions obtained from the combined treatment of two periodontal-endodontic lesions in a heavy smoker may provide some insights for further analysis of the relationship between tobacco use and oral diseases, as well as the treatment options.

Tobacco-induced periodontal destruction is caused by a wide range of effects on the different functions of cells, tissues and organs. Some of these effects are opposed to each other due to the effects of different tobacco constituents. However, when summarizing the properties of the tobacco-induced alterations in metabolism of the vasculature, connective tissue and bone as well as on cell-mediated and humoral immunity, it is very likely that the use of tobacco disrupts the physiological balance between anabolic and catabolic mechanisms due to alterations in the immune system and tissue mechanisms (Palmer *et al.* 2005, Johnson & Guthmiller 2007, Ryder 2007).

A common clinical observation is delayed wound healing after therapeutic interventions (Silverstein 1992). The size of the radiolucencies around the affected roots on both



Figure 7 Periapical radiograph 18 months after surgical treatment and 32 months after root canal treatment.

mandibular incisors decreased after conventional root canal treatment. After an observation period of 14 months, the radiological appearance of the osseous defects seemed unchanged and surgical intervention was initiated. Although it is known that healing of periapical lesions may take up to 4 years (European Society of Endodontology 2006), tobacco use is not considered a modifier of this process. The smoking habit of this patient may have contributed to the delayed healing of the endodontic aspect of the combined lesion, which in turn affected the treatment strategy.

The cleaning and shaping of the root canals were performed meticulously. It is well known that overfilling is more likely to occur when using a thermoplastic gutta-percha obturation technique. Unfortunately, in the present case, root canal filling with Thermafil led to a surplus of filling material on the mandibular right central incisor.

It was taken into account the fact that regenerative procedures in smokers may reveal impaired results. The remaining defect was augmented by GTR using bovine bone substitute and resorbable membrane. This treatment is well established and is often successful despite the fact that several patient-based factors might have an impact (Tseng *et al.* 1996, Needleman *et al.* 2006). Nonetheless, smoking is likely to adversely affect the clinical outcome of regenerative treatment (Trombelli & Scabbia 1997). Recently, a discussion has been instigated on the compensatory use of antibiotics in dental surgical procedures. Decreased gingival inflammation leading to increased wound healing is attributed to the use of a systemic administrated antibiotic (Dastoor *et al.* 2007). In the

present case, Clindamycin was used due to its proven effectiveness in treatment of bony tissues as well as against Gram-negative bacteria.

The patient in the present case was a heavy smoker and was identified as such by the routine questionnaire designed for recording the tobacco use history of patients. Using the motivational interviewing technique, the patient was informed briefly about the pathogenesis of tobacco-related diseases both in general and with special focus on periodontal and endodontic diseases. The likelihood of poor treatment outcomes and delayed wound healing were explained. The patient was advised to stop smoking and help was offered. In spite of these measures, this patient was not willing to give up smoking. However, evidence from a systematic review suggests that counselling conducted by oral health professionals may increase tobacco abstinence rates (Carr & Ebbert 2006). Therefore, the dentists who see this patient regularly may be in a good position to help him change his smoking behaviour (Ramseier *et al.* 2006).

Conclusion

This case report describes the step-wise treatment of two advanced periodontalendodontic lesions in a heavy smoker. Conventional endodontic treatment led to reduced radiolucencies around the affected roots. The remaining osseous defect was augmented using GTR after an extended observation period. The delayed wound healing observed may be attributed to the smoking habit of the patient.

Disclaimer

Whilst this article has been subjected to Editorial review, the opinions expressed, unless specifically indicated, are those of the author. The views expressed do not necessarily represent best practice, or the views of the IEJ Editorial Board, or of its affiliated Specialist Societies.

References

- Armitage GC (2000) Development of a classification system for periodontal diseases and conditions. *Northwest Dentistry* **79**, 31–5.
- Bergström J, Babcan J, Eliasson S (2004) Tobacco smoking and dental periapical conditions. *European Journal of Oral Sciences* **112**, 115–20.
- Carr AB, Ebbert JO (2006) Interventions for tobacco cessation in the dental setting. *Cochrane Database of Systematic Reviews*, CD005084.
- Dastoor SF, Travan S, Neiva RF, Rayburn LA, Giannobile WV, Wang HL (2007) Effect of adjunctive systemic azithromycin with periodontal surgery in the treatment of chronic periodontitis in smokers: a pilot study. *Journal of Periodontology* **78**, 1887–96.
- Dietrich T, Hoffmann K (2004) A comprehensive index for the modeling of smoking history in periodontal research. *Journal of Dental Research* **83**, 859–63.
- Duncan HF, Pitt Ford TR (2006) The potential association between smoking and endodontic disease. International Endodontic Journal **39**, 843–54.
- European Society of Endodontology (2006) Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology. *International Endodontic Journal* **39**, 921–30.
- García B, Penarrocha M, Martí E, Gay-Escodad C, von Arx T (2007) Pain and swelling after periapical surgery related to oral hygiene and smoking. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiolology, and Endodontology* **104**, 271–6.
- John V, Warner NA, Blanchard SB (2004) Periodontal-endodontic interdisciplinary treatment a case report. *The Compendium of Continuing Education in Dentistry* **25**, 601–6.
- Johnson GK, Guthmiller JM (2007) The impact of cigarette smoking on periodontal disease and treatment. *Periodontology 2000* **44**, 178–94.

- Kirkevang LL, Wenzel A (2003) Risk indicators for apical periodontitis. *Community Dentistry and Oral Epidemiology* **31**, 59–67.
- Needleman IG, Worthington HV, Giedrys-Leeper E, Tucker RJ (2006) Guided tissue regeneration for periodontal infra-bony defects. *Cochrane Database of Systematic Reviews*, CD001724.
- Palmer RM, Wilson RF, Hasan AS, Scott DA (2005) Mechanisms of action of environmental factors tobacco smoking. *Journal of Clinical Periodontology* **32** (Suppl. 6), 180–95.
- Ramseier CA, Mattheos N, Needleman I, Watt R, Wickholm S (2006) Consensus report: First European Workshop on Tobacco Use Prevention and Cessation for Oral Health Professionals. *Oral Health and Preventive Dentistry* **4**, 7–18.
- Ryder MI (2007) The influence of smoking on host responses in periodontal infections. *Periodontology* 2000 **43**, 267–77.
- Segura-Egea JJ, Jiménez-Pinzón A, Ríos-Santos JV, Velasco-Ortega E, Cisneros-Cabello R, Poyato-Ferrera MM (2008) High prevalence of apical periodontitis amongst smokers in a sample of Spanish adults. *International Endodontic Journal* **41**, 310–6.
- Silverstein P (1992) Smoking and wound healing. American Journal of Medicine 93, 22S-24S.
- Simon JH, Glick DH, Frank AL (1972) The relationship of endodontic-periodontic lesions. *Journal of Periodontology* **43**, 202–8.
- Solomon C, Chalfin H, Kellert M, Weseley P (1995) The endodontic-periodontal lesion: a rational approach to treatment. *Journal of American Dental Association* **126**, 473–9.
- Tomar SL, Asma S (2000) Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *Journal of Periodontology* **71**, 743–51.
- Tomasi C, Bertelle A, Dellasega E, Wennstrom JL (2006) Full-mouth ultrasonic debridement and risk of disease recurrence: a 1-year follow-up. *Journal of Clinical Periodontology* **33**, 626–31.
- Tonetti MS (1998) Cigarette smoking and periodontal diseases: etiology and management of disease. Annals of Periodontology **3**, 88–101.
- Trombelli L, Scabbia A (1997) Healing response of gingival recession defects following guided tissue regeneration procedures in smokers and non-smokers. *Journal of Clinical Periodontology* **24**, 529–33.
- Tseng CC, Harn WM, Chen YH, Huang CC, Yuan K, Huang PH (1996) A new approach to the treatment of true-combined endodontic-periodontic lesions by the guided tissue regeneration technique. *Journal of Endodontics* **22**, 693–6.
- Vakalis SV, Whitworth JM, Ellwood RP, Preshaw PM (2005) A pilot study of treatment of periodontalendodontic lesions. *International Dental Journal* 55, 313–8.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.