

UiO : **University of Oslo**

**Faculty of dentistry
Department of Endodontics**

Postgraduate Program in
Endodontics

Case Book
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Endodontic Treatment Guidelines

Treatment of teeth without apical periodontitis

Aseptical enlargement, cleaning and obturation of the pulp space, preferably in a single visit.

Preoperative radiograph

Anaesthesia

Removal of plaque, caries and leaking fillings. Tooth build-up if required for isolation.

Application of rubber dam. Disinfection of the working area with 0.5% chlorhexidine in 70% ethanol.

Access cavity preparation is conservative, but aiming for straight line access to orifices, and removal of all coronal pulp tissue remnants.

Localization of canal orifices. Work field is again disinfected by 0,5 % chlorhexidine. (Rubber dam may sometimes be placed after location of canals.)

Measurement of working length, using electronic apex locator (EAL) and working length (WL) radiograph. WL established 0.5-1mm short of the major apical constriction.

Instrumentation to desired apical length and size

- Frequent irrigation with 1% sodium hypochlorite (NaOCl)*
- Final irrigation with 17% ethylenediaminetetraacetic acid (EDTA)

Drying of the canals with paper points

Uncontrollable bleeding, iatrogenic perforations or limited time may necessitate a second visit. In these cases, the canal is dressed and sealed at this point, according to guidelines for cases with apical periodontitis.

Adaptation of master point

Master point radiograph

Root filling:

Obturation techniques:

Lateral compaction

Warm vertical compaction

Sealers:

AH Plus®

Endosequence®/TotalFill®BC sealer™

Real Seal®

Core materials:

Gutta-percha

Resilon

Removal of core material and sealer from the pulp chamber. Temporary IRM top filling with IRM plug in canal orifices. Removal of rubber dam.

(A permanent composite restoration is placed in some cases.)

Final radiograph

Treatment of teeth with apical periodontitis

Aseptical enlargement, disinfection and medication of the pulp space for 7 days or more. Subsequently, the canal space is again cleaned and disinfected before obturation.

FIRST VISIT

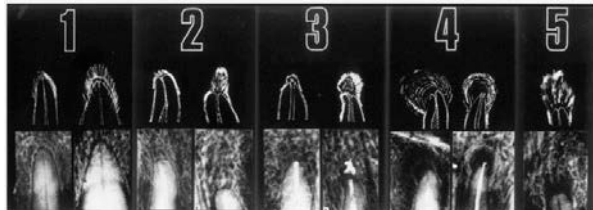
Same as for uninfected cases, with some additional measures:

- Ultrasonic agitation of irrigants (PUI); irrigation tip on a low power setting, 20-60 s/canal.
- Post instrumentation, the canal is dressed with calcium hydroxide in an aqueous solution. The pulp chamber is cleansed and routinely covered by a sandwich Cavit G + IRM temporary filling. On indication, a temporary composite filling may be placed.
- For retreatment cases: Bulk of old obturation material is removed by Gates-Glidden burs and rotary NiTi files on 900 rpm. Remaining material is removed by handfiles and ultrasonic preparation. A solvent (chloroform) is added if necessary. After instrumentation and final 17% EDTA irrigation, in some cases 2% chlorhexidine digluconate (CHX) is used before placement of calcium hydroxide with a lentulo spiral.
- Removal of rubber dam.

SECOND VISIT (1-3 WEEKS LATER)

If symptoms have resolved, root canals are permanently obturated. (See description of treatment of teeth without apical periodontitis.)

The periapical index (PAI), is used for radiographic evaluation (Ørstavik et al 1986)[1].



Reference set of radiographs with corresponding line drawings and their associated PAI scores.

Emergency treatment

ACUTE PULPITIS

Pain relief without impairment of future prognosis - pulpotomy.

Preoperative radiograph

Anaesthesia

Application of rubber dam. Disinfection of the working area with 0.5% chlorhexidine in 70% ethanol.

Removal of caries. Access cavity preparation, removal of inflamed coronal pulp tissue. NaOCl irrigation.

Cotton pellet with eugenol is placed in the pulp chamber and sealed by IRM.

Standard analgesic on indication; ibuprofen, 400 mg/4-6 times a day.

A full pulpectomy is performed within limited time.

ACUTE APICAL PERIODONTITIS

Infection control and pain relief - elimination of bacteria, bacterial nutrients/substrates, and evacuation of pus

A full first visit treatment is instigated; instrumentation and antibacterial irrigation of canals, followed by placement of intracanal medicament. Drainage may be achieved from the root canals. If a fluctuant abscess is present, peripheral incision and drainage of purulent material is attempted.

Empirical antibiotic treatment is initiated on indication of fast evolving, disseminating or systemic infection:

Phenoxymethylpenicillin 1 g x 4 for 5 days.
For penicillin-allergic patients; clindamycin 300 mg x 4-5 for 5 days.

Effectiveness of antibiotic treatment is monitored.

Standard analgesic on indication; ibuprofen, 400 mg/4-6 times a day.

* The Department of Endodontics implemented 0.5% chlorhexidine as a routine irrigant, replacing NaOCl, for all treatment initiated from February through June, in 2013 and 2014

Endodontic surgery

PREOPERATIVE

Treatment planning in a separate visit. Clinical examination. Soft tissue assessment. Photographic documentation of preoperative status.

Periapical radiographs. Patient may be referred for orthopantomogram (OPG), cone beam computed tomography (CBCT), or other modalities.

Information about the surgical procedure, potential risk factors, expectations for the postoperative phase and potential considerations regarding soft-tissue healing.

Oral hygiene control and information. Sub- and supragingival debridement.

If the patient comply with the treatment plan, an appointment for surgery is made.

SURGERY

Relevant radiographs are mounted on screen.

Preoperative information is repeated.

Anesthesia with vasoconstrictor is administered 15-20 minutes before the incision to optimize haemostasis.

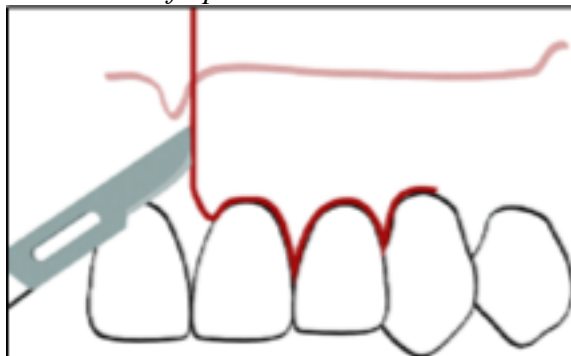
1 minute Corsodyl® mouth rinse (chlorhexidine digluconate 2mg/ml)

Aseptic draping of patient, and non-sterile equipment. Operator and one assistant is in complete sterile attire. A circulating assistant aids procedures involving contact with non-sterile surfaces. All instruments in direct or indirect contact with the surgical field is sterile.

Use of the operating microscope is aimed for during surgery, magnification adjusted according to need for overview.

Incision:

Horizontal incision extending one to several teeth mesial and distal of the involved tooth and one vertical releasing incision, usually at the mesial end of the flap. A microsurgical blade #69 is used for most incisions.

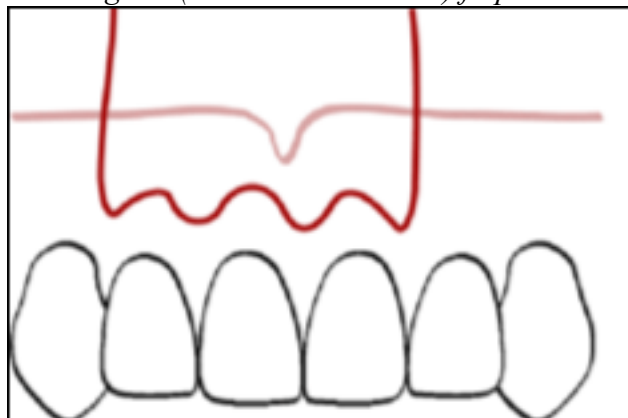
Flap designs*Intrasulcular flap*

(Velvart 2005)[2]

Horizontal incision extending one to several teeth mesial and distal of the involved tooth. Vertical releasing incision, usually at the mesial end of the flap

If limited access, conversion to a rectangular flap by additional release at opposite end of the horizontal incision.

Mainly used in posterior apical surgery, cervical resorptions, perforations, and resections of short roots.

Submarginal (Ochsenbein-Luebke) flap

(Velvart 2005)[2]

Marginal gingiva is untouched, restoration margins will not be exposed.

Similar to the rectangular flap, but the scalloped horizontal incision, roughly parallel to marginal gingival contour, is placed within attached gingiva.

A broad (minimum of 3 mm) attached gingiva is required.

Fear of even small recessions is the driving force for considering the submarginal flap

Elevation

A fullmucosal-periosteal flap is raised by a periosteal elevator. If the flap is strained, release may be extended.

Retraction

The flap is held away from the surgical site, providing maximum access and visibility. Care is taken not to overly traumatize soft tissues. To secure retractor anchorage at a safe distance from the mental nerve, a 15 mm groove in the corticalis may be made by round burs. All bone removal and root-end preparation is under copious sterile saline irrigation.

Osseous entry

Removal of cortical and cancellous bone to gain direct access to the apical portion. Round surgical burs in a low-speed handpiece. Conservative osteotomy is aimed for. A minimum of 3-4 mm diameters is necessary to allow overview and access for microsurgical instruments. The osteotomy may be larger, dependent on root location, lesion size, and overview in the bone crypt.

Surgical curettage

Removal of all pathological tissue, foreign bodies, and root and bone particles from the periradicular area.

Biopsy

Any soft tissue lesion removed during the procedure is prepared in 4% formaldehyde and submitted for histological examination.

Microbiological sample

With paper point directly in the periapical sample, placed in prerduced anaerobic transportmedium, or periapical tissue placed in 4% formaldehyde for scanning electron microscopy (on indication).

Root-end resection

Routinely, 3 mm of the root-end is resected perpendicular to the root axis, with a cylindrical steel bur in a low-speed handpiece. Resected root surface is inspected in the operating microscope after methylene blue staining to identify PDL, root canals, possible isthmi and fracture lines. Inspection is aided by micromirrors placed at 45° angle to the root surface.

Ultrasonic root-end preparation

At least 3 mm retrograde preparation of canals, including isthmi if present. The preparation is performed with surgical ultrasonic tips aligned with the root axis. Visually inspection of the preparation. Control radiograph.

Hemorrhage control

Haemostasis of the the bone crypt is achieved by packing of sterile gauze or cotton pellets soaked in ferric sulfate ($\text{Fe}_2(\text{SO}_4)_3$) or adrenaline. More vasoconstrictor-containing anaesthesia may be administered.

Root-end filling

The retrograde cavity is cleaned and dried with paperpoints. MTA or EndoSequence®/TotalFill® RRM™ root-end filling. MTA is applied with aid of MAP system® or the MTA pellet forming block. Root-end filling is condensed with pluggers. Control radiograph.

Wound toilette

Removal of root-end filling surplus, cotton pellets, gauze and ferric sulfate. Blood cloths between bone and soft tissue removed with moist gauze. Flap is aligned and compressed lightly with moist gauze for a few minutes before suturing to reduce subperiosteal haematoma and relieve stress on suture lines.

Sutures

Simple, interrupted and interdental sutures is the standard. Pseudomonofilamentuous 4-0 or 5-0 sutures, 3/8C 19 and 12 mm needles (Supramid®). Light compression is again applied to the flap after suturing. Control radiograph, at this stage or on the first postoperative control.

Postsurgical care

Haemostasis is evaluated. Administration of an ice-pack for 10-20 minutes.

Information of suspected swelling/haematoma, possible pain and

management, instructed to apply ice-pack intermittenly on surgical day, intake soft foods, avoid strainous excercise the first 24 hour, thorough general oral hygiene/careful in the surgical area.

Unless contraindicated for some reason, the patient is instructed to take 400 mg ibuprofen every 4 to 6 hours for the first 48 hours. Corsodyl® mouth rinse twice daily until suture removal.

Postoperative information is given orally and in writing.

Postoperative control

Postoperative control with inspection of soft tissue healing, and suture removal 3-7 days after surgery.

Anaesthesia in use

Xylocain® Dental adrenalin (lidocaine hydrochloride 20 mg/ml + adrenaline 12,5 µg/ml)

Septocaine® (articaine hydrochloride 40 mg/ml + adrenaline 5 µg/ml)

Septocaine® Forte (articaine hydrochloride 40 mg/ml + adrenaline 10 µg/ml)

Carbocain® Dental (mepivacain hydrochlorid 30 mg/ml)

Citanest® Dental Octapressin (prilocain. hydrochlorid. 30 mg/ml + felypressin. 0,54 µg/ml)

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Case 1

Conservative treatment of a maxillary right second premolar with a bifid root.

A 34-year-old male was referred to the Department of Endodontics from a general practitioner in private practice, for endodontic treatment of the maxillary right second premolar, 15.



Fig. 1.1 - Frontal view 21.05.2013

Chief complaint

The patient presented for completion of treatment of 15. He had experienced pulpitic pain, which ceased after an emergency pulpotomy.

Medical history

Non-contributory.

Dental history

Some caries experience/posterior fillings.

Clinical findings

Soft tissues

Localized signs of gingivitis.

Dental

Disto-occlusal temporary filling in 15.

Clinical tests

Tooth	16	15	13
Cold	no	no	yes
EPT 0-80	-	59	58
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	3mm	3mm	4mm

Radiographic findings

Tooth 16

- root filled

Tooth 15

- disto-occlusal filling, extends into coronal pulp
- two PDL spaces were discernable in apical 5 mm - indicative of bifid root
- root canal is clearly visible
- intact lamina dura

Attachment apparatus

- normal marginal bone levels



Fig. 1.2 - Periapical radiograph 21.05.2013

Diagnosis

Pulpal K04.1 - Pulp necrosis (partial)

Periapical Normal

Periodontal Normal

Responses to sensibility tests were contradictory and 15 was accessed 2 months earlier. The preoperative pulpal diagnosis was set to partial necrosis; positive EPT indicated some vital tissue might remain.

Treatment plan

- Conservative endodontic treatment

Problem list

- Possible bifid root

Assessment of root canal anatomy in the course of the treatment.

Treatment progress

21.05.2013

- Examination and treatment planning
- Instrumentation
- Obturation

Treatment

Anaesthesia: Infiltration, Septocaine 1,7 ml.

Rubber dam/disinfection.

Access through the temporary restoration. A cotton pellet covered a figure 8 shaped canal. Vital pulp tissue. The apical area was reached from both buccal and lingual sides with small handfiles. Working length determined by radiograph and apex locator. Instruments from both sides joined in the same apical canal. The two possible apical PDL spaces also seen on Fig. 1.3 *left*, cast doubt if this was a correct reflection of the apical anatomy. It was decided to explore canal anatomy further after instrumentation.

A size 030 K-file reached the full working length, and the canal was instrumented with a Reciproc® R50 file from both buccal and lingual side.



Fig. 1.3 - Working length radiograph, disto-eccentric (*left*) and ortoradial (*right*) 21.05.2013

After instrumentation, bleeding was seen on the palatal canal wall, 4 mm short of full working length. A canal was found and negotiated by countouring the apical 2-4 mm of small handfiles (#008-020) with a plier. The working length was controlled with apex locator. Instrumentation of the palatal canal was completed with NiTi handfiles. Irrigation, dried with paper points, masterpoint radiograph. Obturation with gutta-percha and AH plus with a cold lateral condensation technique.

Mechanical

- Burs, K-files, Reciproc R50 file, NiTi handfiles
 - B 50/06 - 19 mm
 - P 040/.02 - 19 mm

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus

Temporary filling

- IRM, 3 mm plug in orifice.

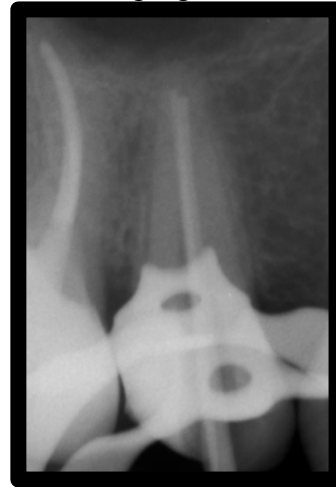


Fig. 1.4 - Masterpoint radiograph 21.05.2013



Fig. 1.5 - Final radiograph 21.05.2013

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

Follow-up



Fig. 1.6 - Control 23.04.2014

No symptoms. Sound apical conditions. The patient was advised to get a permanent coronal restoration.

Evaluation

Treatment was completed without complications. The treatment result appeared satisfactory.

Discussion

As carious lesions progress towards the pulp, increasing histological signs of pulpal inflammation can be seen[1]. According to Reeves[2], a distance between invading bacteria and pulp of 1,1 mm or more, may yield negligible inflammatory response. A bacterial front 0,5 mm from the pulp produced significant inflammation. Different studies find different decisive distances where dentinal infection affect pulpal tissues. This may be coincidental, as a very large amount of histological sections from different teeth would be necessary to exact examine the three-dimensional relationship of dentine invasion and pulpal inflammation[1].

Neutrophil granulocytes are the first cells of the innate immune system recruited to a site of infection. Neutrophil chemoattractants include both exogenous bacterial products and endogenous substances released during the inflammatory response[3, 4]. Pulp reactions range from production of irregular dentine (seen related to insults as superficial as enamel caries) to increasing infiltration of inflammatory cells, and microabscess formation[1, 2, 5].

Langeland observed that with bacterial invasion of pulp periphery, or even of the entire coronal pulp, the radicular pulp might be vital and free from inflammatory cells[1]. The former 'self-strangulation theory' of pulpal inflammation would oppose to this finding. The theory was based on the notion that increased interstitial pressure, resulting from increased capillary filtration during pulpal inflammation, led to compression of pulpal venules. As a result, the capillary pressure would increase further, inducing even more capillary filtration. This vicious cycle was thought to lead to stasis of pulpal blood flow and total pulp necrosis[6]. The basis for the theory was hypothetical, applying knowledge of inflammatory changes in other tissues to the low-compliance pulp space. Studies have proved that pulpal tissue has mechanisms to break the cycle: Increased interstitial pressure results in reduced local filtration, and net absorption in adjacent capillaries. There is also likely an increased

lymphatic drainage, although the presence of lymphatic vessels in the pulp itself is controversial[7, 8]. Arteriovenous anastomosis shunts permit regional control of pulpal blood flow via direct shunting of blood from arterioles to venules[9]. Studies of rat pulps demonstrated that increased tissue pressure in inflamed tissue was localized. Pressures could be normal 1-2 mm from an inflamed area [10]. This agrees with histopathological findings, with sometimes well-defined borders of inflammation, and capacity of healing even after severe reactions, like experimentally induced microabscesses[11, 12].

Although hypersensitivity, spontaneous and lingering pain may occur in irreversible pulpitis, both histological and clinical evidence prove that pulpal inflammation may progress without symptoms [1, 3, 13]. Both in pathological and healthy states, pain thresholds and pain signalling of the dental pulp in some aspects differs from other tissues. The nociceptive (myelinated) A δ -fibres and (unmyelinated) C-fibres transmit pain signals from most peripheral tissues. The lowered threshold for pain elicitation during inflammation is believed to be caused by sensitization of nociceptive fibres by inflammatory mediators[14, 15].

However, pain appears to be the primary sensation evoked on any stimulation of the pulp, even in absence of inflammation. This is evident when healthy exposed dentine is provoked by weak stimuli like air-puffs or probing. The fact that pain is the primary sensation elicited from the pulp have been explained by a high frequency of A δ -and C-fibres in the inner dentine and pulp itself[14]. In healthy tissues, pain signals are not normally generated by these fibres after low-grade stimuli. Fried et al. suggests that in the dental pulp, activation of low-threshold mechanoreceptors (A β -fibres) might be capable of inducing pain. This may be due to activation of pain signalling second-order neurons in the trigeminal nucleus caudalis, relying the original mechanical stimulus as a nociceptive input to higher brain centres[14]. The reason why pulpitis frequently progress without symptoms is not clear. It has been proposed that differences in bacterial virulence, pace of inflammation progression, or central mechanisms might play a role[13].

References

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Case 2

Conservative treatment of a mandibular right second premolar with 3 canals.

A 26-year-old male was referred to the Department of Endodontics from a general practitioner at the Dental Care Unit of SiO (a student welfare organisation), for root canal treatment of the mandibular right second premolar, 45.



Fig. 2.1 - Frontal view 24.04.2013

Chief complaint

The patient presented for completion of treatment of 45.

Medical history

Non-contributory.

Dental history

The patient had presented with radiating pain from 45 three months earlier. A canal was negotiated, but radiographic findings indicated more than one canal, and a referral was made. The patient was without symptoms after treatment initiation.

Clinical findings

Soft tissues

Healthy pink soft tissues

Dental

Composite fillings in molars. Disto-occlusal temporary filling in 45.

Clinical tests

Tooth	46	45	44
EPT 0-80	18	80	26
Cold	yes	no	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	wnl	wnl	wnl

Radiographic findings

Tooth 46

- MOD composite

Tooth 45

- temporary coronal restoration with voids
- radiopaque intracanal medication in coronal canal

- in the apical half of the root, a canal is seen deviating distally from the central axis, hence one or more additional canals were suspected.
- possibly bifid apex, mesially a longitudinal radiolucency extends from the apex towards the crown, believed to represent a PDL space or a groove on the surface of the apical half of the root.
- normal PDL space/lamina dura

Tooth 44

- normal

Attachment apparatus

- normal



Fig. 2.2 - Periapical radiograph 24.04.2013

Diagnosis

Pulpal K04.1 - Pulp necrosis
Periapical Normal
Periodontal Normal
PAI 1

Treatment plan

- Conservative endodontic treatment

Problem list

- Locate and negotiate apical canals

The preoperative radiograph suggested two or more apical canals, possibly calcified as only one was visible. It was planned to search for the canals in the operating microscope. Access could be achieved by LN/Gates-Glidden burs or ultrasonic preparation.

Treatment progress

24.04.2013

- Examination and treatment planning
- Canals located

02.05.2013

- Instrumentation
- Obturation

Treatment - first visit

Anaesthesia: Inferior alveolar nerve block: Septocaine 1,7 ml.

Rubber dam/disinfection.

Removed the temporary filling and the intracanal dressing. Two orifices were seen in the mid-root section of the canal, one to the buccal, and one to the lingual aspect. A third, suspected separate, canal was also negotiated. It was located close to the mesial canal, but at a distal and slightly lingual position, they will be referred to as MB and DB. Working lengths were determined by apex locator and a radiograph (25 mm with #015 handfiles). Instrumentation was not completed as the patient had to leave. The coronal canal was filled with calcium hydroxide and covered by Cavit G/IRM.

Mechanical

- Burs, K-files, Hedström files

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G/IRM



Fig. 2.3 - Working length radiograph 24.04.2013

Treatment - second visit

Anaesthesia: Inferior alveolar nerve block: Septocaine 1,7 ml.

Rubber dam/disinfection.

The overview over the distobuccal canal was refined by coronal flaring with Gates-Glidden burs. The canals were instrumented by handfiles, and the distribution of the canals was controlled by shifted radiographs: Fig. 2.4.

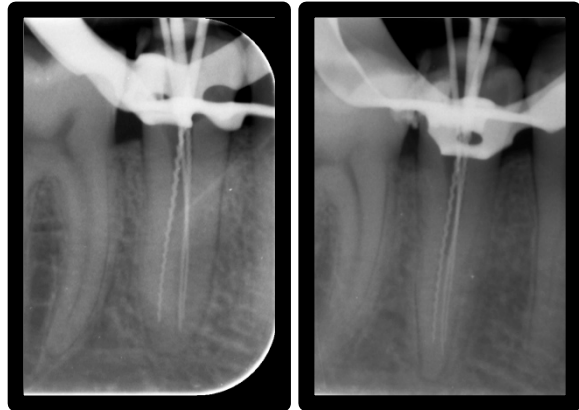


Fig. 2.4 - Control radiographs, disto-angulated *left*, and orthoradial *right* 21.05.2013

Irrigation, dried with paper points. Masterpoint radiograph.

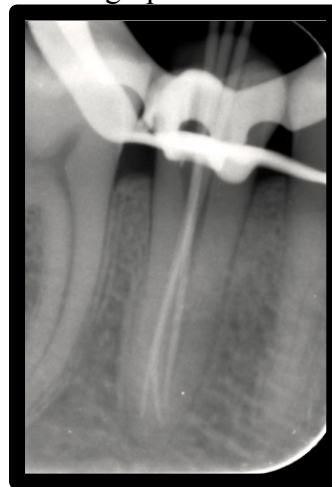


Fig. 2.5 - Masterpoint radiograph 21.05.2013

Obturation with gutta-percha and AH plus, cold lateral condensation technique. Control radiograph. The gutta-percha in the orifice was compacted; sealer excess removed from the access cavity, and an IRM plug was placed.



Fig. 2.6 - Control obturation 21.05.2013



Fig. 2.7 - Final radiograph 21.05.2013

Mechanical

- Burs, K-files, Hedström files, NiTi handfiles
- All canals: 035/.02 - 25 mm

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Obturation

- Gutta-percha and AH Plus®
- 3 mm IRM plug in canal orifice.

Temporary filling

- IRM.

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

Evaluation

Treatment was completed without complications, and the technical result appeared satisfactory. The patient moved, and was not available for follow-up.

Discussion

The anatomy of this mandibular second premolar was unusual. In his extensive study of root canal anatomy, Vertucci found 97,5 of

mandibular second premolars to have a single canal[1]. A type V configuration (one canal leaves the pulp chamber and divides short of the apex into two separate and distinct canals with separate apical foramina) was seen in 2,5%. Although not common, variations like 3-5 canals, and 2 or 3 separate apical roots is identified by others[2]. Investigations have demonstrated less variation of root canal anatomy of mandibular premolars in Caucasians than individuals of other ethnic origins[3, 4].

This case was treated without application of an intracanal medication after instrumentation of the apical canals. There is wide agreement that vital endodontic treatment preferably is performed in a single visit. In necrotic pulps without evidence of periapical inflammation, the preconditions and degree of infection is more uncertain[5].

A wide range of properties have been attributed to calcium hydroxide; antimicrobial activity, capacity to prevent postoperative sensitivity, stimulation of odontoblast differentiation and dental hard tissue formation, tissue-dissolving ability and inhibition of tooth resorption. This caused extensive use of Ca(OH)₂ in restorative dentistry and endodontics; in cavity bases and liners; as a pulp capping agent; for coverage of the pulp wounds at pulpotomies; for apexification/apexogenesis; for treatment of resorptions and as an antimicrobial agent in the treatment of infected canals and apical periodontitis. Today it is known that the beneficial effect of calcium hydroxide is largely because of its high pH and antimicrobial effect, this allows healing to take place in absence of infection[6-8]. The antimicrobial effect of Ca(OH)₂ is dependent on release of hydroxyl ions in aqueous solutions. This may cause protein denaturation, damage to bacterial cytoplasmic membranes, and splitting of DNA strands. Dentine effectively buffers the activity of calcium hydroxide, and the antimicrobial effect is limited in dentine tubuli, ramifications and isthmuses[8]. *E. faecalis* and *Candida* are known to be relatively tolerant to a high pH, and somewhat resistant to calcium hydroxide[6].

After establishing the infectious aetiology of apical periodontitis, effort was taken to investigate the antimicrobial efficiency of endodontic treatment. Byström and Sundqvist examined the effects of instrumentation, antibacterial irrigants and dressings in a series of studies of teeth with periapical lesions in the 1980s. Mere instrumentation with saline produced 100 to 1000 folds decrease in bacterial counts, although all canals still harboured bacteria post initial instrumentation. Left without an intracanal dressing, the bacteria could multiply to close to initial levels between visits[9]. Saline replaced by 0,5% sodium hypochlorite increased the final proportion of bacteria-free samples, and a further increase was seen after addition of EDTA to the irrigation regimen[10, 11]. In another study, calcium hydroxide was used as a intracanal dressing for 4 weeks, and here living bacteria was found in only 1 of 35 canals. For teeth without a dressing, half of the canals produced positive cultures[12]. They concluded that treatment of infected canals could be completed at the second visit when a Ca(OH)₂ medication had been used. In 1991, Sjögren evaluated the effect of calcium hydroxide placed for 10 minutes or 7 days. He found 10 minutes to be ineffective. All 18 canals dressed for 7 days produced negative cultures, both immediately after removal of the dressing, and after 1-5 weeks subsequent sealing without dressing[13].

Other researchers achieved a reduction, but not total elimination of positive cultures after Ca(OH)₂ dressings: Ørstavik, also in 1991, recovered 35% positive samples after reaming of canal walls following 1 week calcium hydroxide medication[14]. In contrast, Peters et al. found proportion of positive cultures increasing after 4 week calcium hydroxide medication[15]. Thus, the ability of calcium hydroxide to predictably produce bacteria free canals was controversial. In a 2004 analysis of studies on antibacterial effectiveness of Ca(OH)₂, Law and Messer endorsed the use of it, but stressed that the main bacterial reduction was seen after instrumentation[16].

Several studies have examined healing of periapical lesions after single vs. multiple visit treatment. Systematic reviews did not find differences in healing rates for single- and multiple-visit endodontic treatment[17,

18]. Su et al. included only studies on necrotic teeth, with or without periapical lesions. Frequency of post-operative pain is often mentioned in this regard, and interestingly, Su found that patients experienced less post-obturation pain after single-visit treatment [18].

In vitro studies have indicated that root canal sealers may disinfect dentinal tubuli more effectively than calcium hydroxide[19]. Thus, when instrumentation is deemed satisfactory, placement of a root filling may be as effective in eliminating residual organisms as a calcium hydroxide dressing. The root filling may entomb bacteria in dentinal tubules, lateral canals, apical ramifications and isthmuses, and access to nutrition and space for multiplication will be restricted[6, 20].

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Case 3

Conservative treatment of a mandibular right first molar with cervical resorption.

46-year-old male. On a CBCT examination a progressing external cervical resorption was diagnosed in a mandibular right first molar, 46.



Fig. 3.1 - Frontal view 12.02.2014

Chief complaint

The patient attended controls after treatment of multiple cervical resorptions from 2007 to 2010. He wished to undergo treatment of a suspected new resorption defect on 46. No symptoms.

Medical history

Non-contributory.

Dental history

Extraction of four premolars and orthodontic treatment in adolescence.

The patient was referred to the Department of Endodontics in 2007, for treatment of cervical resorptions of 11 and 16. The lesion on 16 was too advanced to benefit from treatment. Conservative and/or surgical resorption repair was performed on 13, 11, 21 and 33. After 2010, progression in 16 was limited.

Clinical findings

Soft tissues

Normal.

Dental

Anterior attrition.

Discolorations.

Composite fillings in molars.

Clinical tests

Tooth	47	46	45
Cold	yes	yes	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	4mm	wnl

Increased periodontal pocket depth and bleeding on probing was found on the distobuccal aspect of 46. Other clinical

indications of the resorption were not seen. The distobuccal shadow visible on Fig. 3.2, was caused by external discoloration.



Fig. 3.2 - Detail 46 - 12.02.2014

Radiographic findings

Tooth 16

- mesial cervical radiolucency, 3 mm diameter

Tooth 47

- irregular radiolucency overlapping the pulp chamber mesially 4 mm diam
- normal periapical findings

Tooth 46

- coronal radiopaque filling
- calcified pulp cavity/pulp stone
- indistinct centred irregular cervical radiolucency, overlapping the floor of pulp cavity, 2x3 mm (Fig. 3.4)
- normal periapical findings (intact lamina dura was seen immediate to the apical root surface of the distal root).

Tooth 45

- coronal radiolucency/filling
- normal periapical findings

Attachment apparatus

- normal



Fig. 3.3 - Periapical radiograph from 2008



Fig. 3.4 - After 5 years progression of resorption defect 18.09.2013



Fig. 3.5 - Left side bite wing 18.09.2013

The patient was referred for a CBCT examination for investigation of 47 and 46. Selected CBCT sections Fig 3.6-7 (imaging by M. Kristensen and U.H. Riis, Department of Maxillofacial Radiology)

On CBCT images, a lesion was found in the buccal cervical area of 46. The largest extension was below marginal bone level, and towards the distal aspect of the crown. Perforation, or a close relation to the pulp space, was regarded likely. The CBCT revealed that the intrapulpal radiopacity was presumably a pulp stone, not continuous with the pulp chamber walls.

A lesion was not confirmed in 47.

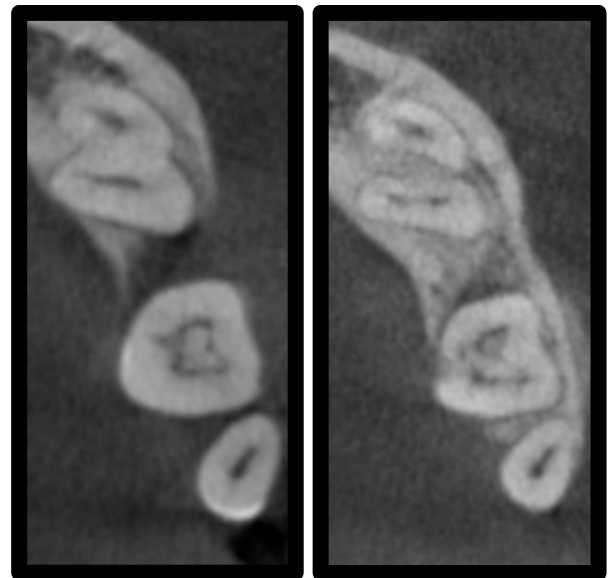


Fig. 3.6 - CBCT mandible, axial view, the *left* is more coronal 04.11.2013



Fig. 3.7 - CBCT coronal view 46 - 04.11.2013

Diagnosis

Resorption K03.38 Cervical resorption, Heithersay class 2

Pulpal K04.2 Pulpal stone

Periapical Normal

Periodontal Normal

PAI 1

Treatment plan

- Conservative endodontic treatment
- Conservative perforation repair

Treatment alternatives

- Accessing resorption from the buccal side

Pulp involvement was suspected during perforation repair. As the bulk of the lesion was located below marginal bone level, it was chosen to approach the defect from the pulpal side. Access from the buccal side would necessitate removal of marginal bone, and considerable amounts of dentine.

Problem list

- Pulp stone/location of pulp chamber floor
- Access and overview of the entire resorption defect
- Risk of perforating buccal root surface

Presence of a pulp stone might increase risk of missing canal orifices or perforating during access cavity preparation. For maximum overview, it was decided to access the pulp and instrument the canals before engaging the resorption.

Treatment progress

12.02.2014

- Instrumentation
- Removal of resorption tissue
- Calcium hydroxide dressing

26.02.2014

- Resorption repair

05.03.2014

- Obturation
- Permanent composite restoration

Treatment - first visit

Anaesthesia: Inferior alveolar nerve block: Citanest Octapressin 2x1.8 ml (patient preferred anaesthetic without adrenaline). Rubber dam/disinfection. Access cavity preparation. Removal of the pulp stone by round burs and ultrasound.

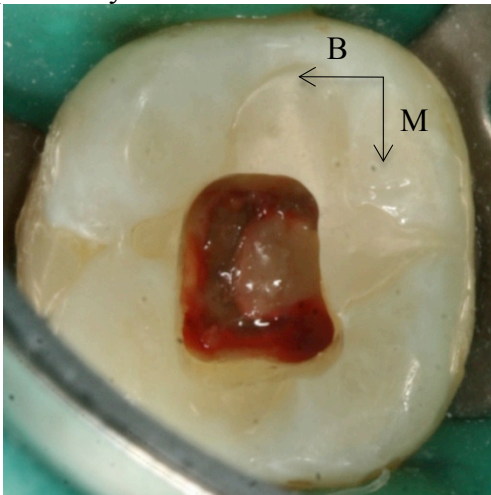


Fig. 3.8 - Pulp stone adhered to the lingual pulp chamber wall 12.02.2014

Bleeding pulpal tissue in all canals. In the buccal periphery of the pulp cavity floor, a sparse bleeding was seen, suspected to originate in the resorption lacuna.

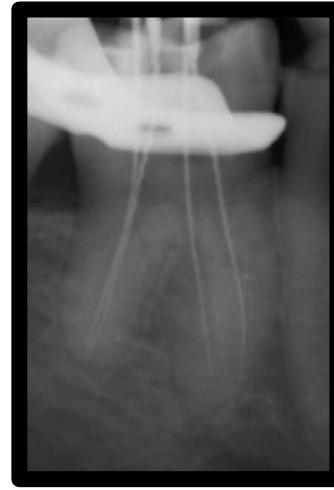


Fig. 3.9 - Working length radiograph 12.02.2014

The working length was determined by EAL/radiograph (18 mm in mesial canals, 16 mm in distal canals). Hand instrumentation to #020, finished with BioRaCe rotary files.

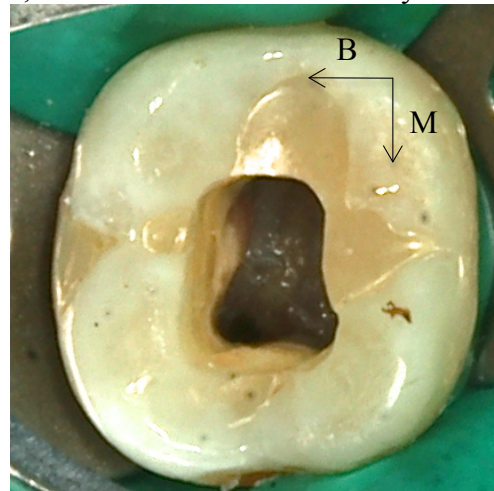


Fig. 3.10 - After instrumentation 12.02.2014

Some dentine covering the buccal side of the distal canal was removed after instrumentation, and bleeding from the resorption tissue resumed.

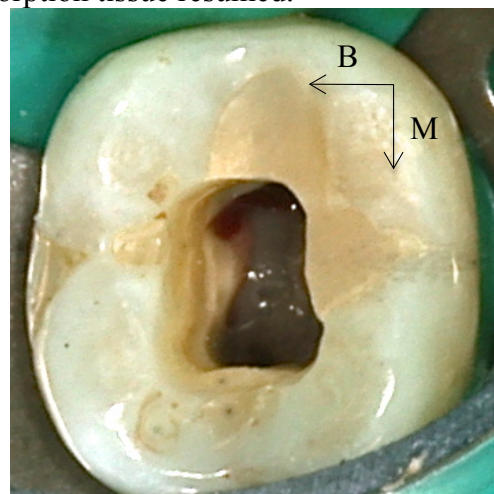


Fig. 3.11 - Bleeding resorption tissue near distal canal orifice 12.02.2014

The resorption tissue was removed by round burs and the access cavity enlarged to the buccal side.

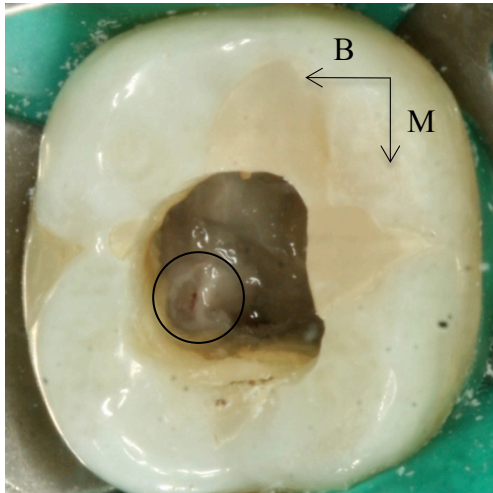


Fig. 3.12 - Resorption canals seen on the base of lesion 12.02.2014

All bleeding tissue was removed by LN burs. Passive ultrasonic irrigation. A calcium hydroxide dressing was placed in the canals and the resorption defect.

Mechanical

- Burs, ultrasonic tips, K-files, Hedström files, BioRaCe files
- MB 040/.04 - 19 mm
- ML 040/.04 - 17,7 mm
- DB 060/.04 - 15,5 mm
- DI 060/.04 - 15,5 mm

Chemical

- 10 ml 0,5% CHX% / 5 ml 17% EDTA
- PUI; NaOCl,

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms.

Anaesthesia. Rubber dam/disinfection
Removed temporary filling. A little more osteoid tissue was seen and removed. 1 mm diameter perforation of submarginal buccal dentine. The resorption defect was repaired with Biodentine, covered by a cotton pellet and a temporary filling.

Mechanical

- Burs, ultrasonic tips.

Chemical

- 10 ml 0,5% CHX% / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - third visit

No symptoms.

Anaesthesia. Rubber dam/disinfection

Removed temporary filling. Biodentine was set. Removed calcium hydroxide dressing. Dried canals. Masterpoint radiograph and obturation by cold lateral compaction of gutta-percha. Permanent composite restoration with cuspal coverage.

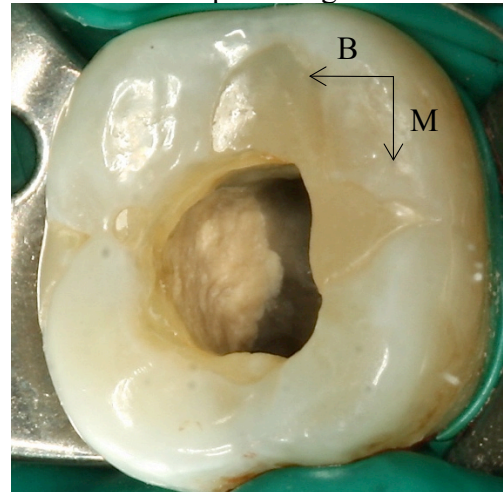


Fig. 3.13 - Set Biodentine 05.03.2014

Mechanical

- Burs, NiTi handfiles

Chemical

- 10 ml 0,5% CHX% / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus
IRM plugs, 3 mm in orifices.

Permanent composite restoration

- Total Etch, 37% phosphoric acid
- Adper™ Scotchbond™ M-P Adhesive
- Filtek™ Supreme XTE: A2B/D2B



Fig. 3.14 - Masterpoint radiograph 05.03.2014



Fig. 3.15 - Final radiograph 05.03.2014



Fig. 3.16 - Permanent restoration 05.03.2014

Prognosis

- Endodontic - favourable
- Tooth - favourable - questionable
- Restoration - favourable

The resorptive defect undermined the buccal cusp, and there is likely a risk of fracture. The lesion perforated the buccal root surface. Perforation was subcrestal, seemingly without contamination, and repair was uncomplicated. There is a risk of missing resorptive tissue during treatment of cervical resorptions, and 46 will be monitored in future controls.

Evaluation

The treatment was carried out according to the plan and, except for the perforation, it was uncomplicated. The access and overview of the defect seemed sufficient, and the approach allowed a relatively conservative treatment.

Discussion

Resorptive conditions result from physiological or pathological processes, and cause loss of dentine, cementum or bone. The process of tooth resorption is thought to be very similar to that of bone resorption, and involves interaction among inflammatory cells, resorbing cells, and hard tissue structures[1]. Immunity of dental hard tissues to osteoclastic cell activity at physiologic

conditions, is likely related to presence of intact cementoblast and odontoblast layers[2]. Root resorptions may be classified according to the site of origin, i.e. external or internal, with subsets[3]. A classification, categorizing resorptions by aetiological factors (pulpal infection, periodontal infection, orthodontic pressure, ankylotic, impacted tooth or tumour pressure), has also been suggested[4]. So far, it has not gained wide use.

Lesions are usually confined to one tooth, and are most often asymptomatic. Clinical signs may be absent, or limited to an irregularity of the gingival contour, followed by cavitation as the lesion progresses. However, the port of entry may be submarginal, proximal or too small to identify. Sometimes a pink discoloration of overlying enamel can be seen. Periodontal suprainfection can cause pain or discomfort, as might pulpitis in advanced cases. Radiographically, only a small cervical radiolucency may be visible in early stages. Later, irregular lucencies, progressing from the crown into the radicular portion of the tooth are seen. A demarcated outline of the root canal, caused by a thin predentine and dentine layer, is characteristic, even in extensive invasive cervical resorptions[5]. In this patient, the resorptive lesion was suspected, but could not be positively diagnosed from intraoral radiographs or clinical examination. The buccal location, and presence of a pulpal stone obscured overview of the process. A CBCT scan was essential for diagnosis, to estimate the extension of the lesion and plan intervention.

Heithersay classified progression of invasive cervical resorptions[6]:

Class 1

Small invasive lesion near the cervical area, with shallow penetration into dentine.

Class 2

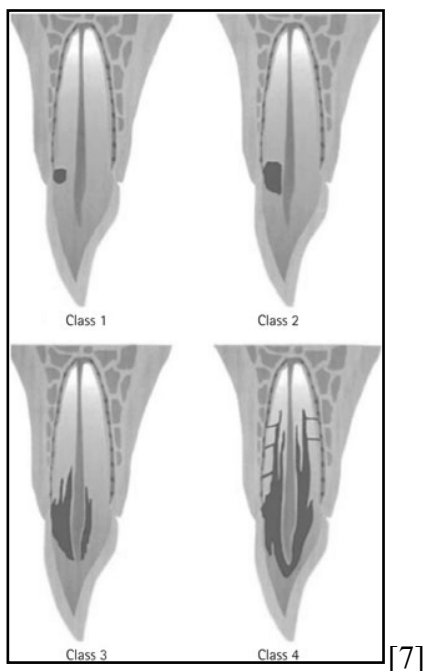
Well-defined lesion that has penetrated dentine close to the coronal pulp. Little or no extension into radicular dentine.

Class 3

Deeper invasion of dentine, extending at least to the coronal third of the root.

Class 4

Large invasive resorptive process, extended beyond the coronal third of the root canal.



The resorptive tissue is initially fibrovascular, devoid of inflammatory cells. As lesions progress, bone-like calcifications are deposited within the resorptive tissue and on resorbed dentine surfaces. Resorption channels burrow into radicular dentine, and interconnects with the periodontal ligament[5].

A defect in the cementum/cementoid layer is likely a prerequisite for invasion of dentine. This may be caused by a developmental deficit, with lack of contact/overlap between enamel and cement fronts, exposing dentine in the cervical area[8]. 5-10% of human teeth display enamel-cemental dysjunctions[9]. Dentine exposure may also be a result of physical or chemical trauma. The resorptive process is mediated by clastic cells, termed odontoclasts, morphologically similar, and possibly identical, to osteoclasts[7]. Osteoclasts are activated through the RANK signal pathway. The TNF-related cytokine RANKL binds on membrane-bound RANK receptors of haematopoietic precursor cells, inducing differentiation and activation of osteoclastic activity. Osteoprotegerin (OPG) is a soluble protein, blocking osteoclast formation *in vitro* and bone resorption *in vivo*. OPG blocks RANK binding to its cellular RANKL receptor. A complex signalling network further regulates osteoclastic activity [10].

The mode of activation of clastic activity in invasive cervical resorptions is not known. However, some aetiological factors have been identified. In a study of 222 patients,

Heithersay analysed potential predisposing factors[11]. All of the factors may in some way injure dental cervical structures, or stimulate cell activity in the adjacent PDL. The results indicated a strong association between invasive cervical resorption and orthodontic treatment, trauma and intracoronary bleaching, either alone or in combination. Association between multiple invasive cervical resorptions and systemic factors have been suggested. Among implicated conditions are hormonal disturbances, hyperparathyroidism, Paget's disease, renal disease, hepatic disease and bone dysplasia. So far, definite associations with systemic disease are not proved. Multiple cervical resorptions are sometimes referred to as idiopathic external root resorptions, indicating the elusive aetiology [2, 12]. Local factors appear to be the most frequent stimulation of resorptive processes. In this patient, the multiple cervical resorptions may have been related to the history of orthodontic treatment.

Invasive cervical resorptions have a potential for recurrence, either due to incomplete removal/inactivation of clastic cells, or development of new resorptive foci. Surgical access and curettage may be necessary. The vascular tissue can impede visualization of the lacuna, or compromise placement of repair material. Application of trichloroacetic acid to induce coagulation necrosis of the resorptive tissue has been advocated, both for inactivation of resorptive tissue, and to improve haemostasis. Subsequent application of a corticosteroid/antibiotic paste (Ledermix), may enhance inactivation of clastic cells [6]. Treatment protocols for resorptive lesions are not standardized, and calcium hydroxide has been endorsed as an interim dressing. An induced pH increase in dentine may inhibit periodontal tissue osteoclastic acid hydrolases and activate alkaline phosphatases, as well as induce chemical burning similar to that obtained by trichloroacetic acid [13, 14].

Prognosis after treatment of invasive cervical resorptions depends on successful inactivation of the resorptive tissue, and restoration of the affected tooth. In a 3-12 year follow-up of repair of 101 affected teeth in 94 patients, treated with topical application of trichloroacetic acid, curettage, and restoration,

Heithersay found complete absence of resorption or signs of periapical or periodontal pathosis in all class 1 or 2 cases. For class 3 lesions, the success rate was 77,8%, whereas only 12,5% of class 4 teeth were deemed clinically sound. On the basis of this, no treatment or alternative therapy was recommended for class 4 cases[6]. The prognosis for this case, with a class 2 defect, may be considered favourable, provided that all resorptive tissue was removed or inactivated.

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Case 4

Treatment of a vital mandibular left second premolar with cervical perforation in a patient with periapical cemental dysplasia.

A 69-year-old male of Middle Eastern origin was referred to the Department of Endodontics from the undergraduate clinic, for endodontic treatment of a calcified mandibular left second premolar, 35.



Fig. 4.1 - Frontal view 01.12.2011

Chief complaint

The patient presented for completion of treatment of 35.

Medical history

Diabetes mellitus type 2, Angina pectoris, Hypothyroidism

Medicines

Albyl-E *acetylsalicylic acid*
Selo-Zok *beta-blocker*
Levaxin *synthetic thyroid hormone*

Dental history

No deep fillings or carious lesions in 35 before access preparation in September 2011. Records revealed previous doubts regarding the vitality and apical status of 35. Treatment was initiated with the diagnosis apical periodontitis, but except for radiographic findings, it was not elaborated what to this.

Clinical findings

Soft tissues

Pink gingival tissue with somewhat coarse appearance.

Dental

Widespread erosion, abrasion and attrition, several gingival composite restorations. Few occlusal restorations.

Note inconsistencies in the sensibility tests. All examined teeth had weak responses, and there was a mismatch between positive results with cold and EPT. This led to the conclusion that sensibility tests should not be emphasized.

Clinical tests

Tooth	34	35	36
Cold	yes	no	yes
EPT 0-80	80	63	73
Palpation	no	no	no
Percussion	no	horizontal	no
Mobility	no	no	no
PPD	wnl	wnl	wnl

Radiographic findings



Fig. 4.2 - Periapical radiograph 01.12.2011

Tooth 34/36

- normal

Tooth 35

- coronal filling to the marginal bone and mesial cervix, possible perforation at marginal level, close relation to the pulp
- apical radiolucency and -density

Other apical lucent and dense areas:

- 25, 35, 31, 45, 46

Attachment apparatus

- moderate bone loss, 2/3 attachment

Radiographic series

22.09.2011

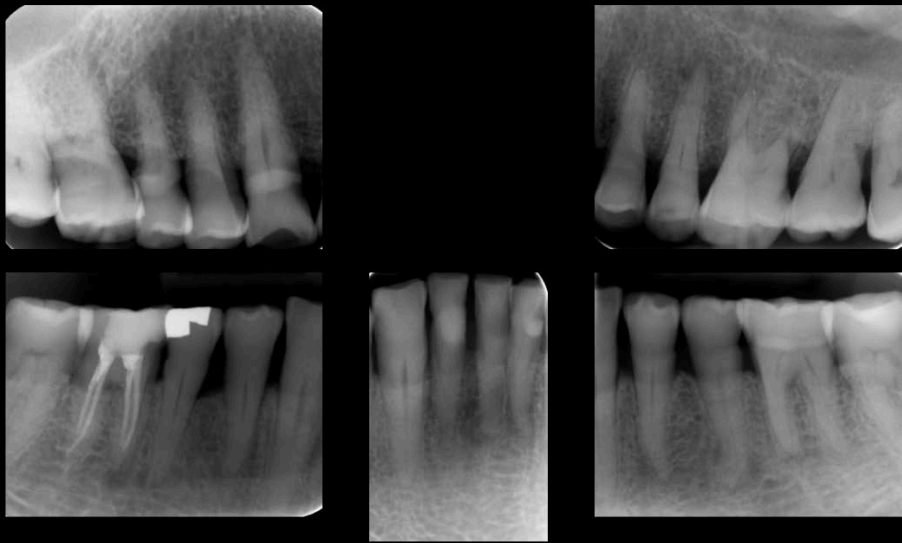


Fig. 4.3 - Preoperative radiographic series 22.09.2011

Diagnosis

- Pulpal** K04.03 Chronic irreversible pulpitis
- Periapical** D16.5 Benign neoplasm of lower jaw bone (periapical cemental dysplasia)
- Periodontal** K05.3 Chronic marginal periodontitis

As a result of the lack of preoperative coronal lesions or fillings (Fig 4.3), inconsistent sensibility tests, report of no preoperative symptoms and the multiple radiolucent and radiopaque areas, the primary diagnosis was set to periapical cemental dysplasia. The pulp was believed to be vital, but involved by the preparation.

Treatment plan

- Conservative endodontic treatment
- (Conservative perforation repair?)

Perforation in the mesial cervical area was regarded likely. A conservative approach was the only treatment alternative considered.

Problem list

- locating canal
- evaluation of outcome

Referred for treatment of an obliterated canal. The canal was visible on radiographs and believed to be negotiable.

If the diagnosis pulpitis and periapical cemental dysplasia was correct, treatment

would not affect the periapical status. As the condition can be misdiagnosed as apical periodontitis, the reverse is also a possibility on future controls.

Treatment progress

01.12.2011

- Examination and treatment planning
- Instrumentation

15.12.2011:

- Obturation
- Perforation repair

29.11.2012

- Follow-up

Treatment - first visit

Anaesthesia: Inferior alveolar nerve block;
Xylocain 1,8 ml.

Rubber dam/disinfection.

The temporary filling was removed with burs. In the cervical area, where the preparation was close to the root surface, a thin dentine wall was suspected. No bleeding after removal of filling and irrigation. Direct probing was avoided. The canal orifice was readily located. Vital pulpal tissue. Working length set by apex locator and radiograph. Instrumentation with handfiles. Intracanal dressing and temporary filling.

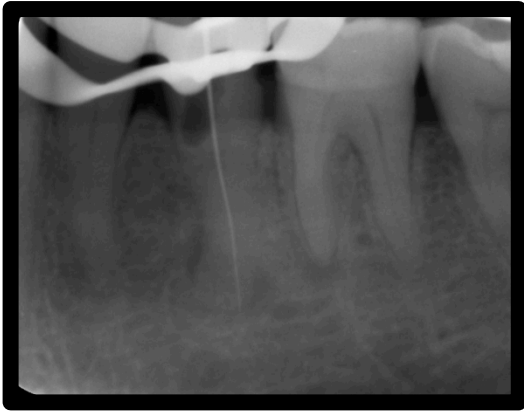


Fig. 4.4 - Working length radiograph 01.12.2011

Mechanical

- Burs, K-files, NiTi handfiles
#055/.02 - 19 mm

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM.

Treatment - second visit

No symptoms. Rubber dam/disinfection. Removed temporary filling. Visual confirmation of a mesial perforation; contours of a 2 mm in diameter perforation was seen in the microscope, and sparse bleeding provoked on probing. The calcium hydroxide dressing was removed and the canal dried with paper points. Masterpoint radiograph. Obturation by cold lateral compaction of gutta-percha before perforation repair.

Mechanical

- Burs, NiTi handfiles
055/.02 - 19 mm

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus
2 mm IRM plug in canal orifice



Fig. 4.5 - Masterpoint radiograph 15.12.2011

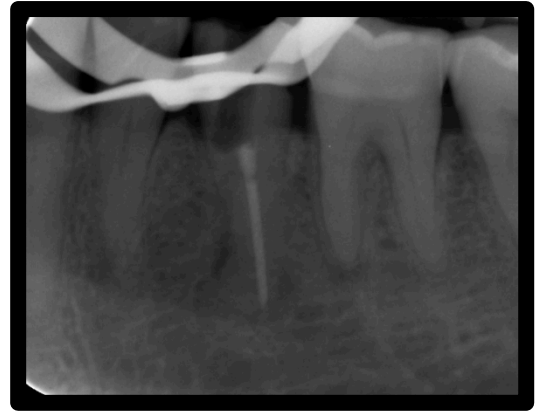


Fig. 4.6 - Control radiograph 15.12.2011

Perforation repair

The access cavity and perforation were irrigated with NaOCl and EDTA. Due to the close proximity to the gingival sulcus, perforation repair was conducted with glass ionomer cement:

GC Dentine Conditioner and Fuji IX™.

A porosity was seen on a control radiograph, and removed by round burs. The access cavity was filled with composite.

Permanent restoration

Acid etch, Scotchbond MP, Filtek™ Z250 A3.



Fig. 4.7 - Control radiograph 15.12.2011

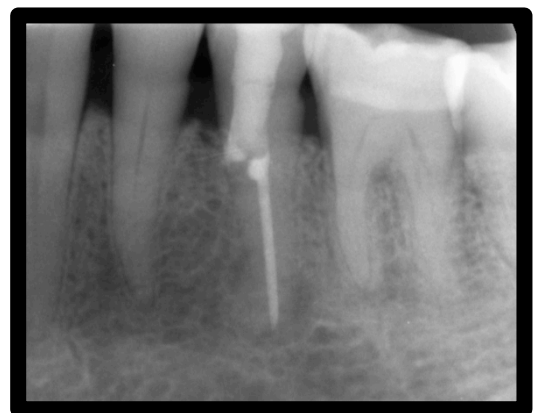


Fig. 4.8 - Final radiograph 15.12.2011

Prognosis

- Endodontic - favourable
- Tooth - favourable - questionable
- Restoration - favourable

The prognosis was considered favourable, with some uncertainty concerning weakening of the cervical area.

Follow up



Fig. 4.9 - 1-year control 29.11.2012

The apical radiolucency showed signs of reduction, which may be consistent with maturation of the dysplastic lesion.

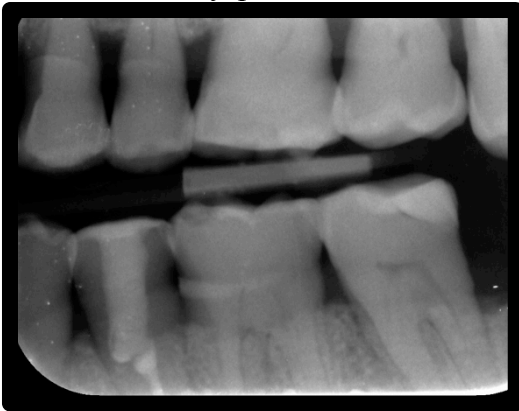


Fig. 4.10 - Bitewing 13.01.2013

No signs of marginal bone loss in relation to the perforation repair.

Evaluation

The perforation should preferably have been detected and obturated earlier. On the first visit, the focus was to avoid breaking the seemingly thin dentine barrier, assuming there was not an actual perforation. When the perforation was verified, no exudation was seen and it was judged to be under visual control during obturation. This approach allowed overview for perforation repair, without risk of obstructing access to the canal orifice.

Discussion

Periapical cemental dysplasia is also known as osseous (cemental) dysplasia, periapical cementoma and periapical osteofibrosis[1-3]. It is a benign, slowly growing connective tissue proliferation, thought to originate in cellular elements in the periodontal

ligament[2]. Although regarded a reactive process, it is idiopathic, as the source of stimulus is unknown[1]. The lesions are asymptomatic, and are most often incidental radiographical findings. They are usually detected between ages of 30 and 50, and there is a predilection for female and black patients. It is estimated to occur in 2-3 teeth per 1000. Periapical cemental dysplasia is most commonly seen in relation to the periapical region of lower incisors, but may be found on other locations. Usually multiple lesions are found, but solitary lesions may occur[3, 4]. Periapical cemental dysplasia develops over time, and the radiographical characteristics change during maturation. In an early, osteolytic stage, proliferation of cementoblasts and resorption of alveolar bone occur. The periodontal ligament space and lamina dura is lost. Radiographically, lesions at this stage are indistinguishable from apical periodontitis, but sensibility tests should indicate vital pulps. During the next stage, foci of cementum deposit are evident, and the appearance is mixed radiolucent and radiopaque. In the third stage, a circumscribed radiopaque calcification, sometimes bordered by a narrow radiolucent line may be seen. The growth of the lesions is seldom, if ever, progressive. Pulpal vitality is not affected by the maturation[3, 4].

In addition to differential diagnostic challenges with respect to apical periodontitis, periapical cemental dysplasia lesions may also resemble hypercementosis, condensing osteitis or cementoblastoma.

Hypercementosis is an excessive formation of secondary cementum, and typically cause a symmetrical enlargement of root circumference[2]. Illustration 4.1.

Condensing apical periodontitis or condensing osteitis, is formation of dense bone in response to low-grade irritants, typically chronic pulpitis[1]. Illustration 4.2.

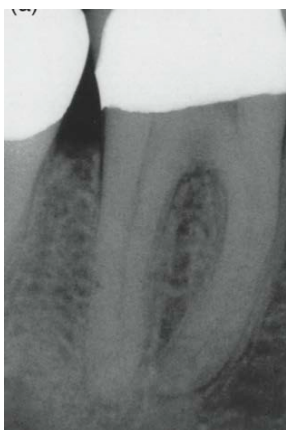
Benign cementoblastoma or 'true cementoma' is a very rare odontogenic tumor, less than 100 cases are described. It is characterized by formation of cement or cementum-like tissue attached to roots. Predilection to lower molar or premolar areas is strong. These neoplasms are usually asymptomatic. Pulp vitality is not affected, but root resorptions may occur[5, 6]. As their growth potential is unlimited, and recurrence have been reported, any

unspecified radiopacity should always be monitored and intervention considered. Illustration 4.3.

In this patient, a cervical perforation was confirmed on the second visit. As discussed in case 14, prognosis for perforations in the cervical area is considered uncertain. This perforation was seemingly enclosed by marginal bone, which lends a more favourable prognosis. Nevertheless, the iatrogenic damage and likely uncalled-for endodontic treatment, led to a considerable impairment of the tooth. Weak and inconsistent responses to sensibility testing presumably represented a particular challenge in obtaining correct diagnosis.



Ill. 4.1 - Hypercementosis, from [2]



Ill. 4.2 - Condensing apical periodontitis[1]



Ill. 4.3 - Benign cementoblastoma[1]

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Case 5

Treatment of a maxillary left second molar with a retained endodontic file.

A 26-year-old female was referred to the Department of Endodontics from the undergraduate clinic, for endodontic treatment of the maxillary left second molar, 27.



Fig. 5.1 - Frontal view 07.03.2013

Chief complaint

The patient presented for completion of treatment of 27. She had concerns over a retained fragment of an endodontic file

Medical history

Non-contributory.

Dental history

Patient at the undergraduate clinic from 2010. Symptomatic pulpitis in 27 after excavation of caries in November 2012. Endodontic treatment was initiated, and during the course of treatment, a K-file #008 separated in the second mesiobuccal canal. The patient was referred for completion of the endodontic treatment.

Clinical findings

Soft tissues

Healthy pink gingival tissue, adequate oral hygiene.

Dental

Fixed orthodontic retainers in maxillary and mandibular front.

Posterior amalgam and composite fillings, temporary fillings in 27 and 37.

Clinical tests

Tooth	25	26	27
Cold	-	yes	no
EPT 0-80	-	45	79
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	wnl	wnl	wnl

RADIOGRAPHIC FINDINGS



Fig. 5.2 - Periapical radiograph 07.03.2013

Tooth 25

- large coronal restoration, mesial caries
- root filled, normal apical conditions

Tooth 26

- small coronal restorations
- normal periapical conditions

Tooth 27

- large coronal restorations
- apical lamina dura on distobuccal and palatal roots
- a small widening of the PDL space on the mesiobuccal root
- a 7 mm long, slim radiopacity centred in the mesiobuccal root, supposedly the separated instrument

Attachment apparatus

- normal periodontal conditions

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	Normal
Periodontal	Normal
PAI	2

The initial diagnosis was pulpitis and three canals were instrumented. Suspected uninfected necrosis in the second mesiobuccal canal.

Treatment plan

- Conservative endodontic treatment

Problem list

- Access to the second mesiobuccal canal

The patient had been notified of the instrument separation. She was informed that an attempt could be made to remove or pass the instrument, alternatively it would remain in the canal.

Treatment progress

07.03.2013

- Examination and treatment planning
- Instrumentation, separation of BR5

23.04.2013

- Removal of separated file from MB2 canal

15.05.2013

- Removal of fragment of BR5 from MB canal
- Obturation

Treatment - first visit

Anaesthesia: Infiltration, Septocaine 1,7 ml.
Rubber dam/disinfection.

Removed temporary filling. Three negotiated canals were identified and the retained file could be seen in the MB2 orifice. Some debris and necrotic pulp tissue in the pulp cavity and orifices. A dentine overhang complicated access to the mesiobuccal canals, this was removed.

All canals and working lengths were not visible on the original WL radiograph. The lengths were controlled by apex locator and a radiograph. It was decided to recapitulate the MB/DB/P canals with BioRaCe files. The full working lengths were readily obtained with the rotary files. When the BR5 instrument was to be withdrawn from the mesiobuccal canal, the patient reduced the opening of her mouth, and vertical movement of the file was restricted. A light cracking sound was heard, and after removal of the file it was evident that 2 millimetres of the instrument apex was missing. A control radiograph (fig 5.5) confirmed the fragment 1-2 mm from the mesiobuccal root apex. The patient was informed, and as time was limited, attempt on

removing both fragments was postponed to the next visit.



Fig. 5.3 - Working length radiograph 07.03.2013

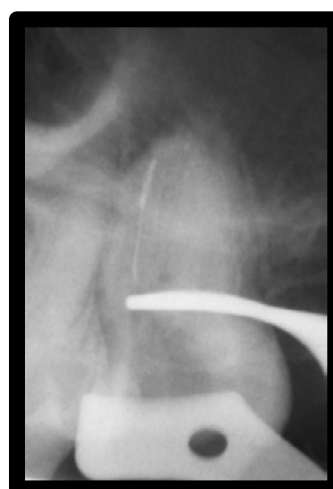


Fig. 5.4 - Control radiograph - separated BR5 07.03.2013

Mechanical

- Burs, ultrasonic tips, K-files, BioRaCe,

MB	040/.04 - 18 mm
DB	040/.04 - 18 mm
P	060/.04 - 17,5 mm

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms.

Anaesthesia. Rubber dam/disinfection.

The proximal 2-3 mm of the file fragment in the second mesiobuccal canal was exposed from the isthmus, by ultrasonic preparation with a K-file tip. The #008 file fragment was removed with aid of the IRS® system. The apical mesiobuccal canal was now accessible

by passing the BR5 fragment on either the buccal or palatal side. An interim dressing and a temporary filling were placed.

Mechanical

- Burs, ultrasonic tips, Hedström files, K-files
- IRS® Instrument Removal System

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - third visit

No symptoms.

Anaesthesia. Rubber dam/disinfection.

The distobuccal and palatal canal orifices were secured with paperpoints, and the BR5 fragment was mobilized and transported to the mesiobuccal canal orifice by a smooth ultrasonic irrigation file and removed.

Obturation by cold lateral compaction.

Mechanical

- Burs, ultrasonic tips, NiTi handfiles

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus®

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM.

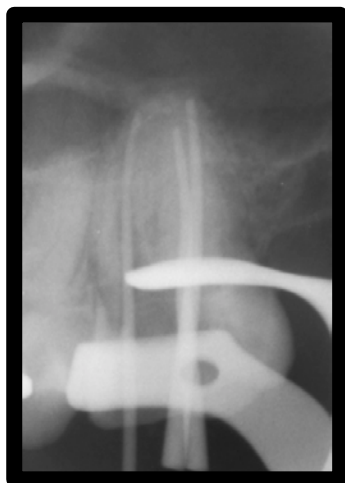


Fig. 5.5 - Masterpoint radiograph 15.05.2013

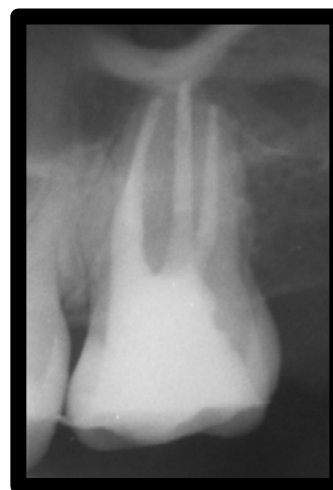


Fig. 5.6 - Final radiograph 15.05.2013

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

Evaluation

If accepted by the patient, a bite block could have supported the mandible during treatment. This might have prevented the BR5 file fracture. Access to the mesiobuccal canals was restricted due to the posterior location of the tooth and the conservative initial access preparation. It is likely that this also contributed to the separation of the file.

Exposure of the second mesiobuccal canal and the process of removing the #008 file fragment resulted in reduction of dentin thickness in the mesial crown. In light of this, and the fact that the canals were not initially infected, the alternative to let the fragment(s) remain, may have been preferred in this case.

Discussion

Rotary instruments may fracture by two modes; cyclic flexural fatigue and torsional fracture. Flexural fractures result from metal fatigue. During instrumentation of curved canals, tension and compression cycles are generated at the maximum point of flexure. The inside half of the file will be subjected to compression, while the outside curvature is in tension. If the rotating instrument is held in a static position, a limited section will be subjected to repeated compression-tension cycles, and this may cause breakage. Torsional fractures occur when a part of the file is locked in the canal. If shaft rotation continues, and the elastic limit of the metal is exceeded, the immobilized section separates. Torsional loads may induce signs of plastic deformation, like unwinding, twisting or

straightening. Reports on which of the two patterns most fractures occur are contradictory, and no study have conclusively answered the question of at which stage of endodontic treatment instruments are more likely to separate[1-3]. Incidence of fractured rotary NiTi instruments are in the range of 0,4-5%, which is similar to figures for stainless steel handfiles (0,7-7,4). Some reservation must be made on accepting these rates, as various methods, all with deficiencies, have been applied in studies of fracture occurrence. Reports on radiographic findings does not account for successfully removed fragments, and examination of discarded files may not yield clinically relevant figures[2]. In a study of patient records at a post-graduate program, the incidence of handfile separations was 0,25%, and incidence for rotary files was 1,68%[4]. These figures may underestimate rotary NiTi separations, as short fractures (0,5-1,5 mm) are not always noted during preparation or filling[3].

Risk of torsional fracture may be reduced by minimizing contact area between file and canal wall. This can be achieved by modifying cross-sectional geometry or varying the taper over the instrument cutting length. In RaCe files, the alternating cutting edges are also claimed to minimize screwing-in tendency. Electropolishing is a method of surface finishing used on some instruments, including RaCe files. Machining of NiTi files produces multiple milling grooves, cracks and pits on the instrument surface. Cracks can propagate along these defects, and the electropolishing may improve mechanical properties[5].

Fracture risk can also be reduced by precautions during rotary instrumentation: An initial glide path preparation is recommended for most rotary systems. All canal instrumentation should be performed with copious irrigation. Smaller instruments are more prone to fracture, and can be limited to single use. Single use systems also appear to gain popularity. Coronal preflaring is advocated. Instrumentation in a 'pecking motion' may distribute the cyclic load over the instrument length. Torque-controlled motors reduce risk of torsional fractures by stopping or reversing rotation if the maximum torque is delivered to the file. This can reduce

incidence of torsional fractures, although it should be noted that frequent engagement of the auto-reverse carries a fracture risk[2, 6].

Separated files can sometimes be bypassed by handfiles, and thus working length may be reached without fragment removal. Ultrasonic files or tips can be applied both for bypassing or dislodging of file fragments. Extraction tube systems like the Masserann Kit (Micro-Mega, Besancon, France) and IRS, rely on exposing coronal fragments with aid of trepan burs or an ultrasonic tip, before locking and extracting the file by a specialized instrument. Use of these systems require straight line access to the fragment, and their application may be limited in posterior or curved canals and for removal of apical fragments[6, 7].

There has been some controversy regarding the prognosis for endodontic treatment after file fracture. Strindberg found 19% higher failure frequency at final follow-up for cases with fractured files[8]. Some studies include too few cases with file fractures to estimate prognosis[9, 10]. Grossmann found no significance of instrument fracture in 47 cases without preoperative apical periodontitis, whereas about half of the pericapical lesions persisted in 19 teeth with retained file fragments[11].

Fu et al. found higher healing rate in cases where separated files had been removed by ultrasonic techniques, compared to cases with fragments left in the canals, but the difference was not statistically significant [12]. In a case-control study of 146 cases and an equal number of matched controls, Spili found success rates influenced by preoperative periapical status, but not significantly affected by presence of retained file fragments[13]. A similar study was performed in 1970 by Crump and Natkin[14]. The result supports that of Spili, in finding no statistically significant difference in failure rate between broken instrument cases and controls.

Endodontic procedural errors in general does not appear to be the direct cause of treatment failure[15]. As removal of separated instruments may not significantly affect the prognosis of endodontic treatment, the decision to remove or leave fragments must be weighed against the risk complications like perforations, heat generation, dislocation of fragments into the periapical tissues or uncalled-for loss of dental hard tissues[6, 7].

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Case 6

Conservative treatment of a necrotic mandibular right first molar.

A 23-year-old female was referred to Tannlegespesialistene AS, Tromsø from a general practitioner in private practice, for endodontic treatment of the mandibular right first molar, 46.



Fig. 6.1 - Frontal view 29.10.2013

Chief complaint

The patient presented for completion of treatment of 46. No symptoms

Medical history

Non-contributory.

Dental history

Emergency treatment after pain of unspecified character two months earlier. A necrotic pulp was encountered in four canals, but adequate working lengths was not reached. The patient stated she had intense, continuous pain before treatment initiation, but only slight discomfort since.

Clinical findings

Some posterior composite fillings, otherwise unremarkable dental and soft tissue findings.

Clinical tests

Tooth	47	46	45
Cold	yes	-	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

Radiographic findings

Tooth 46

- temporary coronal restoration
- pronounced distal curvature on both roots, apical canals were not visible
- the apical PDL space appears widened, but the apical area is not distinct due to root overlap and dense bone interpreted as overprojection of the mylohyoid ridge.

Tooth 45

- normal

Attachment apparatus

- normal



Fig. 6.2 - Periapical radiographs 29.10.2013

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	Normal
Periodontal	Normal
PAI	1

Treatment plan

- Conservative endodontic treatment

No treatment alternatives were considered.

Problem list

Access to apical canals:

- Root curvature:
 - Steps might be established during the initial treatment
 - High risk of instrument fracture
- The apical canals were not visible, which may indicate calcification.

It was intended to first establish straight-line access. Coronal flaring of canals. Negotiation

with small handfiles: #006/#008/#010. Contouring files with a plier if necessary to catch the apical canals. Bulk instrumentation with a new rotary file kit, a pecking motion to limit risk of cyclic fatigue. Copious irrigation.

Treatment progress

29.10.2013

- Examination and treatment planning
- Instrumentation

22.12.2013

- Obturation

Treatment - first visit

Anaesthesia: Inferior alveolar nerve block; Septocaine 1,7 ml. Rubber dam/disinfection Preparation of straight-line access to the canals. Coronal preflaring with Hedström files was repeated throughout instrumentation. The full working length was reached with small handfiles. Files were discarded if signs of unwinding or twisting was seen. Working lengths were controlled by EAL (Sybron Endodontics Elements Apex Locator) and a radiograph. After establishing the WL with a #020 handfile, canals were instrumented by rotary BioRaCe BR0, BR1 and BR2 files. The isthmi of coronal, straight section of both roots were refined with a ultrasonic tip. Passive ultrasonic irrigation. Placed intracanal medicament and temporary filling.

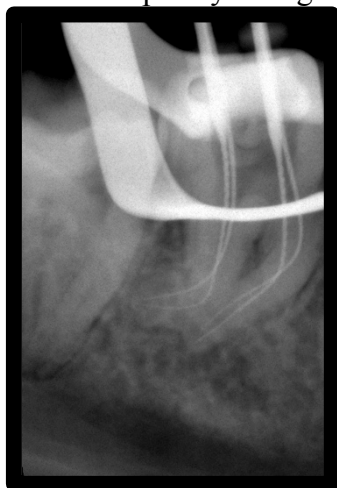


Fig. 6.3 - Working length radiograph 29.10.2013

Mechanical

- Burs, ultrasonic tips, K-files, Hedström files, BioRaCe
 - MB 025/.02 - 25 mm
 - ML 025/.02 - 25 mm
 - D 025/.02 - 25 mm
 - DL 025/.02 - 25 mm

Chemical

- 10 ml 1% NaOCl / 5ml 17% EDTA

- PUI; NaOCl, 2x20s/canal.

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms.

Rubber dam/disinfection.

Removed temporary filling and calcium hydroxide dressing. A 2 mm spiral filler fragment in the DL canal was removed with a Hedström file. The apical canals were instrumented to full working length with #030/.02 NiTi handfiles. Further increase of the apical size was weighted against risk for transportation or instrument fractures, and not performed. Passive ultrasonic irrigation. Masterpoint radiograph. Obturation with cold lateral compaction.



Fig. 6.4 - Masterpoint radiograph 22.12.2013



Fig. 6.5 - Final radiograph 22.12.2013

Mechanical

- Burs, NiTi handfiles
- Irrisafe®

Chemical

- 10 ml 1% NaOCl / 5ml 17% EDTA

- PUI; NaOCl, 2x20s/canal.

Obturation

Gutta-percha and AH Plus
IRM plugs in canal orifices.

Prognosis

Endodontic - favourable
Tooth - favourable
Restoration - favourable

Evaluation

Full working lengths were reached in all canals following the planned strategy. The intracanal dressing could have been placed with handfiles and paper points only.

Discussion

In this case, all canals were instrumented to ISO size 30.

Ingle introduced standardization of diameters and tapers of endodontic instruments and corresponding obturation points in 1961[1]. In 1977, Kerekes and Tronstad published a series of morphometric observations of the apical 1-5 millimetres of roots and root canals of human teeth. The concept of standardization was evolved to include recommendations for minimal apical instrument sizes necessary to predictively achieve a circular preparation and obturation of the apical canal[2-4]. For most roots, last instruments sized \geq ISO 040 was deemed sufficient. An apical ledge supporting the obturating point was strived for[5].

A beneficial effect on treatment outcome if negative cultures were obtained before obturation have been demonstrated [6-8]. This was later confirmed by Sjögren; in teeth with periapical lesions, 94% completely healed within 5 years when cultures were negative at obturation. For cases with positive cultures, the healing was 68%[9]. However, Peters did not find recovery of small number of cultivable bacteria (CFU $< 10^2$) at obturation influential on outcome[10].

Studies have investigated if increased size of apical preparation rendered fewer positive cultures. In 1991, Ørstavik found a trend for fewer positive cultures by instrumentation to sizes >45 , than by size 35 and 40, but the difference was not significant[11]. Yared obtained similar results when comparing instrumentation to size 25 and size 40: No significant difference, but in cultures were fewer after instrumentation to size 40[12]. Dalton et al. and Shuping et al. demonstrated

increasing bacterial reduction by increasing instrumentation sizes[13, 14]. However, in a recent pilot study, Markvart found no significant difference in reduction of positive samples after apical box (#60/.02) or cone (#25/.06) preparation of premolars. The study revealed that a large sample size (> 900) would be necessary for significant results. The result appeared in favour of the smaller apical sized cone preparation, but it could not be excluded that this was due to false negative results as some of the culture negative S3 samples yielded positive S4 samples[15]. Drawbacks of larger apical sizes may include procedural complications like ledge formation, transportation, and perforation[16]. Thus, the association between size of apical preparation and culture results is weak. Moreover, results from prospective studies treatment outcome does not seem to be in favour of large apical sizes. Strindberg found canals instrumented to the smallest apical size to be more successful than larger sizes[17]. Kerekes and Tronstad found no significant difference between canals enlarged to ISO 20-40 and in roots with canals enlarged to ISO 45-100[5]. Similarly, the more recent extensive study of Ng et al. did not find statistic significant differences in success rates for canals instrumented to sizes ≤ 30 (86% success) and >30 (76% success)[18]. To conclude; the apical instrumentation size have not been proven critical for treatment outcome.

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Case 7

Endodontic treatment of a maxillary right premolar with apical periodontitis.

A 61-year-old male was referred to the Department of Endodontics from the Department of Periodontology for endodontic treatment of the maxillary right first premolar, 14.



Fig. 7.1 - Frontal view 04.04.2013

Chief complaint

According to the referral, the patient had received antibiotic treatment for an abscess of suspected origin in 14 three months earlier. He was since without symptoms, but wished to complete treatment of 14.

Medical history

Renal transplantation in 2003.

(Patient also reports medications for cardiovascular conditions and diabetes)

Medicines

Prograf	<i>immunosuppressant</i>
Cellcept	<i>immunosuppressant</i>
Prednisolon	<i>corticostereoid</i>
Persatin retard	<i>platelet inhibitor</i>
Albyl-E	<i>acetylsalicylic acid</i>
Lescol Depot	<i>inhibits cholesterol production</i>
Amias	<i>angiotensin II antagonist</i>
Rocaltrol	<i>calcium and phosphorus balance</i>
Mindiab	<i>anti-diabetic</i>
Zyloric	<i>reduces uric acid production</i>

Dental history

Patient at Department of Periodontology since 2010, has undergone conservative and surgical treatment for periodontal disease. According to the patient records, he was now in a control phase of the periodontal treatment.

Clinical findings

Soft tissues

Gingival retractions, localized gingivitis

Dental

Gingival abrasions.

Full mandibular ceramic-fused-to-metal bridge

Maxillary molars were lost.

Insufficient oral hygiene; gingival dental plaque.

Clinical tests

Tooth	15	14	13
Cold	no	no	no
EPT 0-80	26	80	30
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	wnl	wnl	wnl

Radiographic findings

The development of 14 could be seen on previous radiographs. In January 2011 a deep carious lesion (Fig. 7.2 left), was restored with composite. A periapical lesion was evident on a radiograph from February 2012, but the lesion was unnoticed.

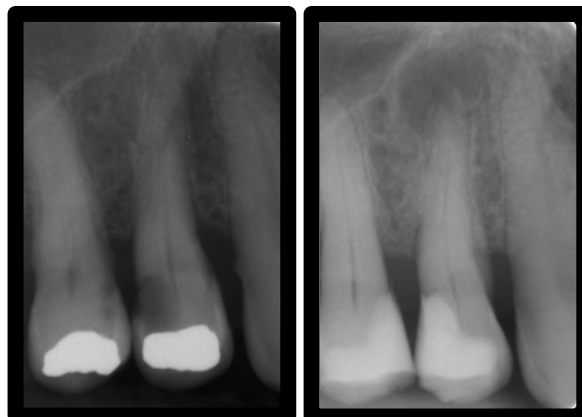


Fig. 7.2 - Older radiographs - January 2011 left and February 2012 right



Fig. 7.3 - Periapical radiograph - 04.04.2013

Tooth 15

- mesio-occlusal restoration
- normal periapical findings

Tooth 14

- deep disto-occlusal restoration, close to, or involving the pulpal space
- apical radiolucency \approx 6 mm diam
- two root apices

Tooth 13

- normal

Attachment apparatus

- moderate loss of periodontal attachment

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	K04.5 Chronic apical periodontitis
Periodontal	K05.3 - Chronic marginal periodontitis
PAI	5

Treatment plan

- Conservative endodontic treatment

No treatment alternatives were considered relevant.

Problem list

- Medically compromised patient
- Inadequate oral hygiene

Conferred with Department of Nephrology, Oslo University Hospital, Ullevål regarding health status. Premedication with amoxicillin was recommended before endodontic treatment.

The patient was encouraged to improve hygiene.

Treatment progress

04.04.2013

- Examination and treatment planning
- Instrumentation

08.05.2013

- Obturation
- 19.02.2014
- Follow-up

Treatment - first visit

2 g amoxicillin 1 h preoperatively.

Rubber dam/disinfection: 0,5% CHX in 70% ethanol.

Reduction of occlusal height. Access cavity preparation. A buccal and a lingual canal were identified. The calcified buccal canal was negotiated with small K-files (#006, 008, 010). Working length determined by EAL (RAYPEX® 6) and a radiograph.

The working length was not immediately reached with a #020 K-file in either canals, thus the R25 Reciproc file was chosen for instrumentation. A glide path for a #020 K-file was established in both canals, and instrumentation was finished with the R25 file.

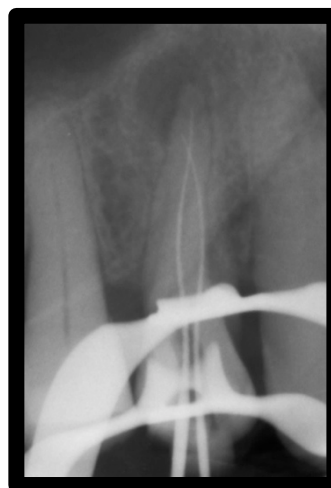


Fig. 7.4 - Working length radiograph 04.04.2013

Mechanical

- Burs, ultrasonic tips, K-files, Hedström file, Reciproc R25
- B 025/.08 - 19 mm
- L 025/.08 - 19 mm

Chemical

- 0,5% CHX \approx 10 ml / 17% EDTA \approx 5 ml

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms after first treatment.

2 g amoxicillin 1 h preoperatively.

Rubber dam/disinfection: 0,5% CHX in 70% ethanol.

Removed temporary filling. The calcium hydroxide dressing was removed by a R25 Reciproc file, irrigation and a ultrasonic tip. Canals dried with paper points. Masterpoint radiograph. Obturation with R25 gutta-percha masterpoints and cold lateral compaction with AH Plus sealer. IRM plug over orifices. The remaining distal composite was removed to explore the possibilities for placement of a permanent coronal restoration. The crown was deemed to undermined for a composite filling, and a prosthetic crown was recommended to the patient. IRM temporary filling.



Fig. 7.5 - Masterpoint radiograph 08.05.2013

Mechanical

- Burs, ultrasonic tips, Hedström files, K-files, NiTi handfiles
- Irrisafe®

Chemical

- 0,5% CHX ≈ 10 ml / 17% EDTA ≈ 5 ml

Obturation

Gutta-percha and AH Plus®
3 mm IRM plugs covered canal orifices



Fig. 7.6 - Final radiograph 08.05.2013

The approximal IRM excess was removed after the final radiograph.

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

The recommendation for a prosthetic coronal restoration was mentioned in the discharge summary.

Follow-up



Fig. 7.7 - Control 19.02.2014

Signs of healing of periapical lesion. Still temporary coronal restoration, some IRM was lost, but the canals were still sealed by the IRM plugs. The patient was encouraged to seek completion of treatment of 14.

Evaluation

The endodontic treatment was without complications, the result appears technically satisfactory and the periapical lesion was healing. A permanent coronal restoration is necessary.

Discussion

In conjunction with endodontic treatment, bacteraemia of isolates with identical phenotypes as recovered from the root canals, have been found[1]. However, the magnitude and duration of bacteraemia after dental procedures is not precisely known. The magnitudes suggested ($< 10^4$ CFU/ml), is similar to that resulting from daily activities like oral hygiene procedures or chewing[2]. In this patient, with suboptimal oral hygiene, dental plaque was likely to cause greater cumulative bacteraemia, than would be induced by endodontic treatment. Nevertheless, antibiotic prophylaxis was administered due to the unreserved

recommendation of the specialist in nephrology.

Prophylactic systemic administration of antibiotics has been recommended for several medical conditions. The American Heart Association (AHA) has been a promoter for antibiotic prophylaxis, with specific guidelines first published in 1955. The latest revision is from 2007, and recommendations are restricted compared with the past. Prophylaxis is advocated only for patients at highest risk for adverse outcome of infective endocarditis (i.e.: with previous infective endocarditis, prosthetic heart valves, or a limited selection of other heart conditions). Doubt is cast whether prophylaxis is effective, and emphasis put on risk of adverse effects. Scientific evidence for efficacy is lacking for all patient groups[2, 3]. The Norwegian Cardiology Association has adopted the AHA guidelines[4].

Other factors frequently suggested to imply a need for antibiotic prophylaxis are prosthetic joints, immunodeficiencies, indwelling catheters or vascular grafts and diabetes mellitus type 1[3, 5]. Among these patients, bacteria of possible oral origin are infrequently isolated in infections. Immunocompromised patients and infective endocarditis is an exception, where viridians group streptococci infections are not uncommon. It has been stated that patients with impaired host defences may benefit from prophylaxis if their white cell count is below 1000-2,500 (normal = 4,000-11,000)[3, 5].

Different antibiotics may be suggested for prophylaxis, but the AHA recommendation of amoxicillin has resulted in a general acceptance for this as the medication of choice for all patient groups[3]. 2 g amoxicillin 30-60 minutes preoperatively, alternatively 600 mg clindamycin for patients allergic to penicillin, is the current regimen advocated by AHA. However, the AHA states that the ability of antibiotic therapy to prevent or reduce the frequency, magnitude, or duration of bacteraemia associated with dental procedures is controversial[2].

Adverse effects of uncalled-for use of antibiotics, is contribution bacterial

resistance, and other harmful outcomes like anaphylaxis, or shift of GI flora with onset of pseudomembranous colitis[3, 6]. A paradox of the practice of prophylaxis, is that involvement of resistant strains is particularly concerning in patients with impaired infection defence.

As guidelines exists for patients with high risk of infective endocarditis, these should receive prophylaxis in conjunction with dental procedures, including 'manipulation of root canals'[4]. Recommendations for severely immunocompromised patients are related to degree of neutropenia, and are also specific[7]. For other conditions, including patients subjected to intravenous bisphosphonate treatment or radiation therapy involving oral tissues, individual considerations should be made in each case. The patient's physician may be consulted before a decision is made[6, 7].

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Case 8

Treatment of a maxillary left lateral incisor with a sinus tract.

A 59-year-old male was referred to the Department of Endodontics from the undergraduate clinic, for endodontic treatment of a maxillary left lateral incisor, 22, with mid-root lateral radiolucencies and a buccal sinus tract.



Fig. 8.1 - Frontal view 23.05.2013

Chief complaint

The patient presented for treatment after a buccal sinus tract was found at an oral examination at the undergraduate clinic. The patient had not noticed it.

Medical history

Non-contributory.

Dental history

Patient at the undergraduate clinic since March 2013. Extensive prosthodontic work, crowns on several teeth, made years ago. 12 was lost after a trauma 30 years before.

Clinical findings

Sinus tract on the mucogingival junction, 5 mm superior to the marginal gingiva of 22.

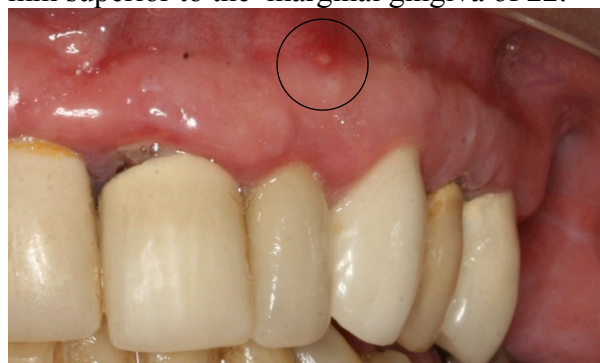


Fig. 8.2 - Sinus tract 23.05.2013

Soft tissues

Unsatisfactory oral hygiene. Gingivitis.

Dental

Metallo-ceramic crowns on all maxillary teeth, except 26. Mesial carious lesion in the margin of the crown on 21.

Discolourations. Attritions on lower incisors.

Clinical tests

Tooth	21	22	23
Cold	yes	yes	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	4mm	3mm	3mm

The patient responded immediately when Endo-ice on a cotton pellet was applied to the crowns. According to records, he had not responded to a cold stimulus on 22. The result of the sensitivity test was not given weight, it was regarded unlikely that ice applied to the porcelain surfaces truly elicited immediate sensation.

Radiographic findings

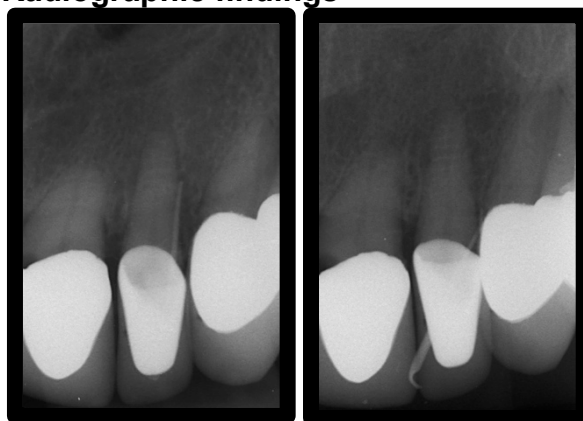


Fig. 8.3 - Periapical radiographs with and without gutta-percha tracing the sinus tract 23.05.2013.

Tooth 21

- metallo-ceramic crown/mesial carious lesion
- short root, normal periapical conditions

Tooth 22

- metallo-ceramic crown
- no lamina dura in the apical half of root
- an apical radiolucency distinguishable on some radiographs (Fig. 8.3, *left*), but not all (Fig. 8.3 *right*)
- 2-3 mm wide radiolucencies on mesial and distal aspects of the mid-root
- sinus tract traced from the buccal mucosa to the distal radiolucency (Fig. 8.3, *left*)

Tooth 23

- metallo-ceramic crown
- normal periapical conditions

Attachment apparatus

- posterior bone loss seen on bite-wings

Older radiographs revealed that the lesion was long-standing (Fig. 8.4).



Fig. 8.4 - Lesion was evident 2 years earlier
24.08.2011

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	K04.5 Chronic apical periodontitis K04.51 Chronic lateral periodontitis K04.62 Sinus tract to the oral cavity
Periodontal	Normal (for 22)
PAI	3

Treatment plan

- Conservative endodontic treatment

The lateral radiolucencies were believed to originate from lateral canals. It might have suffered a root fracture in the trauma 30 years earlier. A fracture line was not seen, thus a

fracture had possibly healed nearly completely before necrosis and infection.

It was decided to instigate access cavity preparation without anaesthetics as the patient responded to the sensibility test.

Problem list

- Access through porcelain crown
- Confirmation of diagnosis; contraindicative findings in regard of sensibility and apical/lateral lucencies

In agreement with the patient, treatment was initiated without administration of anaesthetics.

Treatment progress

23.05.2013

- Examination and treatment planning
- Instrumentation

12.06.2013

- Obturation

20.02.2014

- Follow-up

Treatment - first visit

Rubber dam/disinfection.

Access through porcelain with a cylindrical diamond bur. No sensitivity was noted during treatment. Access cavity preparation and location of canal. No vital tissue and no sensation noted during treatment. The working length was determined by apex locator and controlled by a radiograph. A #020 K-file, but not a #030, readily reached the full working length. A R40 Reciproc file was chosen for instrumentation.

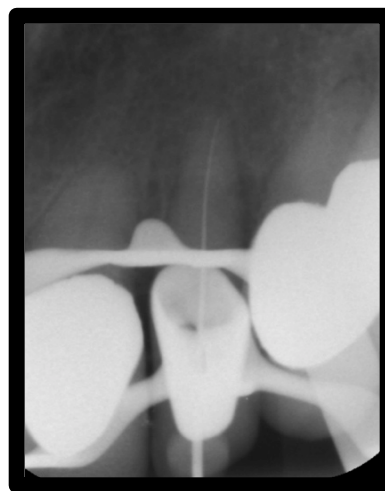


Fig. 8.5 - Working length radiograph 23.05.2013

Mechanical

- Burs, K-files, Reciproc R40 file 040/.06 - 22 mm
- Irrisafe®

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA
- PUI/0,5% CHX

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms. Sinus tract was no longer negotiable with a gutta-percha point.

Rubber dam/disinfection.

Removed temporary filling. The calcium hydroxide dressing was removed by a R40 Reciproc file, irrigation and an ultrasonic tip.

Dried canal with paper points. Masterpoint radiograph. Obturation with gutta-percha and AH Plus sealer, cold lateral condensation technique. IRM.

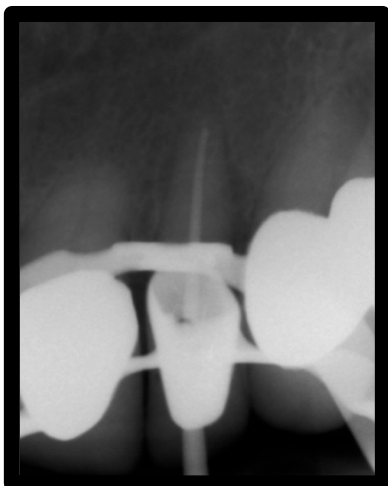


Fig. 8.6 - Masterpoint radiograph 12.06.2013

Mechanical

- Burs, K-files, Reciproc R40 file
040/.06 - 22 mm
- Irrisafe®

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus®

Temporary restoration

IRM.

Prognosis

Endodontic - favourable

Tooth - favourable

Restoration - favourable

The bilateral sealer extrusions were believed to correspond to lateral canals. A fracture of 22 at some time could not be excluded. The patient had no pain on percussion, the negotiation of the apical canal was unhindered, no exudations were seen on paper

points, and the EAL readings were normal. All of this was believed to indicate an intact root. The prognosis was regarded favourable.



Fig. 8.7 - Final radiograph 12.06.2013

Follow-up



Fig. 8.8 - Control 23.04.2014

No signs of the sinus tract, no symptoms, normal mobility, no pain on percussion, periodontal pockets < 4 mm. Establishment of a lamina dura over a slightly widened mesial PDL space. Some reduction of the radiolucency on the distal aspect.

21 had been extracted and was temporarily replaced with a Valplast denture.

Evaluation

The treatment result appears technically satisfactory and with a favourable prognosis.

Discussion

Accessory canals are formed as periodontal vessels are trapped in Hertwig's epithelial root sheet during root formation. As the root grows into the alveolus after tooth occlusion, more vessels are trapped in the apical area of the roots. Hence, the largest number of accessory canals are seen in the apical roots. In furcations, canals may form due to incomplete fusion of the diaphragm. Accessory canals are likely present in most teeth[1, 2].

In the fully developed tooth, blood vessels in accessory canals connect the pulp with the PDL, but contribute little to total pulp blood supply. Necrosis of tissue in accessory canals, provides a pathway for bacteria and toxic substances between the pulp and the PDL. [2]. Ricucci observed that the tissue in accessory canals reflected the status of the pulp. When the level of necrosis had reached the entrance of a ramification, the tissue therein was completely or partially necrotic. The tissue in accessory canals remained vital as far as the pulp tissue in the main canal did so[3].

Lateral canals are accessory canals in the coronal or middle third of the root, usually extending horizontally from the main canal. In anatomical studies, lateral canals is seen in all groups of teeth. In maxillary lateral incisors, researchers have found 4-9 lateral canals per 100 examined teeth[4-6].

It has been considered beneficial to obturate accessory canals[7]. Several *in vitro* studies have been dedicated to compare the efficacy of different techniques, core materials and sealers in this respect[8-13]. However, the significance of filling the accessory canals has not been demonstrated[14]. In a histological examination of 493 human teeth, Ricucci and Siqueira found that that lateral canals were never cleaned and obturated[3]. Filling material that radiographically appeared in lateral canals and apical ramifications was forced into these areas, and enmeshed with necrotic tissue. Bacterial biofilm was sometimes seen on ramification walls, and was always associated with histological inflammation in the immediate PDL. However, radiographic signs of healing of lateral lesions are regularly seen after

endodontic treatment. In these cases, radiographically evident lateral lesions may have been maintained by diffusion of bacterial products from the main canal. The lateral canal itself may harbour infectious material in subcritical counts to maintain a high degree of inflammation[3, 15].

In the presented case it remains to be seen on further follow-up if the remaining lateral radiolucency will resolve completely.

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Case 9

Endodontic treatment of mandibular right canine, lateral and central incisor in a patient with recessive dystrophic epidermolysis bullosa

A 39-year-old female was referred to Department of Endodontics from her general practitioner at the TAKO-centre, for endodontic treatment of mandibular right canine and lateral incisor, 43 and 42.



Fig. 9.1 - Frontal view January 2011

Chief complaint

Pain and swelling in the anterior mandible four months earlier, antibiotic treatment at the time and no symptoms since.

Medical history

Recessive dystrophic epidermolysis bullosa (RDEB).

The disease is caused by a lack of protein anchorage between the epidermis and dermis, and leads to collagen disintegration due to excessive collagenase synthesis. Friction between the layers of skin or mucous membranes causes blisters and erosive lesions – even with minimal trauma, like touch. Lesions heal with formation of dystrophic fibrotic scar tissue.

Both arms and legs were surgically amputated.

Light sensitive

The patient was unable to close her eyes because of atrophic cicatricial tissue of the eyelids. Protection by sunglasses or otherwise covering of the eyes was necessary to preform dental examination and treatment.

Dental history

The patient receives dental treatment at the TAKO-centre, the national resource centre for oral health in patients with rare medical conditions.

The posterior mandibular teeth were lost. Endodontic treatment of the mandibular right first premolar was conducted in 2008.

Endodontic treatment of 43 was initiated in 2011, but not completed, because of the fragile soft tissues and problems with access. 43 had an intracanal medicament ($\text{Ca}(\text{OH})_2$) and an IRM filling.

The patient was very motivated and grateful for dental treatment despite the discomfort and injury it inflicts.



Fig. 9.2 - Erosive lesions on lower lip after dental treatment in 2011



Fig. 9.3 - Limited access caused by trismus and inelasticity of cicatricial soft tissue, in 2011

Clinical findings

Horizontal overbite >10 mm, lingually inclined mandibular teeth

Affection of soft tissue

- Gingival erythema
- Microstomia/vestibular degeneration
- Firm, inelastic tissue:
 - skin/lip/tongue/sublingual
 - Ankyloglossia
 - Lingual depapillation

The patient's soft tissue bore the characteristics of RDEB. Because of her long life with the disease, she had extensive scar tissue formation, and her oral tissues were firm and inelastic. This, and the resulting

trismus, restricted access to her teeth. It was a challenge to examine or treat the patient without additional trauma to her skin or mucosal surfaces.

Clinical tests

Tooth	42
Cold	no
EPT 0-80	-
Palpation	-
Percussion	slight - horizontal
Mobility	-
PPD	-

41, 31, 32 and 33 was sensible to a cold stimulus.

Radiographic findings

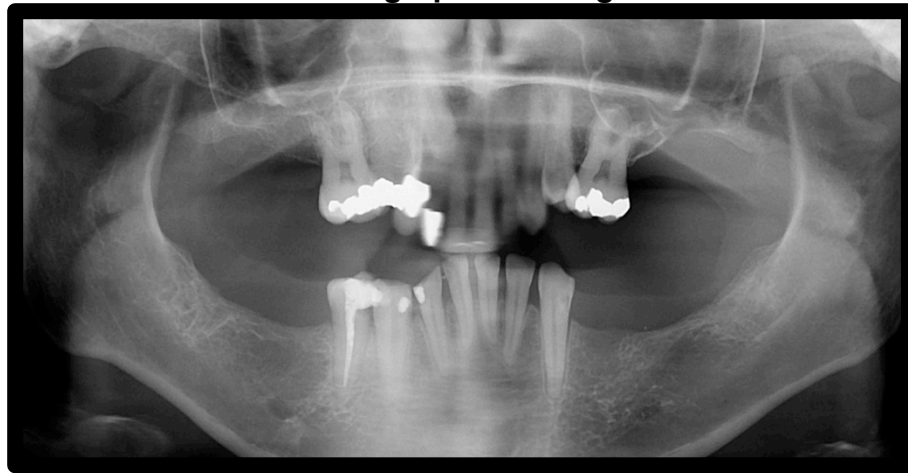


Fig. 9.5 - An earlier panoramic radiograph 01.12.2008

Because of the trismus and inelasticity of the tongue and sublingual tissues, it was not possible to use a paralleling technique for apical radiographs.

Tooth 41

- DI filling - close relation to coronal pulp

Tooth 42

- DBI composite - close relation to pulp
- apical periodontitis

Tooth 43

- incomplete root filling in 2011
- apical periodontitis?

Tooth 44

- endodontic treatment in 2008

Attachment apparatus

- moderately reduced periodontal attachment

Other

- grave attrition on all lower teeth

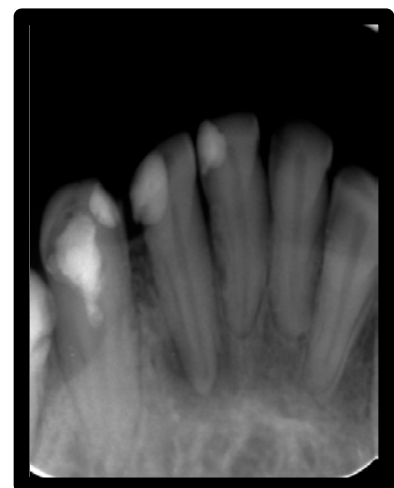


Fig. 9.4 - Periapical radiograph, bisecting angle technique 10.01.2012

On the first visit, time and effort was focused on 42. It was noted that the apical status of 43 had to be examined.

Because of the risk of blisters and ulcerations, a limited clinical examination was performed on the first appointment. The referral from the TAKO-centre stated that the earlier symptoms had its origin in 42. Avitality of 42 was indicated by a sensibility test (cold stimulus), and an apical lesion was apparent on the radiograph. Since meeting for an appointment was complicated by the patient's health, the distance from her home and the need for escort by an assistant, it seemed imperative to conduct some treatment on each visit when a need was revealed.

It was decided to initiate treatment of 42, and investigate the status of the neighbouring teeth during the course of the treatment.

Diagnosis 42

Pulpal	K04.1 Pulp necrosis
Periapical	K04.5 Chronic apical periodontitis
Periodontal	K05.3 Chronic marginal periodontitis
PAI	5

Treatment plan

- Conservative endodontic treatment of 42

It was considered important to keep the remaining dentition, no alternatives to endodontic treatment were explored.

Problem list

- avoiding soft tissue trauma
- access for endodontic treatment
isolation – rubber dam
radiographs

The skin and mucosa was very fragile. Treatment represented a risk of traumatizing the tissues and thereby cause pain and scarring. The lack of tissue elasticity further complicated an atraumatic treatment. However, there appeared to be no alternatives to conducting the endodontic treatment. The patient had very few remaining teeth, and a prosthetic solution did not seem to be an option under the circumstances.

Treatment progress 42

10.01.2012

- Distal GIC-filling

- Instrumentation

24.01.2012

- Obturation
- Permanent composite filling ODI

Treatment 42 - first visit

Precautions to ease access and limit risk of ulcerations:

- Slow and gentle treatment, avoiding mechanical soft tissue trauma.
- Vaseline on instruments/the oral side of the rubber dam, the skin and lip.
- Careful use of vacuum suction; small tip/small suction tube.
- Working length determined by apex locator.
- Occlusal pre- and postoperative radiographs with bisecting angle technique.

During treatment, the rubber dam obstructed access for intraoral film.

Procedure

Rubber dam/disinfection.

The trismus and microstomia complicated placement of the rubber dam. The bow of the rubber dam clamp was medially oriented to avoid interference with the lip.

The treatment was performed without anaesthetics. This was according to the patient's wish, and to minimize manipulation of the soft tissues.

The distal composite filling was lost after access cavity preparation, it was replaced by a GIC filling to secure asepsis during the endodontic treatment.

Preoperative DBI filling

- Dentin conditioner, Fuji II LC.

Mechanical

- Burs, Hedström files, K-files, NiTi handfiles
045/.02 - 18 mm
- Irrisafe®

Chemical

- 1% NaOCl ≈ 10 ml / 17% EDTA ≈ 5 ml
- Passive ultrasonic irrigation/NaOCl.
- 2% Chlorhexidine digluconate, 5 min.

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM.

Treatment 42 - second visit

No symptoms.

Rubber dam/disinfection.

The aforementioned precautions were taken to avoid trauma.

Removed temporary filling and calcium hydroxide dressing. Obturation with a cold lateral condensation technique. Permanent composite restoration.

Mechanical

- Burs, NiTi handfile

Chemical

- 10 ml NaOCl / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus

Permanent restoration

Root filling with sealed with IRM, ≈ 3 mm plug.

Composite filling: Acid etch (37% phosphoric acid), Scotchbond Multi-Purpose adhesive, Filtek Supreme A2.

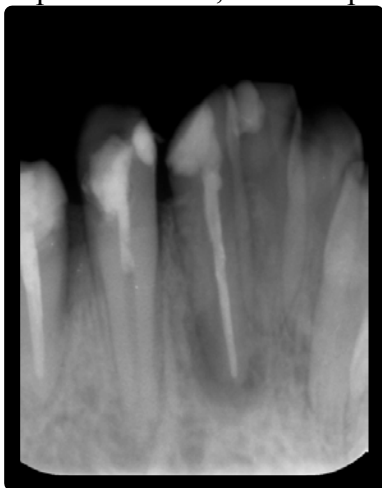


Fig. 9.6 - Final radiograph 42, 24.01.2012

Treatment progress 43

The final radiograph of 42 confirmed an apical radiolucency on tooth 43, which had undergone an incomplete endodontic treatment in 2011.

17.04.2012

- Distal composite filling
- Instrumentation

14.06.2012

- Obturation

05.06.2013

- Follow-up

(The same overall treatment procedure and precautions against trauma as for 42)

Treatment 43 - first visit

Rubber dam/disinfection

The distal IRM was removed and replaced with composite to avoid contamination.

Preoperative D filling

- Acid etch, Scotchbond MP, Filtek Supreme A1.

Mechanical

- Burs, handfiles; Hedström files, K-files, NiTi handfiles
060/.02 - 18,5 mm
- Irrisafe®

Chemical:

- 10 ml NaOCl / 5 ml 17% EDTA
- Passive ultrasonic irrigation/NaOCl.
- 2% Chlorhexidine digluconate, 5 min.

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM.

Treatment 43 - second visit

No symptoms. Rubber dam/disinfection.

Removed temporary filling and calcium hydroxide dressing. Obturation by cold lateral condensation. Temporary IRM filling.



Fig. 9.7 - Final radiograph 43, 14.06.2012

Prognosis 42 and 43

Endodontic - favourable

Tooth - favourable

Restoration - favourable

The prognosis was considered favourable within the limitations of the attrited dentition and the challenges of the primary disease.

Follow-up, 1 year



Fig. 9.8 - 1-year control 42 and 43 - 05.06.2013

At the 1-year control of 42 and 43, there were signs of healing of the periapical lesions, but radiolucent lesion had developed on 41. Sensitivity on cold stimulus of 31/32/33. Treatment of 41 was performed in two visits, following the same procedure as for 42 and 43. A connected buccal and lingual canal were found in 41.

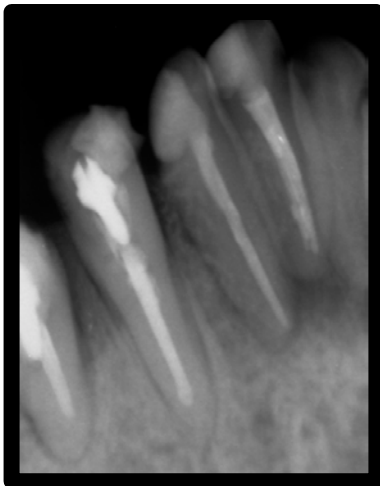


Fig. 9.9 - Final radiograph 41 - 19.06.2013

Prognosis 41

Endodontic - favourable
Tooth - favourable
Restoration - favourable

Evaluation

The use of rubber dam was very efficient in protecting the skin and mucosa, the patient experienced this as comfortable and safe. All treatment was completed virtually without affection of the skin/mucosa.

There appears to be unobturated spaces in the root canal of 42, shown by the irregular periphery of the radiopaque root filling. This may reflect fins or an isthmus towards an

uninstrumented canal, or inadequate lateral condensation. Only one canal was observed during treatment, but access and view was limited. As this was disclosed after completion of treatment of 42, it was decided to await follow-up before any measures were made. At the one-year control, signs of healing was observed on 42 and 43. Retreatment and search for a missed canal in 42 is the treatment of choice if evidence of recurrence of the apical lesion is seen.

DISCUSSION

Epidermolysis bullosa (EB) is a rare inherited disease, with a worldwide incidence of 1/50000 to 1/500000 live births. It presents in three main forms, depending on in which level of the cutaneous/mucosal layers blistering occurs. The (recessive) dystrophic form (RDEB) is the most severe; displaying deepest blistering and subsequent formation of cicatricial scar tissue. Lesions occur on skin and in the entire GI tract. The erosive skin lesions and scar tissue formation leads to flexural contractures of joints and joining of fingers and toes. Morbidity and mortality of RDEB is high[1].

Dental anomalies like hypoplasia and hypomineralizations have a higher frequency in EB patients[2]. This, as well as dietary and hygienic limitations, entails a high caries risk[1]. Fibrotic scarring complicates dental treatment as sequelae like trismus, microstomia, ankyloglossia and general inelasticity of oral tissues evolves[3].

A 2012 consensus report compiled by clinical experts, methodologists and patient representatives, presents guidelines for dental treatment of EB patients[4]. The report emphasize the importance of prophylaxis and dental health in these patients, but also encompass specific advice for treatment. Among the recommendations are: Use of small size (paediatric) instruments, lubrication of lips (Vaseline etc.), avoidance of high vacuum suction and careful use of air-syringe. When local anaesthetics are administered, injections should be deep to avoid mechanical separation of the mucosal layers. It was stated that with caution, a rubber dam could be used. This patient repeatedly expressed she had felt reassured by the protection the rubber dam offered to the skin and oral mucosa. The report states that modified access preparations, like vestibular access to pulps of

incisors, may be necessary if microstomia is severe. Apparently, concern have been raised over the use of sodium hypochlorite if isolation is suboptimal, but the group finds that this is safe in EB patients. If dental treatment causes bullae formation, the blisters should immediately be drained to limit pressure and lesion expansion.

The postoperative radiograph of 42 indicates that some of the coronal and mid-root canal space may have been missed during instrumentation. Despite this, signs of healing were seen at the 1-year control. Vertucci found 98 of 100 mandibular lateral incisors to display one canal at the apex[5]. The most common configuration was one single canal (75%), while a type III (1-2-1) morphology was seen in 18%. A similar Turkish study found two apical foramina in 7 of 100 mandibular incisors[6]. In an examination of the width of roots and root canals between 1 and 5 millimetres from the anatomical apex, Kerekes reported none mandibular incisors with two separate canals apical to 3 millimetres. 20% of the roots displayed two canals in the 3-5 mm level. It was calculated that a circular preparation at 1 mm from the anatomical apex could be obtained with a #045 file in 70% of the cases[7].

As the majority of mandibular incisors have only one apical canal, it is likely that the entire apical section of 42 was instrumented

in this patient. This may have favoured healing, even if the coronal instrumentation might have been incomplete.

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Case 10

Endodontic treatment of a maxillary left central incisor with apical periodontitis.

A 17-year-old female was referred to the Section of Endodontics from a specialist in paediatric dentistry for endodontic treatment of the left maxillary central, 21. Endodontic treatment was initiated several months earlier, but according to the referral, not completed because of unsatisfactory healing of a periapical lesion.



Fig. 10.1 - Frontal view 15.01.2014

Chief complaint

Patient presented for completion of treatment. She had no symptoms, but was not satisfied with the discolouration of 21.

Medical history

Non-contributory.

Dental history

Traumatic injury to the maxillary front in a fall nine years earlier; uncomplicated enamel-dentin fractures of 11 and 21. The teeth were restored with composite and followed regularly after the trauma. In a scheduled control, loss of sensation and a periapical lesion was noted on 21. Endodontic treatment was initiated, but obturation deferred for months as the periapical healing was judged unsatisfactory. According to the referral, calcium hydroxide dressings (including Vitapex) were changed several times. The patient had not experienced any symptoms from 21.

Clinical findings

Soft tissues

Generally healthy pink soft tissue. The incisive papilla was red and slightly oedematous, but not painful on palpation. The patient had not noticed any discomfort. Normal periodontal conditions.



Fig. 10.2 - Red, oedematous incisive papilla, discoloured left central incisor, 15.01.2014

Dental

No fillings except restorations on central incisors. Discoloured composite margins on 21, which had a yellow-grey tone. Talon cusps on all upper lateral incisors and canines.



Fig. 10.3 - Discolouration of 21, 15.01.2014

Clinical tests

Tooth	12	11	21	22
Cold	yes	yes	yes	yes
EPT 0-80	25	35	34	43
Palpation	no	no	no	no
Percussion	no	no	no	no
Mobility	no	no	no	no
PPD	wnl	wnl	wnl	wnl

The patient stated before the clinical tests, that she even after initiation of endodontic treatment of 21, had positive sensibility on electric pulp tests. This was also the case on this occasion. The patient did not see the display counting the stimulus. The results of the clinical tests were not regarded reliable in this patient, and had no influence on the treatment.

Radiographic findings



Fig. 10.4 - Periapical radiographs (*left is mirrored*)
15.01.2014

Tooth 12

- normal periapical conditions

Tooth 11

- coronal radiolucent restoration
- normal periapical conditions

Tooth 21

- temporary filling in access cavity
- radiopaque material in canal
- apical radiolucency, 6 mm diameter
- the outline of the root is not distinct in the apical 3 mm, may suspect some apical resorption

Tooth 22

- canal not distinguishable
- normal periapical conditions

Attachment apparatus

- normal periodontal conditions

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	K04.5 Chronic apical periodontitis
Periodontal	Normal
PAI	4

Treatment plan

- Conservative endodontic treatment
- Intracanal bleach
- New coronal composite restoration

The patient wished to make an attempt on improving the aesthetic of 21.

Treatment alternatives

- Periapical surgery

Surgery was regarded an option if the conservative treatment was not deemed successful on follow-up.

Problem list

- The canal preparation appeared 2-3 mm short of apex, a possible apical step might render access to the apical canal difficult.
- The patient had hopes that the aesthetic appearance of 21 could be improved. She was prepared that the aesthetic outcome may be difficult to predict, especially in a longer perspective.

Treatment progress

15.01.2014

- Examination and treatment planning
- Instrumentation

29.01.2014

- Obturation
- Placed internal bleaching agent

13.02.2014

- Control - changed internal bleaching agent

20.02.2014

- Permanent composite restoration

Treatment - first visit

Anaesthesia: Infiltration, Septocaine 1,7 ml.

Rubber dam/disinfection

Temporary filling removed with burs, exposed canal. Based on its bright yellow colour and radiopacity, the current interim dressing was suspected to be Vitapex. Pulp horns were not completely removed, and discoloured material was excavated from the lateral/incisal pulp cavity walls with a LN bur. Removed interim dressing with NiTi files #050 and ultrasonic tips. Minor apical bleeding was noted when the canal was dried with paperpoints. Over-instrumentation was suspected, but was not be seen in the canal. The working length was determined by apex locator and controlled by radiograph. Working length as determined by EAL (24 mm) appeared 1 mm short on the apical radiograph, this length was kept during instrumentation.



Fig. 10.5 - Working length radiograph 15.01.2014

The canal was instrumented with handfiles, no binding before the size 090 K-file.

Mechanical

- Burs, K-files, NiTi handfiles
090/.02 - 24 mm
- Irrisafe®

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- PUI; NaOCl, 2 x 20 s

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms.

Anaesthesia. Rubber dam/disinfection.

Removed temporary filling and calcium hydroxide dressing.

In the operating microscope, compacted calcium hydroxide was seen in the apical canal. This was carefully removed, and the working length now advanced to 25 mm with a # 080 K-file. Dried canal with paper points, no bleeding. Masterpoint radiograph. Obturation with bioceramic sealer and gutta-percha point. The point was cut 3 mm apical to the orifice, 4 mm IRM plug. Excess sealer removed from the pulpal cavity. An internal bleaching agent (Opalescence® Endo) was placed, and covered with cotton and IRM.



Fig. 10.6 - Masterpoint radiograph 29.01.2014



Fig. 10.7 - Obturated canal with IRM plug 29.01.2014

Mechanical

- K-file - #080/.02 - 25 mm

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- Opalescence® Endo, 35% hydrogen peroxide

Obturation

Gutta-percha and Endosequence BC sealer

Temporary restoration

IRM

Treatment - third visit

No symptoms. The incisive papilla had not changed. It was suspected that the clinical appearance might represent a superficial nasopalatine duct cyst, although no radiographic signs was seen. The patient was referred to the Dept. of oral surgery and oral medicine for examination.

Colour tone of 21 was lightened, but still a darker shade than 11. The discoloured margin of the composite restoration was distinct. It was decided to bleach for one more period.



Fig. 10.8 - Control, 2 weeks internal bleaching, Vitapan A1 left and C2 right 13.02.2014



Fig.10.10 - 1 month postoperatively 18.03.2014

Rubber dam/disinfection.

Removed temporary filling and bleaching agent. A paste of sodium perborate and distilled water was placed in the pulpal cavity, covered by a cotton pellet and IRM.

Treatment - fourth visit

No symptoms. The colour of 21 was similar or lighter than 11.

Rubber dam/disinfection.

Removed temporary filling, and sodium perborate paste by copious water irrigation and a ultrasonic tip. The old composite was removed, and a new restoration was placed.

Composite filling

- Total Etch, 37% phosphoric acid
- Adper™ Scotchbond™ M-P Adhesive
- Filtek™ Supreme XTE composite: A1B in pulpal cavity, incisal/buccal: A2E/A1E.



Fig. 10.9 - Final radiograph 20.02.2014

The patient returned one month later for final photographs. No symptoms, normal findings on palpation, percussion and periodontal probing. The incisive papilla was still unchanged. Signs of an established PDL space on occlusal radiograph.



Fig. 10.11 - Section from occlusal film 18.03.2014

Incisive papilla

Diagnosis from the Dept. of oral surgery and oral medicine:

K09.1 Developmental (nonodontogenic) cyst of oral region (cyst of incisive papilla).

No treatment was considered necessary at the time. The patient was to be recalled for follow-up after one year.

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

Recurrence of discolouration may occur, but the removal of pulp horn necrotic tissue is probably beneficial in this regard.

Evaluation

The patient was satisfied with the aesthetic result.

Placing of an apical MTA plug was discussed after the suspected of presence of an apical perforation at the first visit. Relying on the claimed comparable biocompatible properties, the bioceramic sealer was considered an alternative to MTA in apical canal. The obturation procedure is technically simple compared to apical placement of MTA, and

further cold lateral or warm vertical compaction.

Discussion

6-34% of all school children experience dental trauma. The incidence of trauma in the permanent dentition is highest for males[1]. In this case, trauma occurred when the patient was eight years old. The developmental stage of traumatized teeth influence complication risk. In females, root length formation completes between ages 6,5 and 10 years, the apical closure a little later[2]. Hence, the root of 21 may not have been fully developed at the traumatic incidence. According to the 'Dental Trauma Guide', central incisors with near, but not fully completed root formation, suffering only an enamel-dentin fracture, are not likely to undergo pulp necrosis. If there is an additional concussion or subluxation injury, the risk of necrosis increases to 5% or over 20%, respectively. If root formation is complete, the risk of pulp necrosis after enamel-dentin fracture is approximately 5%[3]. It is likely that the tooth suffered additional injury, or that the apical closure was completed at the time of accident.

Obturation of 21 had been deferred for months, apparently because of unsatisfactory healing of the apical lesion. Little is known about the radiographical characteristics and time frame of initial healing of apical periodontitis[4]. Kerekes and Tronstad found success rates, determined on periapical radiographs, increasing from 71% to 83% between 6 months and 1 year after completion of treatment[5]. Ørstavik recorded that 88% of successful cases were detectable after 1 year [6]. A prolonged observation period is necessary to conclude on periapical healing for some cases[7, 8]. Early interpretation of radiographs underestimate the true success of endodontic treatment.

The effect of long-term calcium hydroxide dressings has been a matter of debate. Cvek observed a high fracture risk after Ca(OH)₂ treatment and obturation with gutta-percha in immature teeth[9]. In an experimental study on immature ovine teeth, Andreasen found decreased fracture resistance after storing Ca(OH)₂ filled specimens for extended time[10]. It should be noted that control teeth were stored for a maximum of 2 months, while teeth in the experimental group were stored up to a year. The largest reduction in

fracture resistance was noted after 360 days, a significant reduction was not seen until after 30 days. A 2013 systematic review of literature on mechanical properties of radicular dentine after Ca(OH)₂ exposure is inconclusive[11]. No clinical studies met the inclusion criteria. The mentioned Cvek study was excluded because of lack of a control group, and based on the fact that high fracture risk was associated with a low developmental stage and resorption defects, and not expressively attributed to Ca(OH)₂. *In vitro*-studies in ovine and bovine teeth demonstrate a tendency for reduced fracture resistance after more than 5 weeks of Ca(OH)₂ exposure, but interpretation should be done with caution as differences in morphological, chemical and physical properties may compromise comparability with human teeth. Underestimation of healing on early radiographs, and the possibility of harmful effects of long-term calcium hydroxide exposure, contradicts the practice of postponing obturation until healing is evident. The routine use of calcium hydroxide is also challenged by the realisation that the dressing may fail to produce sterile canals, and even allow bacterial regrowth in some cases. The effect of single- and multiple-visit root canal treatment appears to not be substantially different[12]. Thus, the treatment of choice should be to finalize root canal treatment of asymptomatic teeth within a limited time, and opt for apical surgery if no evidence of healing is seen on follow-ups.

Bioceramic sealers introduced in recent years claims to incorporate the favourable biological features of MTA in dental materials suitable for obturation with a gutta-percha technique. Handling properties, discolouration and the hardness of set material are issues that have led to some limitations of MTA use. The advantage of combining the biocerams with gutta-percha is obtaining an inert, dimensionally stable, but softer core, allowing retreatment or post preparations. The biocompatibility of MTA have been indicated in numerous studies[13, 14]. So far, relatively few studies investigate the biocompatibility of bioceramic root repair material and sealer, but as discussed in case 20, results are promising.

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Case 11

Treatment of a mandibular left second premolar with apical periodontitis and bifid root anatomy.

A 56-year-old male of Middle Eastern origin was referred to The Department of Endodontics from the undergraduate clinic, for treatment of symptomatic apical periodontitis in the mandibular left second premolar, 35.



Fig. 11.1 - Frontal view 21.05.2013

Chief complaint

Patient came for completion of treatment of 35.

Medical history

Epipharyngeal cancer

Radiation therapy one year earlier,
116 Gy exposure of head and neck

Increased blood pressure

Diabetes mellitus type 1

Medicines

Albyl-E *acetylsalicylic acid*

Selo-Zok *beta-blocker*

Enalapril *ACE-inhibitor*

Simvastatin *cholesterol-lowering
drug*

Insuline

Novorapid *insuline analogue*

Metformin *antidiabetic drug*

Zyloric *inhibits uric acid
formation*

Cipralext *antidepressant*

Loratadin *enhances GI motility*

Dental history

Considerable pain, intra- and extraoral swelling before referral (March/April 2013). 35 responded negatively to sensibility tests and a periapical radiolucency was seen on radiographs, though the crown was seemingly intact (Fig 11. 3). The tooth was accessed at by an undergraduate student; purulence from canal - a cotton pellet soaked in eugenol was placed in the access cavity, and covered by IRM. The patient had received antibiotics as

part of a periodontal treatment regime, and symptoms had subsided.

The buccal cusp fractured, 35 was deemed unrestorable, and the patient was referred to Oslo University Hospital for extraction. On reconsideration, it was decided to perform endodontic treatment and restore 35.

Clinical findings

Soft tissues

Oral hygiene was adequate, healthy pink gingival tissue.

Dental

All molars except 46 were lost. Attrition/erosion on remaining teeth.

A buccal composite restoration restored the buccal cusp of 35.



Fig. 11.2 - Buccal composite 21.05.2013

Clinical tests

Tooth	33	34	35
Cold	yes	yes	no
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	3mm	3mm	4mm

Radiographic findings

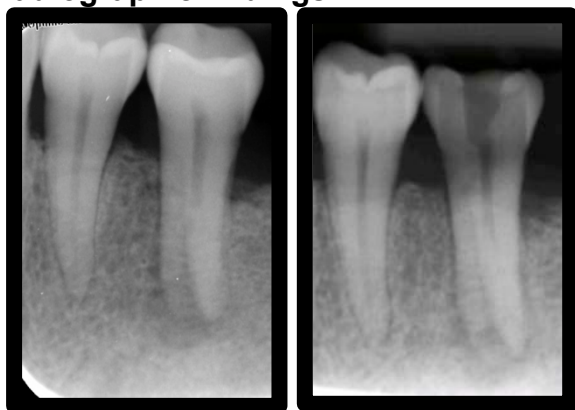


Fig. 11.3 - Periapical radiographs 12.03.2013 *left* and 11.04.2013 *right*

Previous perapical radiographs displayed 35 before and after access.

Tooth 35

- no earlier coronal restorations or lesions
- diffuse apical radiolucency, 6 mm
- anomalous root anatomy; Longitudinal radiolucency on mesial aspect. Two apices or shovel/C-shape-like root anatomy. The apical half of the canal was not distinguishable

Tooth 34

- no coronal restorations/carious lesions
- widened apical PDL space and missing lamina dura was seen on some preoperative radiographs. PAI 2

Tooth 33

- no fillings or coronal lesions
- normal apical conditions on 6 months earlier radiographs (not shown)

Attachment apparatus

- moderate bone loss, distal radiolucency suspected to correspond to bone pocket. Over 2/3 attachment remained.

Diagnosis

Pulpal	K04.1 Pulp necrosis
Periapical	K04.5 Chronic apical periodontitis
Periodontal	K05.3 Chronic marginal periodontitis
PAI	5

Treatment plan

- Conservative endodontic treatment

Treatment alternative

- Extraction of 35.

The patient wished to keep 35. Due to the radiation therapy the patient was at risk for osteoradionecrosis, and any trauma to the mandibular bone, like extractions, should be avoided if possible.

Problem list

- Radiation therapy to head and neck
- General health
- Root anatomy

Section of Oral Surgery and Oral Medicine was consulted as extraction of 35 was considered there, no further information on the evaluation of the patient was obtained. Treatment under antibiotic prophylaxis was recommended.

A CBCT examination was considered, but this would delay treatment initiation, it was decided to access 35 and examine root anatomy as treatment progressed.

Treatment progress

14.05.2013

- Examination and treatment planning

21.05.2013

- Instrumentation

06.06.2013

- Obturation

16.01.2014

- Follow-up

Treatment - first visit

2 g amoxicillin 1 h preoperatively.

Rubber dam/disinfection.

Removed temporary restoration and refined the access cavity preparation. A buccal and a lingual canal was identified. The lingual canal was located in the main canal wall, orifice exposed with small round burs.

Working length was determined by apex locator and a radiograph.

Instrumentation with K-files to size 20, and further BioRaCe rotary files. Passive ultrasonic irrigation. Radiographs with shifted projections to evaluate instrumentation and root morphology. Apical canal preparations appears to be centred in two canals.

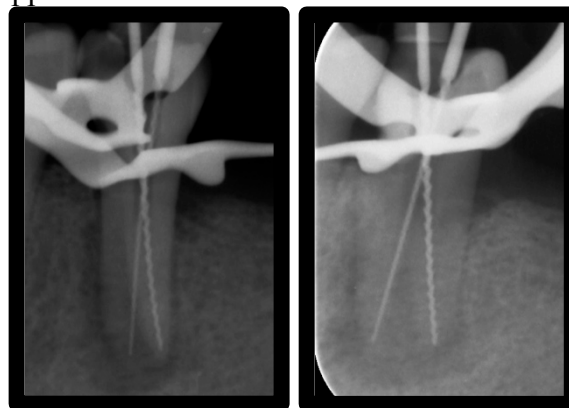


Fig. 11.4 - Control radiographs, ortoradial *left* and disto-eccentric *right* 21.05.2013

Mechanical

- Burs, ultrasonic tips, K-files, BioRaCe
 - B 040/.04 - 18 mm
 - L 040/.04 - 19 mm

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM

Treatment - second visit

Some tenderness in 35 the first week after last treatment. The patient stated 35 initially appeared protruded. Symptoms subsided after the first week. It was suspected that this had been a result of coronal overfilling of IRM.

2 g amoxicillin 1 h preoperatively.

Rubber dam/disinfection.

A small fold distolingual to the buccal canal was opened with hand instruments and a ultrasonic tip; buccal canal had a ribbon shape. Between the canals a no-negotiable fine C-shaped curved line or isthmus was seen.

Modest, clear exudation from apex in the buccal canal, subsided after absorption of the fluid with paperpoints. In the operation microscope, an apically open buccal canal could be seen. The buccal canal was obturated with MTA under light condensation. The lingual canal was secured with a gutta-percha point during MTA placement, and subsequently obturated by cold lateral compaction of gutta-percha with AH Plus sealer. IRM placed over the canal orifices (3 mm submarginal IRM plug seen in Fig. 11.7).

Mechanical

- Burs, ultrasonic tips, Hedström files, K-files, NiTi handfiles
- Irrisafe®

Chemical

- 10 ml 0,5% CHX / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus®

IRM plug to the level of canal orifices



Fig. 11.5 - Masterpoint/control radiograph during placing of MTA 06.06.2013



Fig. 11.6 - Control radiograph 06.06.2013



Fig. 11.7 - Final radiograph 06.06.2013

Prognosis

- Endodontic - questionable
- Tooth - questionable
- Restoration - favourable

The exudation observed in the apical buccal canal was manageable, and viewed as a consequence of apical overinstrumentation and an open apical foramen. On microscopic inspection, all parts of the canal system

appeared instrumented and exposed for chemical disinfection. The view apparent on the radiograph reflects the clinical impression of canal anatomy. The diameter of the periapical/mesial radiolucency appeared increased on radiographs at the last visit. The endodontic prognosis was deemed uncertain, due to the apparently unfavourable development. Coronal dentin to support a permanent restoration remained. The patient was scheduled for a control 2-3 months later.

Follow-up



Fig. 11.9 - Control 16.01.2014

7 months passed before the patient met for a control. He was without symptoms. The buccal composite restoration was lost, but IRM sealed the root canal. Radiographic signs of reduction of apical lucency. The apical periodontal ligament space of 34 still appeared widened and not well defined. 33 and 34 were sensible to cold stimulus, no pain on percussion or palpation. The patient was again referred to the undergraduate clinic for coronal restoration.

The prognosis was re-evaluated in light of the signs of periapical healing:

Endodontic - favourable
 Tooth - favourable
 Restoration - favourable

Evaluation

A CBCT examination was considered for root anatomy investigation. As recent symptoms and prior radiation therapy was considered to press initiation of treatment, this was not performed. In the course of the treatment, overview over the canal system in the operating microscope appeared sufficient.

Two small porosities were observed between MTA layers, 2 and 4 mm from apex. Light pressure was applied during condensation to

avoid extrusion of material to the periapical tissues, a fear which may be considered exaggerated in light of the well-documented biocompatibility of MTA (See case 20). The apparently dense coronal MTA and IRM to the level of the separate orifices left the voids in the apical area well sealed.

Radiation therapy might have rendered the pulpal tissue more susceptible for necrosis and infection (see discussion). This aspect called for consideration of the status of 34. 34 was sensible, and the apical status was unchanged for months. It was chosen to defer endodontic treatment, and control sensibility and periapical radiographs on follow-up examinations.

Discussion

Radiotherapy of the head and neck entails a risk of osteonecrosis of the jaw. The mandible is most susceptible, and osteoradionecrosis risk is estimated to be 2,6-15%. Few cases evolve with exposures <50 Gy. Whereas, over 70 Gy, 9% of patients develops mandibular osteoradionecrosis. Osteoradionecrosis is not regarded a primary infectious condition, although secondary infection of necrotic bone may occur[1, 2]. The pathogenesis of osteoradionecrosis is not clear, but hypoxia, hypocellularity, hypovascularity, or changes in fibroelastic activity might play a part in formation of atrophic bone tissue. Periodontal disease and trauma, including dental extractions, are recognized risk factors for osteoradionecrosis [3, 4]. Thus, preference for conservative endodontic treatment over extractions is especially indicated for patients after radiation therapy of the head and neck.

According to some studies, radiation therapy does not induce pulpal changes, others suggest that fibrosis, atrophy and decreased vascularization may occur[5]. Knowles found immediately reduced sensitivity to electric pulp testing in irradiated mandibular teeth, compared to non-irradiated controls[6]. In maxillary teeth, there was a delayed decrease of sensitivity. SEM examinations 3-5 years after radiotherapy, revealed changes in the pulpal tissue of patients exposed to >60 Gy radiation. The connective tissue was fibrous, arteriole walls thickened, and capillaries almost absent. The teeth exposed to lower radiation doses, displayed reduced sensitivity, but no histological changes. Blood flow throughout a radiation field will be reduced

(50% after 4 months, regains 75% after 1 year), and it was hypothesized that decreased nutrition of pulpal nerves reduce their ability to generate impulses. Another study of irradiated dental pulps, found reduced pulp oxygenation levels during and after radiation therapy, with values increasing again after 4-5 months[5]. In the presented case, one may speculate if pulpal tissue was less vascularized and more prone to infection after the radiation therapy. A possibly long-standing infraction under the later fractured buccal cusp was likely the port of entry for infection.

In this case, the buccal composite restoration fractured and the coronal IRM seal was exposed. The practice of sealing orifices of obturated canals originates from considerations regarding possibility of bacterial penetration of root fillings, and studies that indicate significance of coronal restoration quality for successful outcome of endodontic treatment.

Apical periodontitis of root filled teeth may be the result of reinfection or persistence of primary infection. Poor root filling quality in terms of apical extension or voids has been associated with post-treatment apical periodontitis [7-9]. As *in vitro* studies indicated that bacteria could penetrate canals obturated under ideal conditions, it was postulated that the coronal seal might be of great importance for prevention of infection [9, 10]. In an examination of 1010 endodontically treated teeth, Ray and Trope found that quality of the coronal restoration was of critical importance for outcome[9]. Success rates of both good endodontic fillings (GE), and poor (PE), were affected by a good or poor coronal restoration (GR or PR). Success for GE would fall from 91% (GR) to 44% with PR, and for PE success differed from 68% (GR) to 18% (PR). Tronstad et al. performed a comparable study on an equal number of teeth, but here the quality of the endodontic treatment was most influential on outcome[11]. For good quality root fillings, a good restoration improved on the endodontic success rate. However, if the quality of the root filling was poor, the quality of the coronal restoration was of no importance.

Pisano compared Cavit, IRM, and Super EBA as intraorifice barriers to prevent coronal

microleakage *in vitro* and found that all three leaked less than obturated canals without a barrier. In a retrospective cross-sectional study of root filled teeth with marginal periodontitis, Stassen found significantly more apical periodontitis with the level of the root filling exceeding the marginal bone[12]. They concluded that a hermetic seal of the root-canal space with restorative materials is necessary to obtain a better seal than could be achieved with current root-filling materials. For the observational studies, confounders can be if more resources was invested in restoration when prognosis was expected to be favourable due to pre-operative or intraoperative factors not recognized in the study design. In that case, the differences between the groups may reflect impact of other aspects than merely treatment quality.

In another take on this, Ricucci and Bergenholtz examined 33 coronally exposed root filled teeth histologically[13]. Some of the root fillings had been lacking a coronal restoration for years, some had carious lesions in contact with the root filling material. All but two roots were without visible bacteria in the mid-root and apical canal, and the conclusion was that well-prepared and filled root canals might resist bacterial penetration for years.

Thus, according to these studies, quality of the endodontic treatment affect success, and the quality of the coronal seal might also influence outcome. However, well-adapted root fillings may resist bacterial invasion, even when a coronal filling is missing. It does seem rational to take the measure of a coronal seal as it is a simple, inexpensive way of providing an extra barrier for infection. For some cases, a suboptimal coronal restoration will be made, and the seal may prevent doubts regarding secondary infection when a permanent or temporary restoration fails, as it did in this case.

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Case 12

Treatment of a mandibular left first molar with persistent apical periodontitis and controls of a maxillary left central incisor with horizontal root fracture.

A 22 year old male was referred to The Department of Endodontics from a general practitioner in private practice, for endodontic treatment of a maxillary left central incisor, 21, with horizontal root fracture, and retreatment of a mandibular left first molar, 36.



Fig. 12.1 - Frontal view 08.01.2013

Chief complaint

Patient presented for treatment of a periodically painful tooth 36. 21 was painful on vertical pressure and occasionally when biting.

Medical history

Non-contributory.

Dental history

Patient had received a blow to the maxillary front two years earlier. 21 was diagnosed with a intraalveolar root fracture, and regularly followed up after the trauma. 36 was root filled three years earlier. Patient could not clearly remember circumstances or symptoms at the time. From time to time he experienced pain/tenderness in 36.

Clinical findings

Soft tissues

Gingival retractions.



Fig. 12.2 - 21 two years after trauma 08.01.2013

Dental

Anterior cross bite: 12 and 42/43.

Occlusal composite filling and enamel fracture 36.



Fig. 12.3 - Filling and enamel fractures 36
08.01.2013

Clinical tests

Tooth	11	21	22
Cold	yes	no	yes
EPT 0-80	19	30	36
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

Tooth	35	36	37
Cold	yes	-	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

The pain the patients sometimes experienced on loading 21 was not provoked by normal percussion or palpation.

Radiographic findings



Fig. 12.4 - Periapical radiograph 08.01.2013

Tooth 11

- normal

Tooth 21

- normal PDL space
- horizontal radiolucent line 7 mm from the apex
- no coronal lesions

Tooth 22

- normal



Fig. 12.5 - Periapical radiograph 08.01.2013

Tooth 35

- normal

Tooth 36

- coronal composite filling
- distal root: Root filling 2 mm short of apex. Normal PDL space, apical radiopacity.
- mesial root: Root filling 3 and 5 mm short of apex. Apical radiolucency, 4 mm diameter, surrounded by a 2 mm radiopaque zone.

Tooth 37

- occlusal composite fillings

Attachment apparatus

- normal

Diagnosis

21

S02.53 Root fracture

Pulpal Normal

Periapical Normal

Periodontal Normal

PAI 1

36

Pulpal K04.19 Root filled tooth
K04.1 Pulp necrosis

Periapical K04.5 Chronic apical
periodontitis

Periodontal normal

PAI 4

Treatment plan

21

- Observation

36

- Conservative endodontic retreatment

The patient was encouraged to seek dental treatment if symptoms of necrosis/infection of 21 developed (discolouration, pain, swelling, increased mobility).

Problem list

- Access to apical canals of 36

Apical steps/ledges were anticipated, in the short-instrumented mesial canals.

Treatment progress

08.01.2013

- Examination and treatment planning
- Removal of gutta-percha

10.01.2013

- Established working lengths
- Instrumentation

24.01.2013

- Instrumentation of distobuccal canal
- Obturation

23.01.2014

- Follow-up

Treatment - first visit

Anaesthesia: Inferior alveolar nerve block:

Xylocain 1,8 ml.

Rubber dam/disinfection.

Accessed the canals. The bulk of old obturation material was removed by a rotary BioRaCe BR4 file (#035, 0.04 taper) at 900 rpm. Apical gutta-percha removed with aid of chloroform and hand files. There was not time to complete the instrumentation on the first visit. As seen on the control radiograph, desired working lengths were not reached:

MB 18 mm 1,5 mm short
 ML 17 mm 2,5 mm short
 D 17 mm 1,5 mm short



Fig. 12.6 - Working length radiograph 08.01.2013

Mechanical

- Burs, Hedström files, K-files, BioRaCe

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms. Rubber dam/disinfection. Advanced 1 mm in the mesiobuccal canal. Instrumentation with BioRaCe and hand files. Extruded sealer was seen on the buccal side of the distal canal, this was removed, but a distinct DB canal was not seen. Mesial canals were opened with ultrasonic tips/LN burs towards an isthmus containing sealer extrusion and debris.

Mechanical

- Round/LN-burs, ultrasonic tips, Hedström files, K-files, BioRaCe

MB 040/.04 - 19 mm

ML 040/.04 - 17 mm

D(L) 080/.02 - 17 mm

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- PUI; NaOCl

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - third visit

No symptoms. Rubber dam/disinfection

As the working length radiograph indicated that a second distal canal might be present due to the acentric placement of the file in the distal root, more dentine was removed on the buccal aspect of the D orifice. A distobuccal canal was located and instrumented by hand files.

DB 045/.02 - 18 mm

Canals dried with paper points. Masterpoint radiograph.



Fig. 12.7 - Working length radiograph 24.01.2013

Obturation; cold lateral compaction of gutta-percha in mesial canals. Gutta-percha masterpoints were placed and secured by lateral compaction in distal canals before cutting at 5-7 mm from full length. The coronal distal canal was obturated by warm vertical condensation. AH Plus sealer. IRM.

Mechanical

- Burs, K-files, NiTi handfiles
- Irrisafe®

Chemical

- 1% NaOCl ≈ 10 ml / 17% EDTA ≈ 5 ml

Obturation

Gutta-percha and AH Plus®



Fig. 12.8 - Masterpoint radiograph 24.01.2013



Fig. 12.9 - Final radiographs, shifted; disto-eccentric *right* 24.01.2013

Prognosis

Endodontic - favourable
 Tooth - favourable
 Restoration - favourable

Follow-up

No symptoms. 36 displayed signs of apical healing and establishment of normal PDL structure. The clinical presentation of 21 was unchanged. A subtle reduction in horizontal fracture line width may be due to different radiographic projections, or fracture healing. Narrowing of root canal in the apical fragment and immediate to fracture line in the coronal fragment.

Evaluation

The wide canal dimensions seen on the radiographs reflect removal of sealer and necrotic tissue in the isthmi of the two roots. Preferably, a millimetre working length could have been gained in the ML/DL canals, but this did not appear to impede healing of the apical lesion.



Fig. 12.10 - 1-year control 36, 23.01.2014



Fig. 12.11 - 1-year control 23.01.2014

Discussion

Root fractures occur in only 1% of traumatized teeth[1]. Recommendations for examination after dental traumas are: Radiographic; periapical radiograph with 90° horizontal angle and central beam through the tooth in question, occlusal view and periapical radiograph with mesial or distal angulations. CBCT might provide enhanced visualization. Negative sensibility tests may indicate permanent or transient neural damage[2, 3].

Healing after intraalveolar root fractures is influenced by age, root maturity, immediate sensibility, displacement or mobility of coronal fragments and diastasis between fragments[1]. Andreasen and Hjørting-Hansen classified healing for intraalveolar root fractures[4]:

- 1) Healing with hard tissue (fragments in close contact, fracture line not or slightly visible)
- 2) Healing with interposition of hard and soft tissue (fragments separated by ingrowth of hard tissue, surrounded by a periodontal-like space)
- 3) Healing with interposition of only soft tissue (close fragments close, but separated by distinct radiolucent line, rounding of sharp fracture edges)
- 4) No healing (persistent or widened space between fragments, radiolucency in the alveolar bone adjacent to fracture due to pulp necrosis in the coronal fragment)

Healing of the different groups depends on the aforementioned factors, but overall rates have been found to be 78% successful healing (whereof 30% hard tissue healing), 22% developed pulp necrosis.

Optimal repositioning of dislocated fragments favours healing. Positive effect of repositioning is most pronounced when diastasis is < 1mm. Initial diastasis > 1 mm implies a necrosis risk of 50%. Short term, non-rigid splinting is recommended for repositioned coronal fragments (as for luxated and avulsed teeth). Splinting beyond 3-4 weeks does not improve prognosis, except for fractures close to the cervical area, where splinting up to 4 months may be advisable. Administration of antibiotics is not beneficial, and may in fact increase rate of necrotic cases. If pulp necrosis develops, endodontic treatment of the coronal fragment is indicated[2, 3]. For this patient, the seemingly decreased radiolucent line (Fig 12.4 and 12.10) might indicate that 21 is healing with hard tissue.

The microbial flora of primary and persistent/recurrent root canal infections have different characteristics. Over 450 unique bacterial taxa have been identified in endodontic infections. Primary infected canals generally harbour 10-40 species of a mixed consortium, dominated by obligate anaerobic bacteria. Total bacterial counts vary from 10^3 to 10^8 cells per infected canal. Bacterial composition differs from individual to individual, indicating a non-specific aetiology where multiple organisms may play a role[5, 6]. Molecular methods have contributed to increased knowledge of endodontic infection diversity. *Tannerella forsythia* is not readily cultivated, but is today frequently detected in primary infection. It is of interest as it together with *P. gingivalis* and *T. denticola*, also regularly found in root canals, constitute the red complex of periodontal disease[5, 7]. Species identification, and appreciation of spirochetes as an endodontic pathogen has also been promoted by new techniques. This is compelling, as they are highly pathogenic in other organs, and display virulence factors like host cell adhesion, chemotaxis, motility, penetration capacity, and host evasion[8]. *Fusobacterium nucleatum* is common in long standing infections. It has the ability to coaggregate with other oral bacteria, including several species encountered in root canals, like *P. gingivalis*, *T. denticola*, *A.A.*, *P. intermedia*, *Eubacterium* and *Actinomyces* species[7].

For failed cases with inadequate endodontic treatment, root canal samples may harbour similar bacterial diversity and density as primary infections. However, in well-treated cases, usually 1-5 species are found, and there is an equal distribution of facultative and obligate anaerobes [5, 6]. *Enterococcus faecalis* is the species most often encountered after failed treatment, about 9 times more often than in primary infected canals [9, 10]. Fungi are also occasional findings in primary infections, but *Candida* species have been detected in 18% of retreatment cases. *Candida albicans* is the most commonly found species[5].

E. faecalis and yeasts share properties which may enable them to grow in root filled canals; ability to grow in monoinfections; invade dentine; and resistance against root canal disinfectants[6, 11, 12].

Calcium hydroxide does not predictably eradicate these organisms. *E. faecalis* has been found to be particularly resistant against calcium hydroxide when inoculated in dentin blocks or in the presence of dentine powder *in vitro*[11, 13]. In fact, activity of sodium hypochlorite, chlorhexidine, iodine potassium iodide, may also be delayed or lost in presence of dentine powder, bovine serum albumin, heat killed microbial cells, hydroxyl apatite or collagen[13-15]. Chlorhexidine and iodine potassium iodine have been found more effective in eradication of *E. faecalis* than sodium hypochlorite and calcium hydroxide[13, 14, 16, 17].

Actinomyces israeli is associated with treatment resistant extraradicular infections, and has also been identified in root filled canals[6]. *Actinomyces* species also display relative resistance towards antimicrobials and can survive in monoinfections.

Thus, a shifted and more resistant microbial flora is often encountered in retreatment cases. For the presented patient, where the root filling was inadequate; one missed canal, short mesial root filling and uninstrumented isthmus, the microbial composition may have been comparable with a primary infection, and signs of healing of the periapical lesion was seen on follow-up.

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Case 13

Retreatment of a maxillary right lateral incisor prior to orthodontic extrusion.

A 50-year-old female was referred to the Department of Endodontics from a postgraduate student at the Department of Prosthodontics, for retreatment of the maxillary right lateral incisor, 12. Orthodontic extrusion of 12 was under consideration, the relatively short root was to be restored with a post and crown, or replaced with an implant.



Fig. 13.1 - Frontal view 27.02.2012

Chief complaint

Patient had sought treatment for an aesthetic unsatisfactory 12.

Metallo-ceramic crown on 12. A facial gingival recession exposed a brown-grey discoloured root.



Fig. 13.2 - Frontal view 27.02.2012

Medical history

Non-contributory.

Dental history

The patient wished to improve frontal aesthetics. She was referred to the Department of Prosthodontics for implant replacement of 12, but there was not sufficient bone to secure prospects of the aesthetic outcome and treatment alternatives were considered. Orthodontic extrusion to augment bone was planned before implant treatment or processing of a new crown on 12.

Clinical findings

Soft tissues

Healthy light pink gingival tissue.
Moderate gingival recessions.

Dental

Gingival abrasions.
Posterior composite restorations. Full ceramic crown on 11.

Clinical tests

Tooth	12	11
Cold	-	-
EPT 0-80	-	-
Palpation	no	no
Percussion	no	no
Mobility	no	no
PPD	wnl	wnl

Radiographic findings



Fig. 13.3 - Periapical radiograph 16.04.2012

Tooth 13

- mesial filling, no radiographic contrast

Tooth 12

- root filled, 3 mm short of apex, the apical canal was not distinguishable
- apical radiolucency, 3 mm diameter

- mesial composite filling. According to the patient records, the root filling was exposed to a carious lesion before placement of composite. Radiolucency on the apical composite margin, likely leakage to the root filling.

Tooth 11

- root filled/intracanal post
- the apical PDL-space appear widened on some radiographs

Attachment apparatus

- normal periodontal conditions
- reduced mesial bone attachment on 12

Diagnosis

Pulpal	K04.19 Root filled tooth
Periapical	K04.5 Chronic apical periodontitis
Periodontal	Normal
PAI	3

There were doubts concerning the possibilities of performing aseptic endodontic treatment on 12. It was decided to access the canal and explore the composite margins.

Treatment plan

- Conservative endodontic retreatment

Approaches considered

- Cementing a temporary crown or use of a copper ring to seal the mesial cavity.
- Surgical exposure and replacement of mesial composite.

Problem list

- contamination risk/leakage
- apical curvature and possible obliteration
- ledged canal?

If a leakage was not controllable, cementation of a copper ring or replacement of the composite was to be considered. Sound apical conditions is a prerequisite for orthodontic extrusion[1], even if the tooth will later be extracted. Apicectomy was not regarded an option as then remaining root length of 12 was deemed to be too short.

The patient understood uncertainties both in respect of outcome of endodontic treatment and for the orthodontic bone augmentation procedure, and wished to undergo the proposed treatment.

Treatment progress

18.10.2012

- Examination and treatment planning

29.11.2012

- Definite treatment plan
- Instrumentation

09.01.2013

- Obturation

20.02.2014

- Follow-up

Treatment - first visit

Rubber dam/disinfection

The mesial area of suspected leakage was exposed in the access cavity. Leakage could not be found but a void in the composite was seen and probed. The void was covered with IRM from the canal side, applied with an endodontic plugger.

Most of the old gutta-percha was removed by hand instruments at 19 mm (old working length). To avoid risk of transportation and thereby complicate localization of the apical canal, the last gutta-percha was removed with the aid of chloroform. 25 mm was readily reached with a K-file (#020). Working length was set to 24,5 mm by apex locator and radiograph (at 25 mm) Instrumentation was completed with handfiles.



Fig. 13.4 - Working length radiograph 29.11.2012

Mechanical

- Burs, K-files, NiTi handfiles #045/.02 - 24,5 mm
- Coronal flaring; BR0/Hedström files
- Irrisafe®

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- PUI; NaOCl, 4 x 20 s
- 2% Chlorhexidine digluconate, 5 min

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms. Rubber dam/disinfection.

Removed temporary filling and calcium hydroxide dressing. Dried canal with paper points. Masterpoint radiograph. Obturation by cold lateral compaction of gutta-percha with AH Plus sealer. Temporary restoration.

Mechanical

- NiTi handfiles - #045/.02 - 24,5 mm (+0,5 mm after the masterpoint radiograph)
- Irrisafe®

Chemical

- 1% NaOCl \approx 5 ml / 17% EDTA \approx 5 ml



Fig. 13.5 - Masterpoint radiograph 09.01.2013

Obturation

Gutta-percha and AH Plus®



Fig. 13.6 - Control radiograph 09.01.2013

Temporary restoration

IRM, plug to a level 2 mm apical of mesial composite restoration.



Fig. 13.7 - Final radiograph 09.01.2013

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - favourable

A satisfactory working length was obtained, the IRM in the composite margin appeared to provide a seal.

Follow-up



Fig. 13.8 - Control - 1-year 20.02.2014

Signs of periapical healing. Some augmentation of the mesial bone and the soft tissues was achieved. A temporary crown had been placed.

Evaluation

The endodontic treatment result appeared favourable.

The apical canal was reached without complications. It was suspected that the full working length had been reached during the primary treatment, as the handfiles did not bind the canal wall before size 040-045.

Apical widening of the PDL space of 11 was not notable on all radiographs, and the tooth was without symptoms. In accordance with the patient's wish, it was decided to observe the apical status of 11.

Discussion

Sodium hypochlorite (NaOCl) is the universally most used endodontic irrigation solution[2]. Instrumentation and NaOCl irrigation may be expected to yield negative bacterial samples in 50 to 75% of infected root canals[3-5]. NaOCl is a potent disinfectant, with tissue-dissolving capacity and is minimally tissue irritating at low concentrations[6]. Among general antiseptic products, chlorhexidine digluconate (CHX) is perhaps the most widely used agent. CHX exhibits no tissue dissolving properties, but benefits as an endodontic disinfectant include antimicrobial effect against bacteria and yeast, substantivity and low toxicity[7]. Especially, *in vitro* effectiveness of CHX against *Enterococcus faecalis* have been considered a potential advantage over NaOCl, as the organism is frequently found in endodontic failures[8]. The concept of combining the two irrigants to yield more effective bacterial reduction has been explored[9, 10]. *In vitro*, Kuruvilla found a possible, but not significant, augmentation of antibacterial effect when NaOCl and CHX were combined. Zamany found reduced number of positive bacterial samples after NaOCl instrumentation and an additional rinse with CHX, as compared with saline. It should be noted that the study did not explore if it is the last *antibacterial* rinse, rather than the particular effect of CHX which is of importance for reducing positive cultures.

A standard regimen where instrumentation is performed with NaOCl, followed by a chelator, most commonly ethylenediamine tetraacetic acid (EDTA), to remove the inorganic component of the smear layer, and a final antibacterial irrigation (NaOCl or CHX) has been proposed[11].

If NaOCl and CHX solutions are mixed, an insoluble brown-red precipitate forms. Attention has been paid to the nature of the precipitate, and potential hazards of it. Adverse effects like staining and interference with the seal of root fillings are mentioned, but more concerning are reports of presence of para-chloroaniline (PCA) in the precipitate. PCA is known to have a toxic effect by converting haemoglobin to methaemoglobin, which does not bind oxygen, and can thereby cause cyanosis. In rats and mice, targeting of the haematopoietic system; anemia, extra-medullary hematopoiesis and splenomegaly is found related to PCA exposure. Carcinogenicity is also reported, with spleen sarcomas and hepatocellular carcinomas in rats and mice[12].

In a dilution series, colour change occurred when 2% CHX was combined, even with the lowest concentration of NaOCl (0,023%). A precipitate was evident with 2% CHX and NaOCl concentrations of 0,19% and higher. Mass spectrometry revealed that the precipitate consisted of PCA and other substances[12]. Nowicki casts doubt over if mass spectrometry is reliable for identifying PCA, and found no PCA, but two other degradation products of CHX after mixing the two irrigants[13].

It seems that the true composition of the precipitate is not fully understood, and current recommendations are to avoid formation of it[12-15]. Different opinions exist on how this should be practiced. It has been suggested to avoid direct combination of NaOCl and CHX by intermediately flushing the root canal with alcohol, saline, EDTA or citric acid. In the presented case, EDTA was used as an intermediate flush. When EDTA and CHX are combined, a white precipitate is formed. This mainly consists of a salt of the two compounds. Rasimick found that the precipitate was soluble, no degradation of CHX was evident and PCA was not detected[16].

In opposition to the studies advocating intermediate flushes between NaOCl and CHX, Mortenson found that flushes neither with EDTA, citric acid or saline were capable of completely eliminating PCA formation[15]. Again, one may criticise use of mass spectrometry for identification of PCA

in this study. Mortenson states that the combined use of NaOCl and CHX should strictly be avoided.

Since the case appear unresolved in regard of presence of PCA in the precipitate, the toxicological effects of PCA or other potential substances, thresholds for human tissue damage and whether the precipitate leaks out of the root canal system, more caution is practiced in combining the two.

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Case 14

Repair of a large lateral perforation in the distal root of a mandibular left first molar.

A 53-year-old Caucasian female was referred to the Department of Endodontics from the undergraduate clinic, for treatment of a root perforation the mandibular left first molar, 36. The perforation occurred during preparation for a post in the distal canal, after completion of conservative endodontic treatment.



Fig. 14.1 - Frontal view 21.03.2012

Chief complaint

The patient was informed of the procedural mishap and wished to keep 36.

Medical history

Non-contributory.

Dental history

Endodontic treatment of 36 was completed in October 2011. Perforation one month later.



Fig. 14.2 - Occlusal view 21.03.2012

Radiographic findings



Fig. 14.3 - Periapical radiograph 31.10.2011



Fig. 14.4 - Periapical radiograph 21.03.2012

CLINICAL FINDINGS

Soft tissues

Healthy pink gingival tissue.

Dental

A temporary occlusal filling sealed the access cavity of 36.

Clinical tests

Tooth	35	36	37
Cold	yes	-	yes
EPT 0-80	29		51
Palpation	no	no	no
Percussion	no	no	no
Mobility	no	no	no
PPD	wnl	wnl	wnl

Tooth 35

- radiolucent occlusal filling

Tooth 36

- widened apical PDL spaces
- radiolucency in coronal distal canal, corresponding to post preparation
- interradicular radiopacity, 3x1 mm, likely extruded root filling material

- a furcal radiolucency was not distinct, but could not be excluded

Tooth 37

- radiolucent coronal filling

Attachment apparatus

- normal periodontal conditions

Diagnosis

Iatrogenic perforation of root

Pulpal	K04.19 Root filled tooth
Periapical	K04.5 Chronic apical periodontitis (appears to be healing)
Periodontal	Normal
PAI	3

Treatment plan

- Conservative perforation repair

Problem list

- risk of contamination during post preparation
- access to extruded material
- restorable after treatment?

It was not regarded advisable to place a post in the distal root after treatment. The patient was informed of this, but wished to make an attempt on repair.

Perforation repair

Anaesthesia: Inferior alveolar nerve block:

Xylocain® Dental adrenalin 1,8 ml

Rubber dam/disinfection: 0,5% CHX in 70% ethanol.

Removed temporary filling, exposed perforation, estimated diameter 2-3 mm (Fig. 14.5). Sparse bleeding from the wound surface.

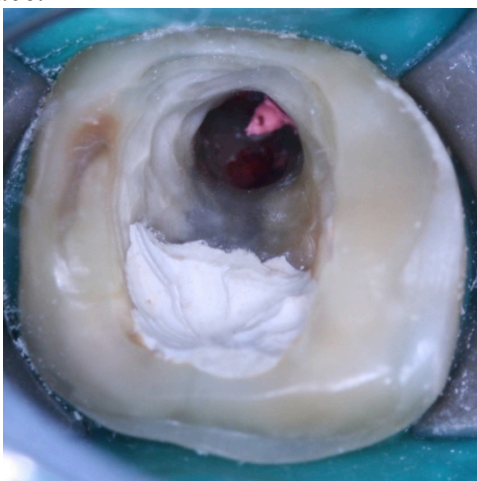


Fig. 14.5 - Perforation is exposed 21.03.2012

Gutta-percha was removed 3 mm below the perforation (Fig. 14.6). Some extruded material was visible, and removed with an endodontic probe and Hedström files.

Irrigated (NaOCl), and dried with cotton pellets and paper points. MTA was placed in the canal and condensed with light pressure, covering the perforation to a level 1 mm below the orifice (Fig. 14.7)

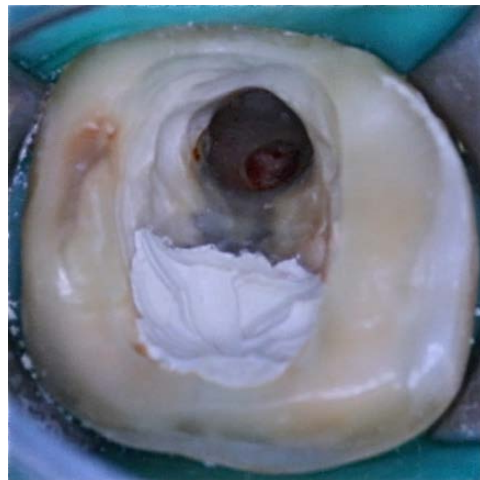


Fig. 14.6 - View before placing MTA 21.03.2012

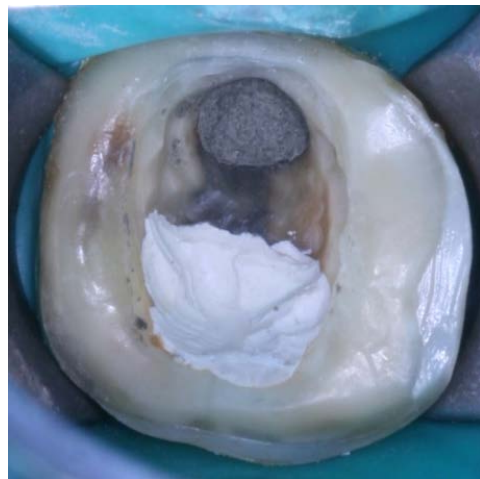


Fig. 14.7 - After repair 21.03.2012

A cotton pellet moistened with distilled water was placed over the MTA for 15 minutes and removed before placing a temporary IRM restoration.

Mechanical

- Burs, Hedström files #015-020/.02

Chemical

- 1% NaOCl ≈ 5 ml

Obturation

Grey MTA Angelus.

Temporary restoration

Sealed with IRM.



Fig. 14.8 - Final radiograph 21.03.2012

Prognosis

Endodontic - questionable
 Tooth - questionable
 Restoration - favourable

On the expectation that the perforation was contaminated and regarding the radiolucency in the adjacent bone, reservations were made on the prognosis.

Evaluation

There were no remarks neither on use of rubber dam during post preparation, or on disinfection of the perforation site, and one must suspect the perforation was contaminated. In light of this, it was questioned if more radical measures should have been made to access the periradicular area, i.e.: enlarge the perforation. Because of the further weakening it could cause, it was decided to only remove what was reachable through the perforation.

The perforation repair was uncomplicated and appeared technically satisfactory.

Follow-up



Fig. 14.10 - 2-year control 30.01.2014

A crown without a post had been made. No symptoms. Healthy periodontal tissue; 2 mm pockets on probing. The furcal radiolucency was no longer distinguishable.

Discussion

Root perforations may result from pathological processes like caries and resorptions, or procedural mishaps. In a study by Kvinnsland[1], about half of iatrogenic perforations were found to occur during endodontic treatment, post preparations accounted for the other half. Reported incidences of root perforations range between 1,5 and 10% [2, 3]

Time delay, perforation size and location have been regarded key factors for the success of perforation repair - all of which are related to risk of infection of the site[3]. In dogs, destruction of periodontal tissue occurred when aseptically induced perforations were left unsealed. A more favourable outcome was seen after immediate repair, compared to delayed treatment[4-6]. In monkeys, similar results were found[7, 8], and also signs of healing after immediate repair [9]. The importance of perforation size is derived from findings in both animal studies and human outcome studies[1]. The significance of size was rationalized with the ease of immediate repair of smaller perforations, the probability of more extensive traumatic periradicular injury, extrusion of repair material and increased risk of contaminating larger perforations, either directly or indirectly after failing to provide a complete seal [3, 10, 11]. More recent studies assess factors determining prognosis for perforation repair in a somewhat different light. Time delay or size of the perforation *per se* are not as emphasized, but rather solely the infection status at the site[12-14].

Concerning location, perforations not involving the critical zone of marginal bone and epithelial attachment, are expected to have a more favourable prognosis. Perforations at the margin of the crestal bone and gingival sulcus are at highest risk for bacterial contamination. Apical migration of the epithelium and a resulting periodontal defect involving the perforation is likely in these cases [1, 4-8]. Furcal perforations have also been considered prone to adjacent periodontal breakdown, and to represent a particular risk, as a periodontal furcation defect may evolve. In contrast to this, in monkeys, Beavers et al. found a high rate of repair for furcal perforations covered with calcium hydroxide or a teflon disc and sealed

with zinkoxide-eugenol cement and amalgam [9]. The favourable results were attributed to controlled aseptic operative procedures and a bacteria tight seal over the access cavity.

Application of an antibacterial dressing to the perforation site does not seem to improve healing of an infected perforation[15].

A wide range of materials have been applied for perforation repair; gutta-percha, amalgam, ZnO-eugenol, Cavit, composite resins, glass ionomer cement and more [1, 3]. Another approach is internal matrix materials (like hydroxyapatite, freeze dried bone, tricalcium phosphate or plaster of Paris), aiming to induce osteogenesis and cementogenesis in the defect while the material itself is resorbed[16]. After introduction of MTA, it has become the material of choice for subcrestal perforation repair. The biocompatibility, sealing ability and favourable setting in presence of moisture are important characteristics in this respect[14].

In four retrospective studies of MTA perforation repair, including 16-90 teeth, 73-100% healing was reported[12-14, 17]. The most prominent prognostic factors were location of the perforation, presence of a radiographic lesion at the site and coronal leakage. Among factors failing to reach significant levels, were time between perforation and repair, perforation size, preoperative symptoms, preoperative pulp status, tooth type/location, and coronal restoration type.

In the current case, a favourable outcome was achieved despite the suspicion of contamination. If conservative perforation repair is unsuccessful, a surgical approach may be attempted on accessible defects. A root resection might have been an option in this case.

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Case 15

Treatment of a mandibular right first molar; retreatment of the distal root, hemisection and surgical removal of the mesial root.

A 53-year-old female was referred to the Department of Endodontics from the undergraduate clinic, for evaluation and endodontic treatment of the mandibular right first molar, 46.



Fig. 15.1 - Frontal view 16.11.2011

Chief complaint

Patient expressed wishes for a new coronal restoration on 46. No symptoms.



Fig. 15.2 - Occlusal view 16.11.2011

Medical history

Non-contributory.

Dental history

Previous endodontic treatment and crown on 46, the crown was lost 2 years earlier.

47 was lost years earlier



Fig. 15.3 - Buccal view 16.11.2011

Clinical findings

Soft tissues

Normal findings. Adequate oral hygiene, healthy gingival tissue.

Dental

Coronal restoration of 46 was missing, discoloured dentine and root canals exposed to the oral cavity.

Supra-eruption of 17

Clinical tests

Tooth	46	45
Cold	-	yes
EPT 0-80	-	29
Palpation	no	no
Percussion	no	no
Mobility	no	no
PPD	wnl	wnl

Radiographic findings



Fig. 15.4 - Periapical radiograph - 16.11.2011

Tooth 46

- relatively short roots (distal root <10 mm)
- exposed root filling
- caries in mesial orifices
- inadequate root filling, short;
2,5 and 5 mm M
4-5 mm D

- radiolucencies; mesial root (2x4 mm) and interradicular (2 mm diam)

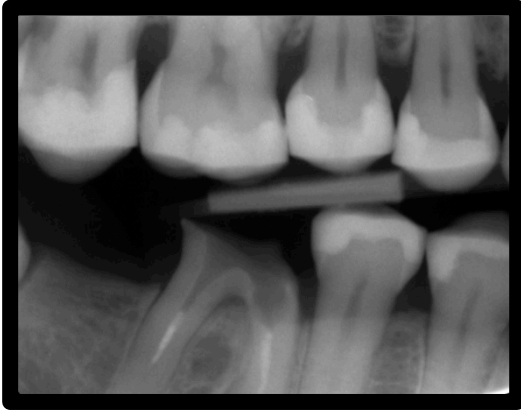


Fig. 15.5 - Bitewing 16.11.2011

Tooth 47

- lost, several years ago

Tooth 48

- impacted

Premolars

- minor composite fillings

Attachment apparatus

- slight bone loss, over 2/3 attachment left

Diagnosis

Pulpal K04.19 Root filled tooth

Periapical K04.5 Chronic apical periodontitis

Periodontal Normal

PAI 4 (mesial root)
2 (distal root)

Possible apical resorption, rounded distal root end, irregular outline mesial root.

Treatment alternatives

Several options were discussed:

- keep both roots
- resection mesial root
- complete removal of 46

Specialists in prosthodontics were consulted regarding the possibilities for restoring 46. A conventional post-retained crown, hemisection/premolarization of both roots, or to retain only the distal root were discussed. After caries excavation of the mesial cervical area and orifices, the mesial root was deemed to be impaired to retain a restoration. Extraction, with or without implant replacement of 46, was also suggested. The patient did not want implant treatment. After the initial examination and evaluation, the patient was undecided. The canals and access cavity was covered with IRM.

After a few weeks, she came back, determined to treat 46.

Final treatment plan

- 1 Conservative endodontic retreatment of distal canal.
- 2 Hemisection and resection of mesial root.

Problem list

Distal root

- apical obliteration? Canal not visible
- restorable after treatment?

Mesial root

- cervical caries and wide apical circumference might complicate removal of the root

These factors would be addressed in the course of the treatment.

Treatment progress

16.11.2011

- Clinical and radiographic examination.
- Interdisciplinary evaluation.
- Presented treatment options.

24.01.2012

- Treatment plan decided.
- Instrumentation distal canals.

06.03.2012

- Obturation distal root.
- Hemisection/surgical removal of mesial root

14.03.2012/22.03.2013

- Controls

Treatment - first visit

Rubber dam/disinfection.

Old root filling was removed by handfiles, desired working length obtained and set by apex locator and radiograph.



Fig. 15.6 - Working length radiograph 24.01.2012

Mechanical

- Burs, BioRaCe, K-files, NiTi handfiles
050/.02 DB 14mm
DL 13,5mm
- Irrisafe®

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- PUI, NaOCl, 3 x 20 s.
- 2% Chlorhexidine digluconate, 3 min.

Intracanal medicament

- Ca(OH)₂

Temporary filling

- IRM.

Treatment - second visit

No symptoms.

Rubber dam/disinfection

Removed temporary filling and calcium hydroxide dressing. Dried canals with paper points. Masterpoint radiograph. Obturation by cold lateral compaction. IRM.

Mechanical

- Burs, K-files, Hedström files, NiTi handfiles

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus®

Temporary restoration

IRM, 3 mm plugs in coronal canal.

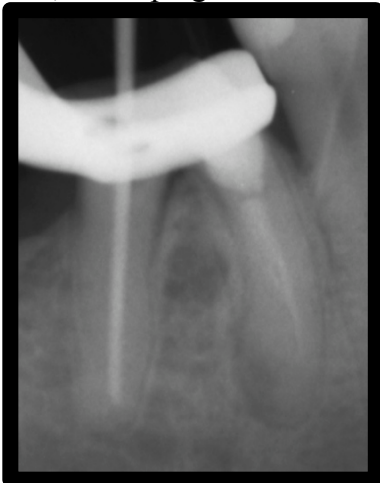


Fig. 15.7 - Masterpoint radiograph 06.03.2012



Fig. 15.8 - Final radiograph 06.03.2012

Hemisection/resection mesial root

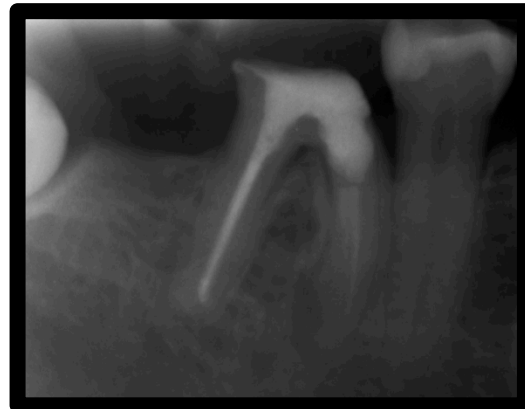


Fig. 15.9 - Final radiograph 06.03.2012

Anaesthesia: Infiltration and inferior alveolar nerve block, Xylocaine 2x1,8 ml.

Hemisection

- Diamond and tungsten carbide burs
- Controlled by bite-wing radiograph



Fig. 15.10 - Control during sectioning 06.03.2013



Fig. 15.11 - Hemisection 06.03.2012

Luxation

- Root elevators

The crown and cervical root fractured during luxation. The apical root fragment was removed surgically:

Incision

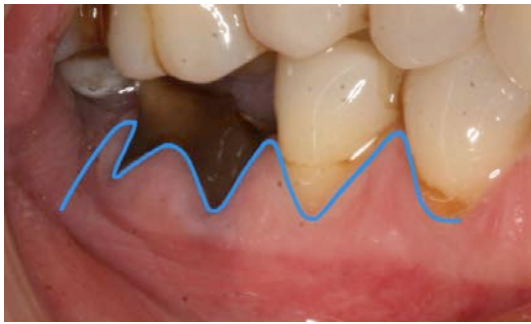


Fig. 15.12 - Incision 06.03.2012

Sulcular incision from buccal aspect of 44 to the distal margin of 46. 10 mm vertical releasing incision distal to 46. Elevation of the mucoperiosteum.

Removal of mesial root

Removal of buccal bone - round carbide burs, with external saline irrigation. Luxation and removal of fragmented root. 1 interdental and 3 simple interrupted sutures.

Postoperative information

Possible pain and swelling. Hygienic measures. Recommended pain management; ibuprofen and paracetamol.

Post operative control

Patient reported considerable post-operative pain. Intense pain on first days after surgery, decreased the next few days, to increase again. Patient experienced malodour and radiating pain from regio 46, also pain on gentle palpation of right chin.

Normal healing of surgical field, normal coloured gingiva, no swelling.

Diagnosis: K10.3 Alveolitis - jaw

Removed 3 remaining sutures.

Prescribed 400 mg ibuprofen and 500 mg paracetamol for the following days.



Fig. 15.13 -Post operative radiograph 14.03.2012

Post operative control

Significantly reduced pain, but still some discomfort. Pain on chewing and intake of hot and cold foods and drinks. The cervical distal root surface of 45 was exposed.

Applied Duraphat® on root surface of 45. The patient was instructed to contact the Dept. of Endodontics if pain did not subside.

Prognosis

Endodontic - favourable

Tooth - favourable

Restoration - favourable

The prognosis for the endodontic treatment was deemed favourable. The Dept. of prosthodontics regarded the prognosis sufficient to make a ceramic crown on the distal root, supported by an onlay on 45.

Follow-up

After two years, there were no signs of periapical pathology, signs of healing of the mesial alveolus. 45 was sensible to a cold stimulus. The patient perceived the restoration as well functioning.



Fig. 15.14 - Ceramic restoration 13.03.2013



Fig. 15.16 -Follow-up 20.02.2014

Evaluation

The retreatment of the distal root was uncomplicated and appeared technically satisfactory. Healthy periapical conditions were seen at follow-up.

After the surgical treatment, the patient had a significant amount of pain. The total time spent on removing the mesial root (initial luxation and surgical removal of the apical fragment) was approximately 2 hours. In retrospect, the surgical procedure should have been prepared and initiated earlier, as the preoperative radiographs indicated the root morphology might complicate removal.

Discussion

Root resection techniques were first described in detail by Farrar as early as in 1884[1]. Indications for hemisection or root resection may be localized loss of periodontal attachment, furcation involvement, vertical fractures, endodontic failures and limited loss of tooth substance caused by caries, resorptions or iatrogenic damage. Contraindications are fused roots, deep furcations, and inability to restore or obtain successful endodontic treatment of remaining roots[2-5]. Root canal treatment is normally performed prior to sectioning. This promotes isolation during endodontic therapy and assessment of intracanal anatomy before separation, as root fusions or C-shaped canals are not always evident on preoperative radiographs[5].

In the last decades, the practice of retaining one or more separate roots of multirooted teeth has been challenged by the reported higher success rate of osseointegrated implants[3].

Langer found that the majority of failures of root resected teeth occurred between 5-7

years after the procedure[6]. 38 of 100 teeth had failed after 10 years, within 5 years the failure rate was 16%. Comparable results after 10 years (32% failure of 28 teeth) was recorded by Bühler[7]. A sufficient follow-up therefore seems essential to evaluate outcome. The quality of endodontic treatment or restorations was not assessed in these studies, thus the preconditions of the relatively limited numbers of included teeth might be quite inconsistent. This is a point emphasized in a later comment by Langer, refuting that the material from his 1981 study should be regarded a justification to delete root resections as an acceptable procedure[8]. More favourable outcomes are also recorded: A 93% ten-year survival of 72 root resected furcation involved molars was found by Carnevale[9].

Endodontic failures accounted for 19-56% of extractions of root-resected teeth, periodontal complications 21-26% and fractures 10-47%[6, 7, 9].

In recent years, comparable (and higher) survival rates of root resection therapy and implant treatment have been recorded. Fugazzotto found success rates of 96,8% in root resected molars and 97% in molar implants after 15 plus years[10]. The lowest success for both modalities was seen with lone-standing terminal teeth. Only 1 in 160 distal roots of mandibular first molars in fixed prosthesis failed (99,4% success). Another study found higher complication rates, and reduced survival in hemisectioned mandibular molars (80% survival), as compared with implants (97%)[2].

A 1998 review comparing success of root resections and posterior single implants found added success rates of 84% and 96%, respectively[4]. It should be noted that observation periods were pronouncedly shorter in the implant studies. The authors stresses the requirements for equally high levels of expertise in the endodontic, periodontal, prosthodontic and restorative disciplines to secure favourable outcomes of root resection therapy. Another facet of this, is the large number of variables affecting prognosis of resection and comparison of resected teeth or different studies.

For this patient an implant was not an option at the time, and the treatment allowed

retaining molar occlusion on her right side. Reviewing the literature, reservation on the long-term prognosis may be appropriate. But thus far, supraeruption of 16 and further vertical bone loss in the posterior right mandible was prevented.

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Case 16

Conservative retreatment and surgical endodontic treatment of a mandibular left first molar with persistent apical periodontitis and an intracanal post.

A 64-year-old male was referred to the Department of Endodontics from the undergraduate clinic, for endodontic retreatment of the mandibular left first molar, 36.



Fig. 16.1 - Frontal view 10.10.2012

Chief complaint

Patient presented for treatment of infection on 36. No symptoms.

Medical history

Type II diabetes mellitus

Coronary stent

Psoriasis

Medicines

Glucobay	<i>antidiabetic</i>
Acetyratio	<i>thrombosis prophylaxis</i>
Selo-Zok	<i>beta-blocker</i>
Methotrexate	<i>chemotherapeutic/ immunosuppressant</i>
Omnic	<i>relaxes prostate and urinary muscles</i>

Dental history

Patient at the undergraduate clinic since 2008. Root filling and crown on 36 several years before this. 37 was recently extracted because of an extensive coronal carious lesion.



Fig. 16.2 - Occlusal view 10.10.2012

Clinical findings

Soft tissues

Gingival hyperkeratosis on posterior gingiva in all four quadrants.

Dental

Posterior full crowns and fillings.

Clinical tests

Tooth	34	35	36
Cold	yes	yes	-
EPT 0-80	21	23	-
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

Radiographic findings

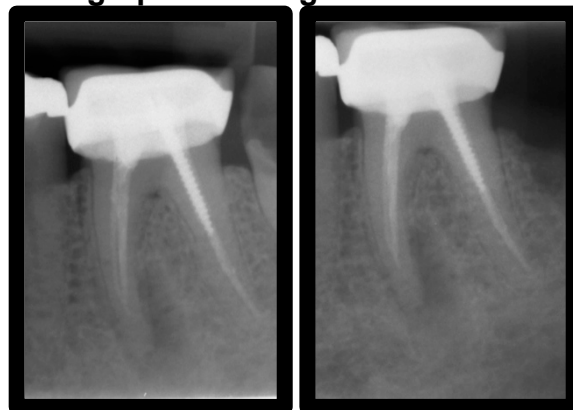


Fig. 16.3 - Periapical radiographs, *left* 27.02.2012 and *right* 10.10.2012

Tooth 36

- ceramic-fused-to-metal crown
- root filled, voids and low radio contrast, suspected due to lack of sealer.
- distal root: Prefabricated intracanal post. Visible apical lamina dura.
- mesial root: Root filling 4 mm short of apex. Apical radiolucency, 4x7 mm. Apical root canals not distinguishable.

Tooth 35

- distoocclusal radiolucent filling
- normal periapical findings

Attachment apparatus

- over 2/3-bone attachment left.

Diagnosis

Pulpal K04.19 Root filled tooth

Periapical K04.5 Chronic apical periodontitis

Periodontal K05.3 Chronic marginal periodontitis

PAI 3

Treatment plan

- Conservative endodontic retreatment

Treatment alternative

- Periapical surgery

As the root filling was regarded inadequate, a conservative retreatment was the first choice in this case.

Problem list

- Access to apical canal in mesial root
 - Canals not visible on radiograph
 - Possibility of ledges
- Post removal
 - Conserve dentine and crown
 - Retention loss for restoration?

Apical gutta-percha was planned removed with aid of solvent if intended working lengths were not readily reached. The patient was informed that retention for the existing crown could be lost after endodontic treatment, and a new crown might be needed.

Treatment progress

10.10.2012

- Examination and treatment planning
- Post removal
- Instrumentation

20.11.2012

- Obturation

23.10.2013

- Follow-up

19.02.2014

- Apicectomy mesial root

26.02.2014

- Postoperative control

Treatment - first visit

Rubber dam/disinfection.

Access cavity preparation. Exposed mesial canals and distal post exposed. Discoloured cement surrounding the post. Mobilization of

post by removal of the cement with LN burs and activation with an ultrasonic tip.

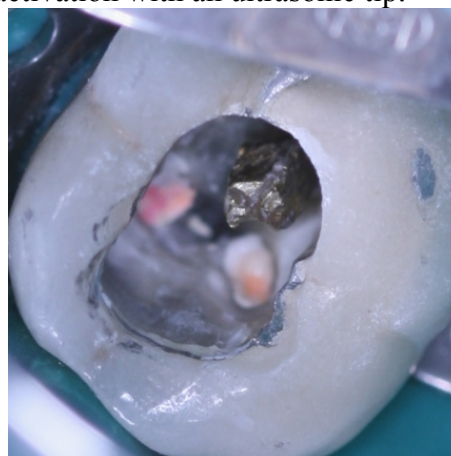


Fig. 16.4 - Post exposed 10.10.2012

Apical canals were reached with small handfiles. Solvent was not used. The working length of the distal canal was advanced with 0,5-1 mm, +3 mm in mesial canals, which were joined apically.

Instrumentation with handfiles to size 020, finished with BioRaCe rotary files. Passive ultrasonic irrigation. Intracanal medicament and temporary filling



Fig. 16.5 - Working length radiograph 10.10.2012

Mechanical

- Burs, ultrasonic tips, K-files, BioRaCe

MB 040/.04 - 21 mm

ML 040/.04 - 19 mm

D 060/.04 - 20 mm

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA
- PUI; NaOCl, 20s/canal.
- 2% Chlorhexidine digluconate, 5 min.

Intracanal medicament

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Treatment - second visit

No symptoms.

Rubber dam/disinfection.

Removed temporary filling and calcium hydroxide dressing. Masterpoint radiograph (lost length in mesial canals were corrected).

Obturation with cold lateral compaction.

Mechanical

- Burs, NiTi handfiles
- Irrisafe®

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA

Obturation

Gutta-percha and AH Plus®

3 mm IRM plugs in canal orifices.



Fig. 16.6 - Masterpoint radiograph 20.11.2012



Fig. 16.7 - Final radiograph 20.11.2012

Prognosis

- Endodontic - favourable
- Tooth - favourable
- Restoration - questionable

If retention of the crown relied on the distal post, there was a risk of mobilization of the crown.

Follow-up, 1 year

No clinical symptoms.



Fig. 16.8 - 1-year control 23.10.2013

The apical radiolucency was unchanged. Still IRM in the access cavity. The patient was informed that a permanent seal should be made.

Treatment plan 2

- Surgical endodontic treatment; Apicectomy and retrograde filling of mesial root.

Problem list

- General health. The patient consulted his physician regarding contraindications for surgery. It was stated that apical surgery could be performed without special considerations.
- Haemostasis during and after surgery. The patient was prescribed daily acetylsalicylic acid. It was decided that the medication should not be seponated. If problems with haemostasis occurred, this would be addressed clinically during surgery.
- Relation to the mental nerve? The patient was referred to the Department of Maxillofacial Radiology for an orthopantogram.

Radiographic findings - opg



Fig. 16.9 - Orthopantogram 19.02.2014

Foramen mentale and the mandibular canal could be seen, minimal distance to the periapical lesion was 6 mm. Exposure and protection of the mental nerve was planned during surgery.

Surgical treatment

Preoperative information. Chlorhexidine mouth rinse

Anaesthesia

Inferior alveolar nerve block and infiltration: Xylocain 1,8 ml x3.

Incision

A sulcular incision was made from the mesial margin of 34 to the distal aspect of 36, with a 15 mm vertical releasing incision at the mesial margin of 34. Elevation of triangular mucosal-periosteal flap. The mental nerve was localized, and an oblique groove was prepared in the cortical bone immediately superior/posterior to the mental foramen to provide a stop for positioning of the surgical retractor. Sterile saline irrigation

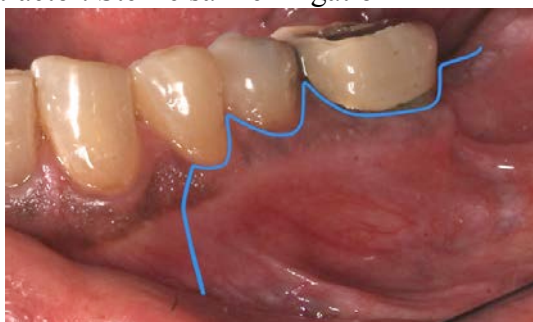


Fig. 16.10 - Incision 19.02.2014

Osseous access

The apical lesion was exposed with a round steel bur. Care was taken to keep distance to the mental foramen; the preparation was aimed at the central part of the lesion, and extended towards its inferior margin after localization of the mesial root and lesion.

Apicectomy

The apical 3 mm of the root end was resected horizontally with a cylindrical steel bur. The root end and apical lesion was removed with a surgical curette, and prepared for histological examination.

Retrograde preparation and filling

Root end and two canals with a connecting isthmus were visualized by methylene blue staining. No fracture lines were noted.

A retrograde canal preparation was made with a 3 mm ultrasonic retrograde tip. Controlled by radiograph.



Fig. 16.11 - Control radiograph 19.02.2014

Haemostasis was achieved by compressing cotton pellets with ferric sulphate in the bony crypt. The retrograde preparation was filled with ProRoot MTA. A control radiograph was exposed before removal of excess MTA, and wound toilette.



Fig. 16.12 - After placing of MTA 19.02.2014

Sutures

The flap was repositioned, compressed with moist gauze. 5 simple interrupted sutures and 2 interdental sutures (Supramid 5-0, 3/8c, 19 mm and 12 mm) were used.



Fig. 13.13 - Sutures 19.02.2014

Postoperative care

The haemostasis was satisfactory. Ice pack for 15 minutes. Standard postoperative

information, with emphasis on hygiene measures, pain control and suspected postoperative discomfort.

Histological findings

Soft tissue with chronic inflammation.

(Histological diagnosis by Anne Kristin Goplen)

Postoperative control, day 7

Patient reported very little post-operative discomfort and swelling. He described some hypersensitivity on touch of skin of left lower chin. Hyperesthesia on light probing of the skin corresponding to the innervation of the mental nerve. Very clean and healthy oral tissues, satisfactory healing. Suture removal.



Fig. 13.14 - Control 19.02.2014

Prognosis

Endodontic - favourable
Tooth - favourable
Restoration - favourable

Evaluation

Healing of the periapical lesion was not achieved after primary treatment. The surgical option could have been chosen initially, but it was suspected that improving the technical quality of the root filling would lead to a favourable result.

A composite filling had been placed in the access cavity between the 1-year control and surgical procedure. From the intraoperative

radiographs, it appeared that a post was placed in the distal canal. The preparation is close to the mesial surface of the distal root, but no evidence of perforation was seen.

The hyperesthesia experienced after surgery was likely caused by trauma to the mental nerve resulting from pressure by the surgical retractor and/or stretching of the flap. The releasing incision could have been placed posterior to 36, this may have caused less strain on the anterior soft tissue. This was discussed preoperatively. The chosen incision was thought to allow overview and control over the mental foramen and nerve, but in retrospect, a posterior release may have been preferred.

Discussion

When a periapical lesion is present, the success rate of conservative retreatment is in the lower range (58-74%)[1-4]. In this patient, a technical satisfactory result was obtained in increasing the working lengths, but as demonstrated by Sjögren[1], the length of the root filling is not a strong predictor for success in retreatment cases.

The principal cause for refractory periapical disease is persisting intraradicular or extraradicular microorganisms[5]. Failure of eliminating intraradicular microorganisms may be due to insufficient removal of infectious material, or bacterial tolerance towards antimicrobial agents. Extraradicular microorganisms have been found both on root surfaces and in periapical lesions [6, 7]. Root canals are typically colonized by bacteria in patterns of oral biofilm[8]. Bacterial biofilm can be seen the main canal, ramifications and isthmuses, but also extraradicularly in some cases[9]. The community offered in a biofilm is one of the most successful strategies for bacterial adaptation to their environment, for evading host defence or antibacterial treatment. The bacteria of biofilms are embedded in self-produced extracellular polymeric substance (EPS), which constitutes 80-85% of biofilm volume[10]. The EPS maintains biofilm integrity, prevents desiccation and acts as a diffusion barrier, thereby limiting pervasion of antibiotics and disinfectants[11, 12]. Cells in deeper, oxygen and nutrition-deprived layers exhibit a reduced growth rate, reducing efficacy of antibiotics targeting DNA replication and translation. 100-1000 folds increased

tolerance against antibiotics have been seen in biofilms compared with planktonic bacteria [11]. Signalling substances and systems in biofilms resembles those found in multicellular organisms. The biofilm community functions across species, and by 'quorum sensing' bacteria communicate and gene expression can be regulated. The close interaction of bacteria also facilitates genetic exchange, contributing to pathogenicity.

Biofilms create broader habitats, with growth conditions for a wider range of microorganisms[10]. Most biofilms are polymicrobial, but even monospecies biofilms distributes phenotypic heterogeneity[11].

Bacteria in biofilms appear to elicit a lower-grade response from the host defence, representing a state of chronic infection. However, the biofilm may act as a reservoir for planktonic bacteria - phenotypically rapidly growing, more mobile and expressing enzymes and toxins provoking an aggressive host response, characteristic of acute infection[10].

The pathogenesis of apical periodontitis appears more complex than allowed by the earlier preferred reductionist approach, attributing disease to key 'causative pathogens'[10, 11]. The biofilm model offers an understanding of the pathogenesis resonating with the diverse microbial findings of root canals and apical lesions. As biofilms exhibit inherent resistance against antimicrobial measures, the treatment of biofilm infections appears to a large extent to depend on physical removal of the biofilm; instrumentation of the root canal, or surgical removal of apical ramifications or extraradicular lesions.

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Case 17

Apicectomy of a maxillary right canine with a sinus tract

A 66-year-old male was referred to the Section of Endodontics from the undergraduate clinic, for surgical endodontic treatment of the maxillary right canine, 13.



Fig. 17.1 - Frontal view 30.04.2013

Chief complaint

Patient presented for treatment of a persisting sinus tract on the buccal gingiva of regio 13, 10 months after conservative endodontic treatment at the undergraduate clinic.

Medical history

Periodic high blood pressure
Prostate cancer (years ago, patient was fully recovered)

Medicines

No prescribed medications.
Daily omega-3 supplements.

Dental history

An old overdenture replaced missing maxillary teeth. Retention was failing after extraction of 14 and cervical fracture of 13. A new overdenture was planned and 13 was to serve as attachment.



Fig. 17.2 Occlusal view without overdenture 30.04.2013

Clinical findings

Soft tissues

Gingival inflammation/bleeding on probing. Poor oral hygiene. Inadequately supported overdenture retained bacterial plaque and debris.

Sinus tract on buccal mucosa in regio 13.

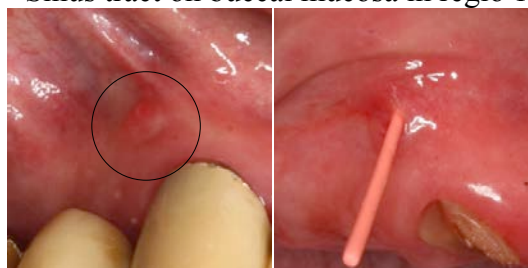


Fig. 17.3 - Sinus tract 30.04.2013

Dental

Cervical fracture of 13 was fractured. IRM seal covers the root filling. Telescopic crowns on left maxillary canine and first premolar.

Clinical tests

Tooth	13	23	24
Cold	-	yes	yes
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

Radiographic findings

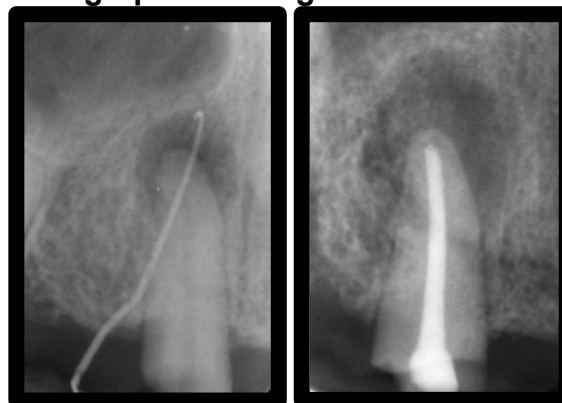


Fig. 17.4 - Undergraduate's preoperative radiograph *left* and nine months postoperative control *right*, 17.04.2012 and 20.03.2013



Fig. 17.5 - Tracing of the persisting sinus tract
30.04.2013

Tooth 13

- root filled, 0,5 mm short of apex
- apical radiolucency, 11 mm diam, increased after initial treatment
- sinus tract originating in radiolucency
- IRM, 3 mm plug covers root filling

Remaining dentition

- all maxillary molars and incisors lost

Attachment apparatus

- localized periodontal attachment loss, 15 and 34 (not shown)

Diagnosis

Pulpal	K04.19 Root filled tooth
Periapical	K04.5 Chronic apical periodontitis K04.62 Sinus tract to the oral cavity
Periodontal	K05.0 Acute gingivitis K05.3 - Chronic marginal periodontitis
PAI	4

Treatment plan

- Surgical endodontic treatment: Apicectomy and retrograde filling 13.

Treatment alternatives

- Extraction. Dept. of prosthodontics deemed root length sufficient for restoration.
- Conservative retreatment. Discarded as technical quality of existing root filling and coronal seal appeared adequate.

Problem list

- Retain sufficient root length for later post and telescopic coping on 13.
- Soft tissue healing after surgery. Inflamed gingival tissues

The patient understood the uncertainties concerning prognosis, but wished to undergo treatment.

Treatment progress

30.04.2013

- Examination and treatment planning
- Preoperative information

15.05.2013

- Apicectomy and retrograde filling 13
- Postoperative information

22.05.2013

- Postoperative control

25.09.2013

- Postoperative control

Surgical treatment

Preoperative information. Chlorhexidine mouth rinse

Anaesthesia

Infiltration: Xylocain 1,8 ml x2.

Incision

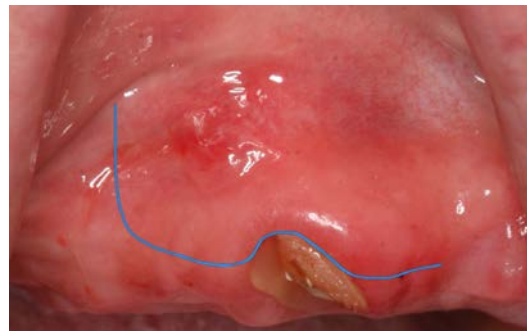
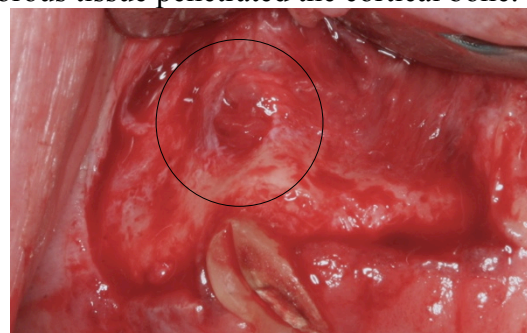


Fig. 17.6 - Incision, illustration 15.05.2013

Continuous incision on the alveolar ridge from regio 11 to 14, sulcular incision buccal to 13. 10 mm vertical release regio 14/15. Elevation of triangular mucosal-periosteal flap. A pathological process covered by fibrous tissue penetrated the cortical bone.



Fi g. 17.7 - Penetrating pathological process
15.05.2013

Apical curettage/removal of lesion

The apical lesion was removed *in toto* with a surgical curette and pliers, and prepared for histological examination in 4% formaldehyde.

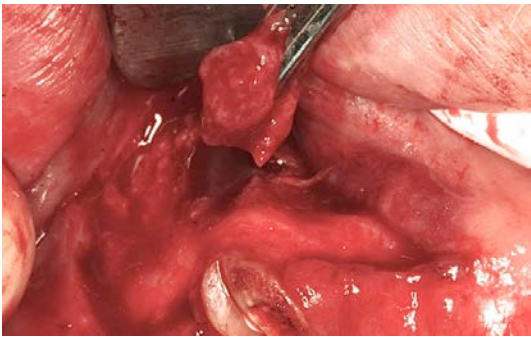


Fig. 17.8 - Removal of apical lesion 15.05.2013

Osseous access

Refined with round bur in a low-speed handpiece. External saline irrigation.

Apicectomy

Horizontal resection of 2-3 mm of root-end.

Retrograde preparation and filling

Ultrasonic retrograde preparation. Haemostasis; cotton pellets with ferric sulphate packed in the crypt. Filled with ProRoot MTA. Control radiograph, removed MTA excess, wound toilette.

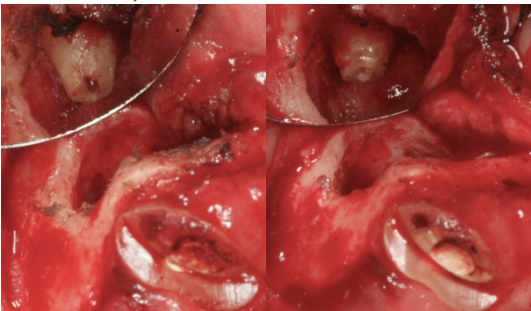


Fig. 17.9 - Root end after retrograde preparation *left* and placement of MTA *right* 15.05.2013



Fig. 17.10 - Control MTA placement 15.05.2013

Sutures

Flap repositioned and compressed with moist gauze. 2 continuous and 1 simple interrupted suture (Supramid 4-0, 3/8c, 12 mm).

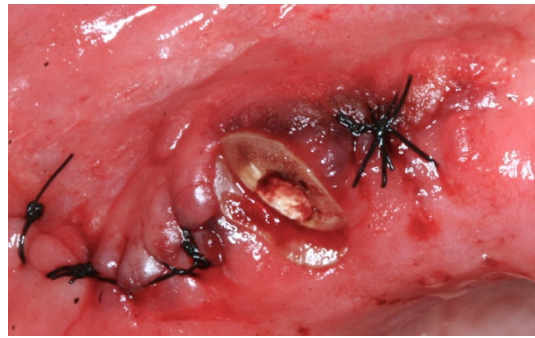


Fig. 17.11 - Sutures 15.05.2013

Postoperative care

Satisfactory haemostasis. Ice pack for 10-15 minutes. 600 mg ibuprofen immediately postoperatively. Standard postoperative information, special emphasis on hygiene measures for the overdenture.

Histological findings

Diagnosis: Partly epithelial lined cyst wall with chronic to acute inflammation, consistent with an infected radicular cyst.

(Histological diagnosis by Tore Solheim)

Postoperative control, day 7

Minor post-operative swelling and discomfort. Satisfactory soft tissue healing, less inflamed than preoperatively. Suture removal.

The overdenture was relined with Kerr F.I.T.T.® in the regio 13 to 15 where two telescopic crowns now lacked support and was retaining debris.

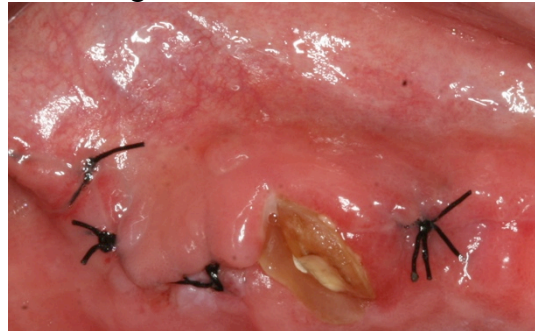


Fig. 17.12 - Control 22.05.2013

Post operative control, 4 months

Patient experiences no symptoms. Sinus tract was healed. Radiographic signs of initial healing.



Fig. 17.13 - Control 25.09.2013

Follow-up

Radiographic evidence of continued healing. 13 falls into Rud's complete healing category; with signs of bone fill of the cavity.

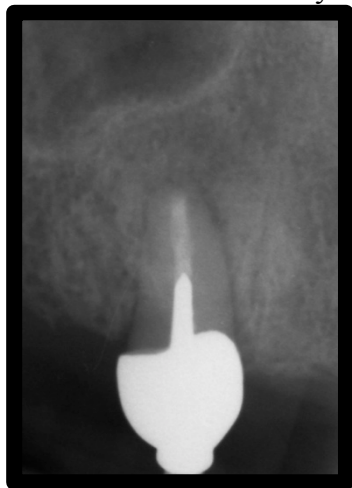


Fig. 17.14 - Control 10.04.2014

Prognosis

Endodontic - favourable
Tooth - favourable - questionable
Restoration - favourable - questionable
Questions can be asked regarding prognosis for 13, considering root length, and load from

the new overdenture. It was expected to serve as an attachment at least during adaptation to a new denture.

Evaluation

Apparently, the sinus tract persisted for nine months before referral for apical surgery. The failure might have been identified, and surgical treatment initiated, earlier.

More emphasis could have been placed on the inflamed soft tissues preoperatively. The patient responded well to information. Preoperative relining was considered, but not performed, fearing it could inflict undue pressure to the wound surface. In retrospect, an initial relining with relieve of the surgical area, might have promoted hygiene.

Another consideration that could be made, with sparse coronal dentine and a planned prosthodontic restoration, is a crown lengthening procedure during the endodontic surgery. This was not discussed.

Discussion

The rationale for root-end resection is removal of infectious agents, and to provide access for inspection and management of the root end. In addition to bacteria residing in apical canals, deltas or isthmi, biofilm has been found in irregularities, crypts and holes of root surfaces of teeth with apical lesions[1]. Approximately 75% of teeth have canal irregularities such as accessory or lateral canals in the apical 3 mm[2]. 98% of apical ramifications and 93% of the lateral canals have been found in the apical 3 mm of roots, hence is a 3 mm resection considered adequate[3]. In this patient, the root end appeared rounded, which may be attributed to apical resorption. One can hypothesize that resorption may have shortened the area with apical ramifications, and that removal of 2-3 mm may be deemed sufficient. Regardless of this, if conservation of root length was not as important, resection of a minimum of 3 mm would be the procedure of choice.

In her study of human upper incisors, Brynolf found that histological and radiographical features of apical periodontitis or periapical health could be correlated[4]. After apical surgery, a radiographic rarefaction may represent different states; a stage in bone regeneration; a fibrous scar; or inflammatory changes. In a series of studies published in 1972, Rud and Andreasen described

histological modes of healing after apical surgery, correlated histologic and radiographic features, formulated radiographic criteria of healing, and applied the criteria to 1000 surgery cases to determine long-term development of the different healing groups[5-8].

70 bloc biopsies, including root apices, periodontal structures and bone, was removed *ad modum* Nygaard-Østby, 1-14 years after periapical surgery[6]. The majority of biopsies were retrieved from suspected unsuccessful cases. Based on correlation of histology and radiographs, a classification system with four groups was proposed[8]:

Group 1: Complete healing

Radiographs: An apical PDL space less than twice the normal width, but it may also be indiscernible. Intact lamina dura (allowing a 1 mm² defect near filling material). Evidence of healing of the bone cavity, although its radiopacity might differ from surrounding bone. There was a 95% agreement between different observers in placing findings in this category.

The histological examination of this group revealed few cases with inflammation (24% with moderate - severe inflammation)

Group 2: Incomplete healing (scar tissue)

Radiographs: Decreased or stationary rarefaction, with or without central bone structure; fine meshed or trabecular bone. An irregular periphery, which may be demarcated. The location of the rarefaction is often asymmetric or at an angular position in relation to the apex. An apical lamina dura may form in time, isolating the radiolucency from the root.

Histologically, 61 % had signs of inflammation (moderate to severe).

Group 3: Uncertain healing

Radiographs: Decreased rarefaction, with circular or semi-circular periphery, which may be surrounded by lamina dura-like bone. Lamina dura close to the apex may have a collar-shaped increase, and the rarefaction is symmetric or funnel shaped. Surrounding bone structure is usually normal. This was the group with lowest agreement for the three observers (75%).

Histology: Severe inflammation in 86%. Scar tissue was relatively rare.

Group 4: Unsatisfactory healing

Radiographical features were similar to the uncertain healing-group, but size of rarefaction was unchanged or enlarged.

Histology: Severe inflammation in all cases.

The 1000 patient follow-up[5], radiographically categorized patients according to the groups after one year. On further follow-up, it was disclosed that *Healed* or *Unsatisfactory healing* were stable results. However, within four years, the *Uncertain* or *Unsatisfactory* cases were fewer, some evolving into the 'success' or 'failure' categories. Few changes were seen after four years. Based on this, a proposition was made to dismiss *Healed* cases after one year. Whenever radiographical findings fell under the *Unsatisfactory* category, they should be reoperated, as should cases still *Uncertain* after four years. The recommendation for the *Incomplete healing* category was to perform periodic controls after four years, and consider reoperation or extraction whenever clinical signs of inflammation were evident.

Based on these studies, Molven proposed an observer strategy for the evaluation of healing after apical surgery[9]. 93% agreement was achieved after joint evaluation of deviating cases. A valuable contribution from this study is drawings illustrating the hallmarks of the healing categories, which were of help in obtaining agreement during evaluation. The group also followed 23 patients with *Uncertain* healing for 8-12 years after surgery[10]. With only one patient failing, the conclusion was that these patients did not need systematic controls beyond one year. Thus, only the *Uncertain* group was considered in need of follow-up after one year[11].

In recent years, it has been made clear that even if healing is observed after periapical surgery, 'late failures' evolve in some cases. A randomized clinical study by Kvist and Reit, assigned cases failed after primary endodontic treatment to either conservative or surgical retreatment[12]. The healing rate after one year was higher for patients undergoing surgery, but after four years, healing rates were not significantly different in the two groups. Surgical removal of an apical lesion likely promotes immediate healing and bone-fill, but disease may recur. In the majority of

cases included in Rud and Andreasen's studies, ortograde retreatment preceded surgery. This may account for few relapses of disease.

A systematic review of outcome of conservative and surgical retreatment, was even less favourable in regard of long-term outcome of surgery [13]. After 2-4 years, surgery yielded the highest success rate, but at 4-6 years, the relationship was reversed with 83% success for nonsurgical retreatment, and 72% for surgery.

A sufficient follow-up period is important both in the clinical setting, to provide care for failing cases, and for studies evaluating outcome of different treatment modalities.

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Case 18

Surgical treatment of a mandibular right second premolar with persistent apical periodontitis.

A 65-year-old male was referred to The Department of Endodontics from a general practitioner in private practice; for evaluation and treatment of a root filled mandibular right second premolar, 45. The referral also included other teeth.



Fig. 18.1 - Frontal view 19.04.2012

Chief complaint

No symptoms, the patient complied with the referral.

MEDICAL HISTORY

Elevated blood pressure (controlled)

Benign prostate hyperplasia

Medicines

Cozaar comp *angiotensin receptor blocker*

Duodart *blocks testosterone conversion*

Dental history

Several fillings and prosthetic works, made years ago, patient did not recall details.

Clinical findings

Soft tissues

Healthy gingival tissues, some dental plaque.

Dental

Missing mandibular first molars. Anterior attrition. Composite restorations. Posterior single crowns, bridge 33³⁴35.

Clinical tests

Tooth	47	45	44
Palpation	no	no	no
Percussion	no	no	no
Mobility	wnl	wnl	wnl
PPD	wnl	wnl	wnl

Radiographic findings



Fig. 18.2 - Periapical radiograph 19.04.2012

Tooth 47

- normal periapical conditions (not shown)

Tooth 45

- root filled
- crown and casted post
- apical radiolucency 4 mm

Tooth 44

- root filled
- prefabricated post and coronal filling
- slightly widened apical PDL space, within normal limits

Attachment apparatus

- moderate periodontal bone loss. Over 2/3 bone attachment remained.

Diagnosis

Pulpal K04.19 Root filled tooth

Periapical K04.5 Chronic apical periodontitis

Periodontal K05.3 Chronic marginal periodontitis

PAI 4

Treatment plan

- Surgical endodontic treatment; Apicectomy and retrograde filling.

Alternatives

- Conservative retreatment
- Implant replacement of 45 (and 46).
- Concurrent apicectomy of 44.

The patient wished obtain healthy periapical conditions, and keep 45 if possible. Conservative retreatment was ruled out considering the coronal restoration and the relatively voluminous casted post. A possible further weakening of the cervical section because of post removal could be an untoward result. He did not feel a need for replacement of 46. Implant replacement could be an alternative if 45 was lost. The periapical condition of 44 was deemed healthy, and surgical intervention was decided against.

Problem list

- The mandibular nerve and foramen mentale could interfere with the osteotomy and surgical field

The patient was referred for an orthopantomogram to determine the location of the mandibular nerve and foramen mentale.

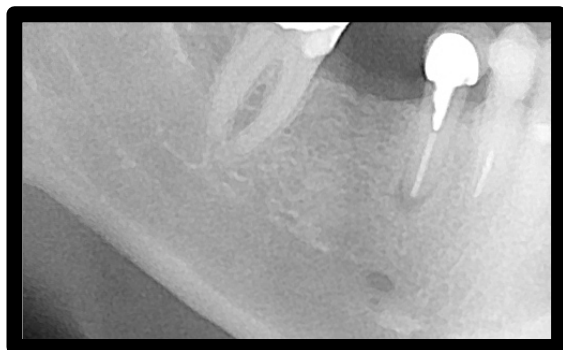


Fig. 18.3 - Orthopantomogram 22.08.2012

Foramen mentale was found 10 mm inferior to the apical lesion, this was regarded a safe distance.

Treatment progress

19.04.2012

- Examination and treatment planning.
- Preoperative information.

19.09.2012

- Apicectomy and retrograde filling 45.

26.09.2012

- Postoperative control.

22.05.2013

- Postoperative control.

03.02.2014

- Follow-up

Surgical treatment

Preoperative information. Chlorhexidine mouth rinse

Anaesthesia

Inferior alveolar nerve block and infiltration: Xylocain 1,8 ml x3.

Incision

A sulcular incision was made, from the mesial margin of 47 to 44, with a 10 mm vertical releasing incision at the mesial margin of 44. The triangular mucosal-periosteal flap was elevated. Care was taken to avoid interference with the mental nerve and foramen during incision, flap elevation, placement of the surgical retractor, and osteotomy.

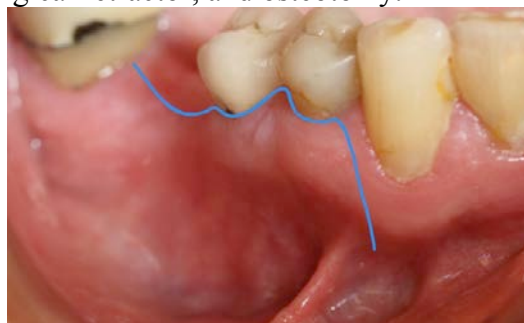


Fig. 18.4 - Incision 19.09.2012

Removal of lesion/osseous access

The apical lesion was exposed with a round steel bur in a low-speed handpiece with external saline irrigation. Lesion removed in toto with a surgical curette, and prepared for histological examination. The root end was exposed with a cylindrical bur.

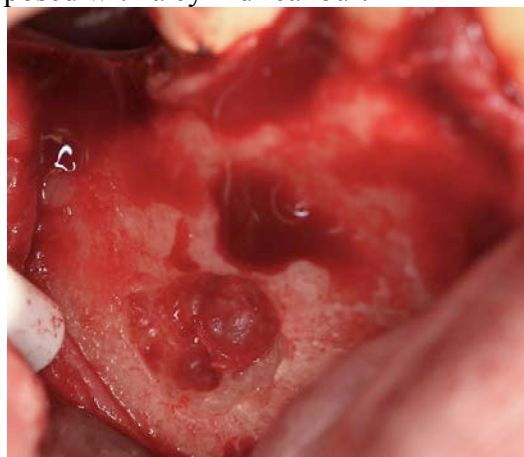


Fig. 18.5 - Lesion exposed 19.09.2012

Apicectomy

Horizontal resection of the apical 3 mm of the root-end.

Retrograde preparation and filling

Root end and canal visualized by methylene blue staining. No fracture lines were noted.

A retrograde canal preparation was made with a 3 mm ultrasonic retrograde tip. Controlled by radiograph. Haemostasis by packing gauze

with ferric sulphate in the crypt. The retrograde preparation was filled with ProRoot MTA. A control radiograph was exposed before removal of excess MTA and wound toilette.

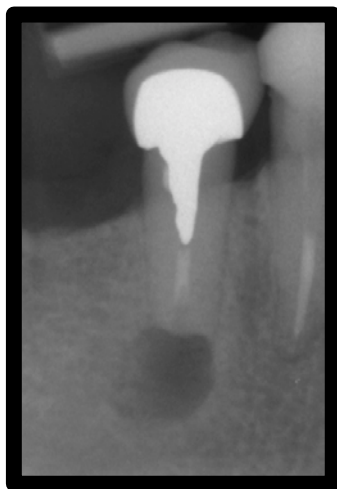


Fig. 18.6 - Control radiograph 19.09.2012

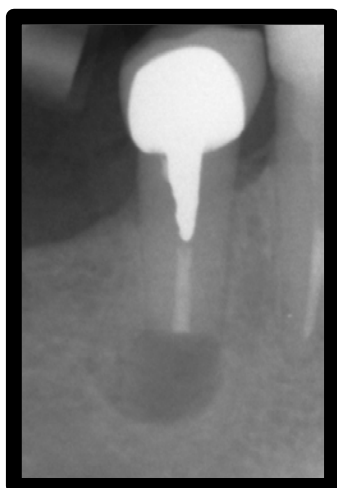


Fig. 18.7 - After placing of MTA 19.09.2012

Sutures

The flap was repositioned, compressed with moist gauze and left for a few minutes before suturing. 6 simple interrupted sutures and 2 interdental sutures (Supramid 4-0, 3/8c, 19 mm) were used.

Postoperative care

The haemostasis was satisfactory. Ice-pack for 15 minutes. Standard postoperative information, emphasis on hygiene, pain control and suspected postoperative discomfort.

Histological findings

Granulation tissue with moderate to intense infiltration of lymphocytes and plasma cells, partly lined by fibrous capsule, with mild lymphocyte infiltration. Findings consistent with apical periodontitis.

(Histological diagnosis by Hanna Strømme Koppang)

Postoperative control, day 7

Patient reported some post-operative discomfort and swelling. Initial numbness in the right lower lip, sensation was regained. Normal healing. Suture removal.

Post operative control, 8 months

The patient could not meet for the 3 month control because of recurrence of lymphatic cancer, and had undergone surgical, cytostatic and radiation therapy (thorax). No symptoms from 45. Normal healing, normal sensation in right lower lip. Radiographical signs of apical healing and establishing of a periodontal ligament space.

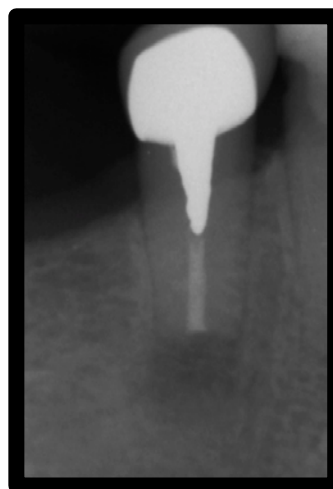


Fig. 18.8 - Control 22.05.2013

Prognosis

Endodontic - favourable
Tooth - favourable
Restoration - favourable

Follow-up

Radiographic evidence of continued healing. 45 bears signs of falling into Rud's complete healing category; with signs of bone fill of the cavity; and a minute apical PDL space.

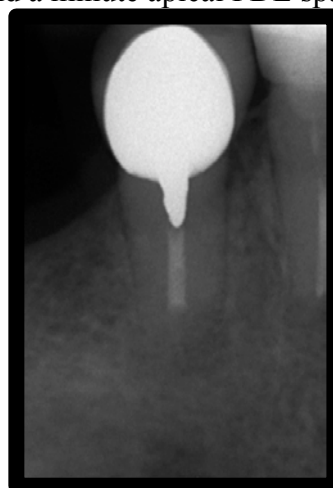


Fig. 18.9 - Control radiograph (over-angulated) 03.02.2014

Evaluation

The history of lymphatic cancer was not mentioned in the anamnesis. To the patient's knowledge, cancer was in remission.

The numbness experienced immediately postoperatively is a sequela that may occur after surgery in this area. When the nerve is not irreversibly damaged, full sensation should be regained, as it was in this case.

The treatment result appeared satisfactory.

Discussion

Dental implants provide an opportunity for fixed replacement of missing teeth. As success rates of implants are considered high (over 90%), some view them a valid or even preferable alternative to endodontic procedures[1]. Comparison of outcome of implants and endodontic treatment is challenging, as the criteria for evaluation differs.

The goal of endodontic treatment is to prevent or cure apical periodontitis[2]. Thus, endodontic success is usually defined as sound periapical conditions, recognized by absence of symptoms and no radiographic signs of periapical disease[3]. Radiographic findings are usually evaluated by Strindberg's criteria and PAI. After surgery, Rud and Molven's classifications are applied[4-8].

For implants, success implies indications of osseointegration[9]. However, a majority of implant studies assess outcome by survival rather than success criteria. Stability, function, and aesthetics are defined aims of implant treatment. This may allow cases with persistent pain, neuropathy, increased probing depths, peri-implantitis, prosthesis instability or peri-implant radiolucencies to be categorized as positive outcomes. Factors like aesthetics, soft tissue aspects, or patient satisfaction are disregarded in some studies. In addition, inclusion criteria are often strict, excluding patient factors like diabetes, poor oral hygiene, para-functions or smoking[4]. Results may thus not apply to the general population. According to the much used criteria of Smith and Zarb[10], implant success should only be evaluated after osseointegration, and on functionally loaded implants. This means that early failures are not included in success/survival rates, which is remarkable, considering that most failures occur before placement of the supra-construction[4]. Iatrogenic complications like

impingement of neighbouring roots or the mandibular canal, also disqualify for evaluation. These situations are regarded not related to implant material or design, and thus ignored. Implants placed in the floor of the nasal cavity or the maxillary sinus, constitute 'a separate entity', and are not included[10].

Survival rates in implant studies may range from 91 to 100%, while estimated success in the same materials are 71%-84%[4].

Some studies have described outcome of endodontic therapy in terms of survival. Ng et al. found 95% 4-year survival for both primary treatment and retreatment[11]. Doyle et al. did a match pair study of implants and endodontically treated teeth, finding the exact same failure rate (6,1%) for both after 7-9 years[12]. However, postoperative complications requiring intervention occurred on 18% of implants, and only 4% of teeth. In a systematic review, Iqbal and Kim compared survival rates of single-tooth implants and endodontic treatment[13]. Survival rates in implant studies were ranging from 89,5 to 100%, for endodontic treatment from 81,2 to 100%. Included studies were pooled to depict survival at different follow-up lengths. Eight pools were made, from 6 months to 72-300 months follow-up. For every follow-up stage, confidence intervals of implant and endodontic treatment survival overlapped, indicating there was no evidence of a difference. The decision to treat a compromised tooth endodontically or replace it with an implant should be based on factors other than treatment outcome. When subjected to therapy, teeth with even advanced periodontal disease have a favourable outcome, comparable to implants[4, 14]. In terms of survival, a need for conservative or surgical endodontic retreatment is not a necessarily a failure, but a potential 'survival with intervention' case. It may be likened with treatment of peri-implantitis, abutment failures or other unfortunate, but often manageable events occurring with dental implants.

Regarding prognosis for apical surgery, both retrospective study design (Tsesis 2006) and a systematic review/meta-analysis (Setzer 2010) have found significantly improved outcomes by modern surgical techniques (magnification/illumination, horizontal root-end resection, ultrasonic preparation and

Super-EBA/IRM/MTA retrograde fillings)[15-17]. Tthesis found 91% complete healing after 6-48 months follow-up of cases subjected to 'modern' surgical treatment, in contrast to 44% by 'old' techniques[15]. The review/meta-analysis has been criticised for a methodology enhancing the difference between modern techniques and old, or even inflating the outcome of modern surgical treatment due to short follow-up (see case 17)[18]. However, current knowledge still support that the low success rates stated by those who endorse implants over surgical retreatment, deny advances of modern endodontic surgery[1, 18-20].

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Case 19

Surgical endodontic treatment of a maxillary left central incisor.

A 18-year-old female of Middle-Eastern origin was referred to the Section of Endodontics from the public dental health service in Oslo, for endodontic treatment of the maxillary left central incisor, 21. Orthograde endodontic treatment was recently conducted by another postgraduate candidate.



Fig. 19.1 - Frontal view 13.03.2013

Chief complaint

Patient had suffered prolonged pain in the maxillary front.

Medical history

Patient was in car accident at the age of 6-7 years. She was thrown from the vehicle, and stayed in hospital for several days. Chronic headache since the accident.

Dental history

Enamel-dentin fracture of mesial corner of 21 at the accident. According to the referral, pain evolved and endodontic treatment was initiated in 2012. The symptoms from 21 persisted, and an intracanal calcium hydroxide dressing was changed several times.

At initial treatment planning, the patient had complaints of pain originating in 22. 21 responded negatively to cold and electric sensibility test. 11 and 22 tested positively. On radiographs, evidence of a *dens invaginatus* in 22 was seen, and a referral for CBCT-examination was made to the Section of Radiology. A large radiolucent lesion in was found in relation to 21.

Conservative endodontic treatment of 21 on diagnosis symptomatic apical periodontitis was completed two weeks earlier at the Department of Endodontics. Intense pain persisted, and it had been decided to proceed to surgical treatment for removal of the periapical lesion.

Clinical findings

Soft tissues

Normal findings. Adequate oral hygiene, healthy gingival tissue with some pigmentation.

Dental

A few posterior composite restorations. Enamel-dentine fracture of mesial corner of 21. Yellow discoloration. A temporary IRM filling sealed the palatal access cavity.

Foramen coecum seen on 22

Intense continuous and provoked pain. 21 was pointed out as the origin of symptoms. This and neighbouring teeth were tender to gentle percussion, as was the apical area to palpation.

Radiographic findings

Radiographic examinations before conservative treatment:



Fig. 19.2 - CBCT - coronal view 19.03.2013

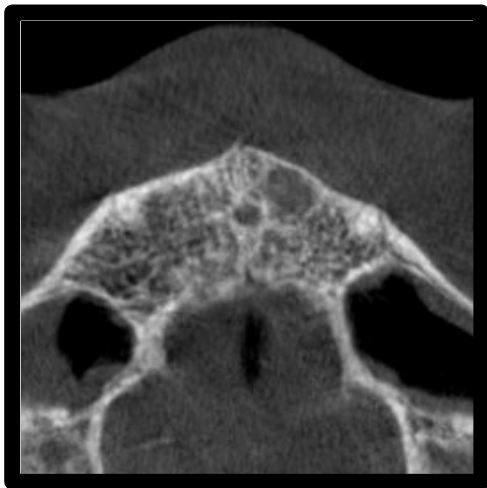


Fig. 19.3 CBCT - axial view 19.03.2013

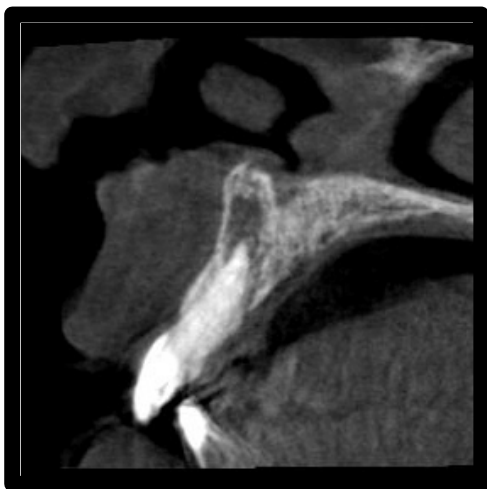


Fig. 19.4 CBCT - sagittal view 19.03.2013

CBCT revealed a radiolucency continuous with the PDL of 21, sized $\approx 8 \times 12$ mm. Normal findings in the apical area of 22. Communication between the invagination and the pulp was not confirmed. The large periapical lesion was not distinguishable on preoperative intraoral exposures.

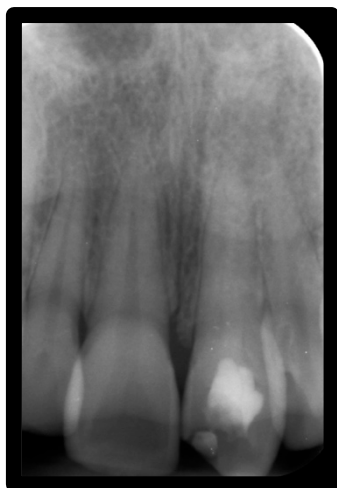


Fig. 19.5 - Preoperative periapical radiograph 02.04.2013

Radiographic findings after orthograde treatment:



Fig. 19.6 - Periapical radiograph 30.04.2013

Tooth 11

- normal

Tooth 21

- root filled

- apical radiolucency $\approx 8 \times 10$ mm

Tooth 22

- *dens invaginatus*, type 2 according to Oehlers classification

Attachment apparatus

- normal

Diagnosis

Pulpal K04.19 Root filled tooth

Periapical K04.5 Chronic apical periodontitis

Periodontal Normal

PAI 5

Treatment plan

- Surgical endodontic treatment.

It was decided to surgically remove the lesion, with concurrent apicectomy. Evidence of overinstrumentation was seen during the orthograde treatment. A #040 file could penetrate the apical foramen. MTA had been placed in the apical 8 mm of the canal.

Alternatives

- defer surgical treatment, observe if symptoms subsided

The symptoms was unaffected by the treatment completion. Considering this, and the large lucency evident on CBCT, it was decided to intervene surgically.

Problem list

- Surgery in the aesthetic zone
- The patient was somewhat attentive to her symptoms, it was feared that she

would suffer experiencing postsurgical discomfort and pain.

A submarginal incision was considered. The gingival contours of the incisors were intact, hence there was no risk of exposing restoration margins by postoperative soft tissue retraction. Because of the risk of gingival scarring, an intrasulcular incision was chosen. This was believed to likely yield the most satisfactory aesthetic result.

The patient was informed of postoperative discomfort, the normality of experiencing some pain, and possibilities for analgetic medication.

Treatment progress

16.05.2013

- Clinical examination and radiographic evaluation.
- Preoperative information.
- Surgical treatment of 21, removal of apical lesion and apicectomy.
- Postoperative information

22.05.2013

- Scheduled postoperative control.

26.09.2013

- Scheduled postoperative control.

In addition, the patient on own initiative made several post-operative appointments.

Surgical treatment

Preoperative information. Chlorhexidine mouth rinse

Anaesthesia

Infiltration: Xylocain 1,8 ml x2. Additional 4 carpules were administered during the procedure.

Incision



Fig. 19.7 - Incision, illustration 16.05.2013

Sulcular incision from the buccal margin of 13 including 23, 15 mm vertical release at distal margin of 23. Elevation of flap. Several small perforations of the corticalis by the apical lesion was seen:

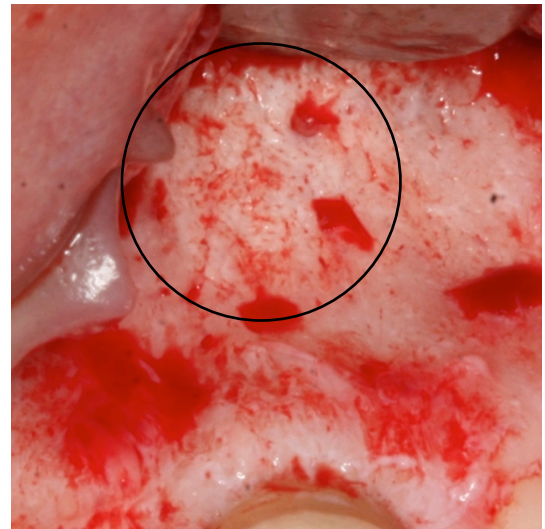


Fig. 19.8 - Corticalis regio 21 was perforated 16.05.2013

Osseous access

The apical lesion was exposed with round burs. External saline irrigation. The lesion was removed *in toto* and prepared for histological examination.

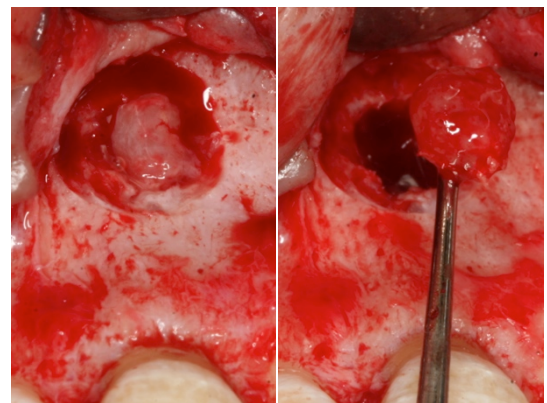


Fig. 19.9 - Osteotomy and removal of lesion 16.05.2013

Apicectomy

Apical 3 mm was removed with a cylindrical steel bur. Refined the resection plane to a horizontal surface with smooth MTA in the canal. Tissue debris removed by apical curettage and saline irrigation.

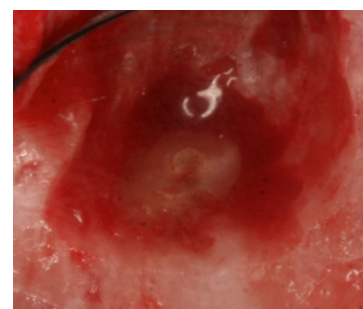


Fig. 19.10 - Apicectomy 16.05.2013

Sutures

Repositioning of flap, compressed with moist gauze. 3 simple interrupted sutures on the releasing incision (Supramid 5-0, 3/8c, 12 mm), and 5 interdental sutures (Supramid 5-0, 3/8c, 19 mm).

Postoperative care

Haemostasis was satisfactory. Ice pack for 10-15 minutes. 600 mg ibuprofen immediately postoperatively. Standard postoperative information, emphasis on the normality of some postoperative pain and discomfort.



Fig. 19.11 - Sutures 16.05.2013

Histological findings

Fibrous connective tissue. Diffuse moderate infiltration of chronic inflammatory cells. No granulation tissue or epithelial cells. There was not confirmed any foreign substance, but voids of variable sizes surrounded by giant cells may have contained such.

Diagnosis: Fibrous connective tissue with moderate chronic inflammation and possible foreign bodies.

(Histological diagnosis by Tore Solheim)

Postoperative control, day 6

Patient reported normal postoperative discomfort and pain. Satisfactory soft tissue healing. Removal of sutures.



Fig. 19.12 - Control - 22.05.2013

Patient was to be recalled after 3 months. During this period, she made several appointments at the Institute of Clinical Dentistry. For practical reasons, she met with other dentists, at different departments. Records described variations in pain characteristics and findings; pain on percussion of all maxillary incisors/ yet responding positive to sensibility tests, pain at night/ disrupting sleep, pain from 21/ tenderness on palpation. Positive sensibility on 11/22/23/24.

On different occasions, she was prescribed ibuprofen, paracetamol/codeine phosphate (Paralgin Forte) and penicillin. Evidence of healing was seen on radiographs.



Fig. 19.13 - Control 18.09.2013

Postoperative control, 4 months



Fig. 19.14 - Control 26.09.2013

Scheduled control. Minor gingival recession. Pain persisted; spontaneous or provoked by chewing or touch. Only 21 was tender on percussion, no tenderness on palpation. 13/12/11/22/23 was sensitive to a cold stimulus, 21 was not. Normal mobility and healthy periodontal conditions (probing).

Radiographical signs of apical healing on 21. The vertical line in the mid-root root filling was interpreted as level of MTA in the canal. Possible widening of apical and lateral periodontal space of 22. The feature was seen on radiographs dating back to April (Fig. 19.6). As 22 responded normally to sensibility tests, endodontic treatment was deferred.

The patient was quite focused on the condition of 21. She was encouraged ignore symptoms and settle with that the infection was successfully treated. Information of clinical and radiographic findings.

A new control was scheduled 3 months later, but before that, she again contacted the clinic. According to records, endodontic treatment of 22 was instigated, but symptoms not relieved. She was discharged, and advised to contact her physician for medical examination and follow-up of chronic pain.



Fig. 19.15 - Signs of healing of periapical lesion
22.01.2014

Prognosis

Endodontic - favourable

Tooth - favourable

Restoration - favourable

Prognosis for endodontic treatment 21 was deemed favourable. The expectations for further development of symptoms was regarded uncertain, or even unfavourable.

Evaluation

The technical and biological treatment result seemed satisfactory.

The periapical lesion of 21 was not seen on the preoperative intraoral radiographs. Thus, the CBCT was gave valuable information.

It is not clear if the symptoms initially originated in a pathological pulpal condition of 21. The histological findings did not reflect an acute infectious state, and consequently the periapical lesion was unlikely the source of pain at the time of surgery. Voids reported in the histological examination may be explained by extrusion of calcium hydroxide dressing in the initial treatment phase. The records describe communications in the apical floor of the invagination. It cannot be excluded that 22 was periodically symptomatic, although the early examinations gave no indication of this. The apparent chronic pain condition may have been related to the accident the patient suffered as a child (see discussion).

Discussion

CBCT has the potential to show periapical bone loss not readily visualized by periapical radiographs[1-3]. In endodontics, CBCT may yield information in many respects, like identification of accessory canals, diagnosis of root canal anomalies, diagnosis and management of trauma and root resorptions, and diagnosis of nonspecific periapical pathosis[4].

Periapical radiolucencies mainly consist of inflammatory lesions. Most frequently found are periapical granulomas (43.4%) and radicular cysts (21,2%), whereas non-specific inflammation constitute 0,9%[5]. For intraoral radiographs, histological findings have been correlated with radiographic signs[6-8]. The diagnostic accuracy for CBCT is not known. CBCT is a valuable diagnostic tool, and indications exist on higher sensitivity, and equal or higher specificity than intraoral periapical radiography[1, 2]. Observer

variation has been found to be lower than for conventional radiographs[2, 9]. However, CBCT has not yet been measured against a true reference method[10]. A study on assessment of healing after periapical surgery, revealed that remaining defects were detected by CBCT after one year, that were not seen on periapical radiographs[9]. This is interesting, because radiographic evaluation of healing after periapical surgery have been demonstrated to correlate with histology. Moreover, the radiographic classification of healing one year after surgery have a high predictive validity for long-term outcome[8, 11]. Periapical radiographs have limitations, as demonstrated in this case, where a periapical lesion was clearly present. However, *post-mortem* studies or examination of surgical biopsies is needed to document CBCT accuracy[10].

Sensory neurons have a potential for plasticity in response to inflammation or tissue injury. In animal studies, long-lasting changes in expression of neuropeptides and their receptors have been observed after injury. Changes may represent adaptive responses to limit consequences of damage to the organism as a whole[12]. Redistribution of sodium channels within the trigeminal neurons may be important in establishing peripheral nerve hyperexcitability and resultant neuropathic pain[13]. Irreversible pulpitis is associated with significant changes in expression of ion channels, receptors, and neuropeptides. Inflammation of a single tooth may be sufficient to trigger central sensitization[14]. Orofacial pain and headache may have a comorbid nature. Research on migraine usually focus on the ophthalmic branch of the trigeminal nerve[15]. Excitation of one of the trigeminal branches may cause excitation of the other two[16]. In the trigeminal nucleus caudalis intracranial and extracranial nociceptive input is integrated, and activation of second order neurons may lead to central sensitization[15].

In some patients with chronic pain disorders, autonomic symptoms like lachrymation, nasal congestion, rhinorrhoea, or oedema is observed. Autonomic postganglionic sympathetic and parasympathetic neurones do not communicate with afferent neurones to generate sensations under normal conditions.

In chronic pain conditions, nociceptive activity may be maintained by sympathetic neurones, generating a state of central sensitization and hyperexcitability, leading to spontaneous pain and secondary evoked pain[17].

Non-physical factors such as history of pain or trauma, on-going emotional states and coping skills may interact with actual physiological factors, and determine the individual pain experience[18]. A multidimensional approach is often necessary because of these factors, and the complexity of dysfunctions within the trigeminal system. Pain related to extrusion of calcium hydroxide are usually of an immediate character, this was not likely causing the pain condition in this patient[19]. However, any insult that may elicit pain is regrettable in a patient with chronic pain. It was not clear what the source of pain was in this case, but the trauma in her childhood, her chronic headache and a long-standing pulpal inflammation may have affected the condition.

The aetiology of *dens invaginatus* is not clear, but most consider it a result of deep folding of the foramen coecum during tooth development. Genetic factors cannot be excluded. It is most common in the maxillary lateral incisor, where occurrence is bilateral in 43% of cases[20].

Oehlers characterized and classified *dens invaginatus* in 1957[21]:

Type 1

Enamel-lined and confined within the crown, not extending beyond the level of the ECJ.

Type 2

Enamel-lined and invading the root, confined within it as a blind sac, which may communicate with the pulp.

Type 3

Extending throughout the root, penetrating it, and forming an apical or lateral 'second foramen'. Lined by enamel, but a portion is often lined by cementum. Usually without pulpal communication

Crowns of involved teeth may range from normal with a deep lingual or palatal pit; conical, barrel-shaped or peg-shaped with an incisal pit. Invagination and crown/root may be dilated.

If no entrance to the invagination is detected and there are no signs of pathosis, observation

is sufficient. Deep palatal or incisal invaginations/foramen coeca should be sealed by composite, and reviewed periodically[22]. If pulpal or periapical pathosis evolve, the mode of treatment depend on extent of the invagination and wheter pulpal tissue is affected. Root canal treatment may be performed on the invagination only in type 3 cases. In teeth with pulpal pathosis, endodontic treatment involving both pulp and invagination is necessary. Surgical intervention is indicated if root canal configurations are complex, or conservative treatment unsuccessful. If pathosis occur before completion of root formation, apexification procedures are called for[20, 22, 23].

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Case 20

Surgical treatment of a maxillary left second premolar, 25, with persistent apical periodontitis. Conservative endodontic treatment of maxillary left first and second molars, 26 and 27.

A 51-year-old female was referred to The Department of Endodontics from an endodontist in private practice, for endodontic treatment of root filled maxillary left second premolar, 25, and first molar, 26.



Fig. 20.1 - Frontal view 29.08.2012

Chief complaint

Patient reported tenderness in left maxilla on palpation or chewing.

Medical history

Elevated blood pressure (controlled)

Medicines

Selo-Zok *beta-blocker*

Dental history

Patient at the undergraduate clinic 2005-2008. Endodontic treatment and subsequent post retained crown restoration of 25 in 2008. Later, three and four unit bridges have been made in posterior segments.

Four episodes of swelling and considerable pain in left posterior maxilla in the last year. Symptoms resolved after antibiotic treatment.

Clinical findings

Soft tissues

A 10 mm diameter wide, fluctuant swelling with rubor in buccal mucosa, 5 mm superior to the gingival margin of 25.



Fig. 20.2 - Buccal swelling 29.08.2012

Dental

Upper four unit and lower three unit posterior bridges were not well adapted to abutments or gingival tissues. Cervical discolorations on frontal mandibular teeth.

Clinical tests

Tooth	23	24	25	26	27
Cold	yes	no	no	yes	no
Palpation	no	no	yes	no	no
Percussion	no	yes	no	yes	yes
Mobility	-	-	-	-	-
PPD	wnl	wnl	4	4	wnl

Radiographic findings



Fig. 20.3 - Periapical radiograph 29.08.2012

Tooth 24

- normal periapical findings

Tooth 25

- root filled – casted post
- widened periodontal ligament space
- apical radiolucency 7x11 mm

- lateral radiopacity \approx 1 mm

Tooth 26 and 27

- radiopaque material mesiobuccal root
- apical canals not distinguishable
- widened PDL/no lamina dura 26, PDL space not distinguishable on 27

Attachment apparatus

- moderate periodontal bone loss. Over 2/3 of bone attachment remained.

Bridge

- over and under-extensions of restoration are seen on 24 and molars respectively.

Diagnosis

25

Pulpal K04.19 Root filled tooth

Periapical K04.5 Chronic apical periodontitis

Periodontal Normal

PAI 5

26 and 27

Pulpal K04.1 - Pulp necrosis

Periapical K04.5 Chronic apical periodontitis

Periodontal Normal

PAI 4 for 26, 3 for 27

The buccal swelling near 25 and former pain were suspected to originate in the premolar, but it could not be excluded that 26 and 27 were symptomatic.

Treatment plan

25

- Surgical endodontic treatment; Apicectomy and retrograde filling.

26

- Conservative endodontic retreatment

27

- Conservative endodontic treatment

Treatment alternatives

- Resection of 25

Tooth 25 was not essential for retention of the bridge, and full resection of the root was an alternative. As the patient was motivated for conserving 25, an attempt on a surgical approach was chosen.

It was decided to initiate treatment of 26 before surgery of 25, as concomitant apicectomy of the molar was possible if apical canals were not negotiable.

Problem list

25

- Access/overview palatal root

26 and 27

- Access through the bridge
- Locate and negotiate canals
 - Canals not visible on radiograph
 - Step in mesiobuccal root 26?

Treatment progress

29.08.2012

- Examination and treatment planning.
- Preoperative information on apicectomy.
- Access/working length determination 26.

20.09.2012

- Instrumentation 26.

26.09.2012

- Apicectomy and retrograde filling 25.

03.10.2012

- Postoperative control.

04.10.2012

- Obturation 26.
- Instrumentation 27.

17.10.2012

- Obturation 27.

05.12.2012

- Postoperative control

Treatment of 26 was initiated before surgery of 25.

(Summary of treatment of 26 and 27 is described after the surgical procedure)

Surgical treatment

Preoperative information. Chlorhexidine mouth rinse

Anaesthesia

Infiltration: Xylocain 1,8 ml x2.

Incision

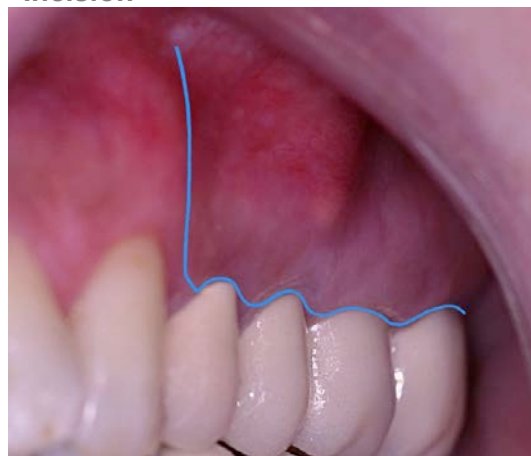


Fig. 20.4 - Incision 26.09.2012

A sulcular incision was made from the distobuccal margin of 27 to 24, with a 15 mm

vertical releasing incision at the mesial margin of 24. Elevation of flap.

A pathological process, approximately 8 mm in diameter, penetrated the cortical bone.



Fig. 20.5 - Penetrating pathological process
26.09.2012

Removal of lesion/osseous access

The apical lesion was removed *in toto* with a surgical curette, and prepared for histological examination in 4% formaldehyde.



Fig. 20.6 - Removal of lesion exposes buccal root end
26.09.2012

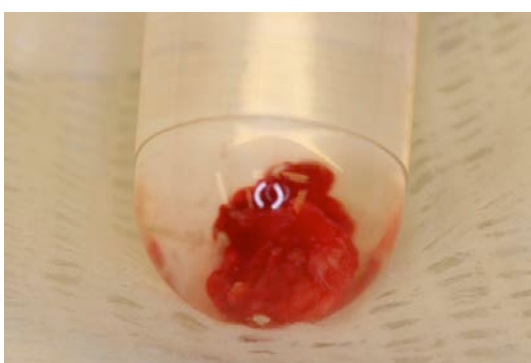


Fig. 20.7 - Prepared tissue sample 26.09.2012

Apicectomy

The apical 3 mm of the buccal root was resected horizontally with a cylindrical steel bur. The palatal root-end was exposed with the same bur, and the apical 3 mm resected.

Retrograde preparation and filling

Root-ends and canals visualized by methylene blue staining, no fracture lines noted.

Retrograde canal preparation with a 3 mm ultrasonic retrograde tip. Controlled by radiograph. Haemostasis by packing cotton pellets with ferric sulphate in the crypt. The retrograde preparations were filled with ProRoot MTA. A control radiograph before removal of excess MTA and wound toilette.

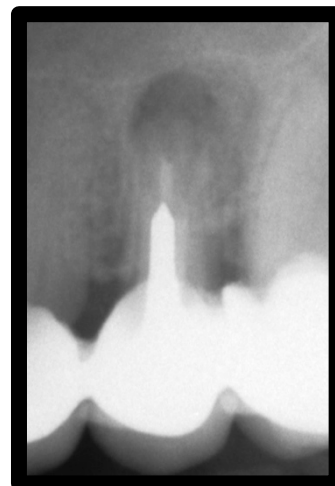


Fig. 20.8 - Control radiograph 26.09.2012

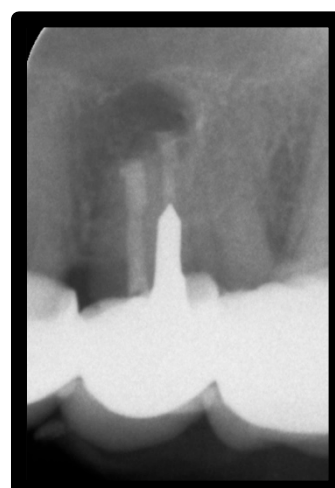


Fig. 20.9 - After placing of MTA 26.09.2012

Sutures

Flap repositioned and compressed with moist gauze for a few minutes before suturing. 4 simple interrupted sutures (Supramid 4-0, 3/8c, 12 mm) and 3 interdental sutures (Supramid 4-0, 3/8c, 19 mm) were used.

Total time of the surgical procedure: 2 hours.

Postoperative care

Satisfactory haemostasis. Ice-pack for 10-15 minutes. 600 mg ibuprofen immediately postoperatively. Standard postoperative information, emphasis on hygiene measures, pain control and normal postoperative discomfort.

Histological findings

In the specimen, a central lumen partly lined with stratified squamous epithelium was found. Subepithelial granulation tissue

intensely infiltrated with inflammatory cells and surrounded by an outer fibrous connective tissue capsule. Fine granules and globular bodies of foreign substance, consistent with an endodontic filling material, in lumen and in tissue infiltrates.

Findings consistent with a radicular cyst.

(Histological diagnosis by Hanna Strømme Koppang)

Postoperative control, day 7

Patient reported little post-operative discomfort and swelling. Normal healing. Suture removal.



Fig. 20.10 - Control 03.10.2012

Post operative control, 2,5 months

No symptoms, normal healing. Gingival retraction exposed restoration margins. Radiographic signs of healing and establishing of a PDL space.



Fig. 20.11 - Post operative control 05.12.2012

Treatment 26 and 27

Instrumentation of 26 was performed one week before surgery, the rest of the conservative endodontic treatment after. Procedures according to the standard regime. BioRaCe rotary instrumentation of canals.

Technical details 26 and 27

Rubber dam/disinfection. Diamond burs to cut thorough ceram. Working lengths determined, (corrected in MB canals of 26 after EAL control during instrumentation). Instrumentation. PUI.

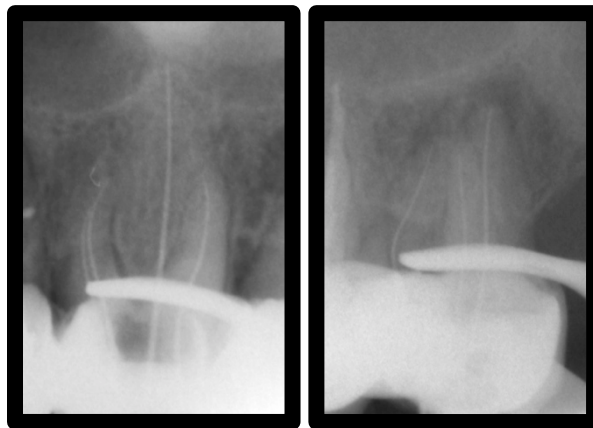


Fig. 20.12 - Working length radiographs 26 left 20.09.2012 and 27 right 04.10.2012

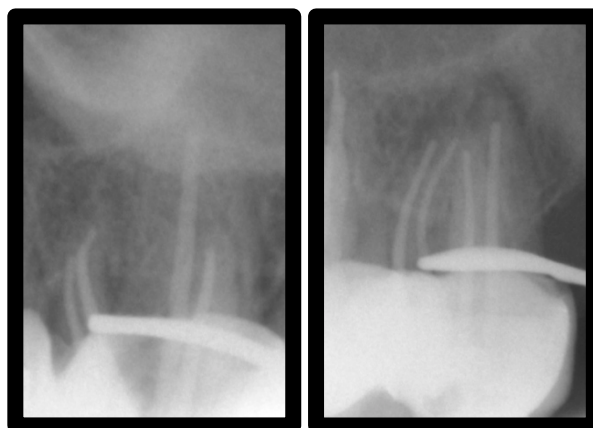


Fig. 20.13 - Masterpoint radiographs 26 left 04.10.2012 and 27 right 17.10.2012

Mechanical

- Burs, ultrasonic tips, K-files, BioRaCe

Chemical

- 10 ml 1% NaOCl / 5 ml 17% EDTA, in each tooth on each visit
- PUI; NaOCl, 20s/canal after instrumentation.

Intracanal medicament (2 weeks)

- Ca(OH)₂

Temporary filling

- Cavit G, IRM.

Obturation

- Gutta-percha and AH Plus. IRM

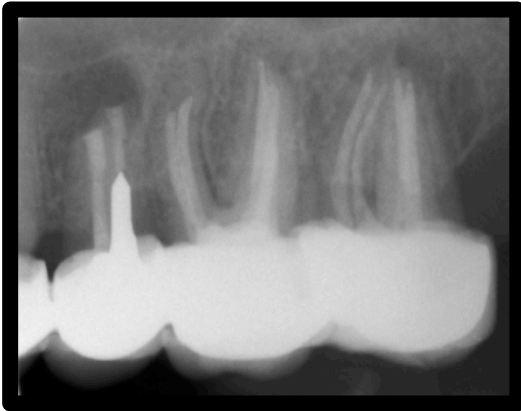


Fig. 20.14 - Final radiograph 17.10.2012

Prognosis 25 - 26 - 27

Endodontic - favourable
Tooth - favourable
Restoration - favourable

Follow-up

The patient did not meet for scheduled follow-up, but continued treatment at the undergraduate clinic. Radiographic evidence of healing of all apical lesions was seen.



Fig. 20.15 - 19.09.2013

After one year, the bridge was replaced, and 25 was removed. The patient stated she again had experienced pain in the area, and received antibiotics after an emergency visit to a dentist outside of the university clinic.



Fig. 20.16 - 26.11.2013

Evaluation

The overall technical treatment result appears adequate, and lesions were healing. The marginal incision was imprecise, this may be attributed to lack of experience with the microsurgical blade.

Root filling of mesiobuccal canals of 26 is seen 2-3 mm short of radiographic apex on postoperative radiographs. MB lengths were corrected with apex locator during instrumentation, the readings may have been influenced by the metal restoration.

Discussion

Although introduced as a root-end filling material, MTA today has a wider range of use, including perforation repair, vital pulp therapy, and apical barrier formation[1, 2]. An ideal endodontic repair material should provide a hermetic seal of the root canal, be nontoxic, noncarcinogenic, nongenotoxic, biocompatible, insoluble in tissue fluids and dimensionally stable[2]. Within a few years it was established that MTA had superior properties compared to contemporary root-end filling materials like amalgam, composite resins and zinkoxide eugenol-based alternatives.

Numerous leakage studies have been performed on MTA, methodologies ranging from dye leakage, fluid filtration to protein and bacterial leakage. The vast majority of these studies demonstrate superior sealing effect of MTA compared to materials like Super EBA, IRM, amalgam, glass ionomer cement, and Geristore[3]. Biocompatibility in regard of lack of mutagenic, neurotoxic or cytotoxic properties is also demonstrated. Inertness is more pronounced after initial setting[3]. The setting time of MTA is by manufacturers generally claimed to range from 2-4 hours. In some compositions, like MTA Angelus, the setting time allegedly is as short as 10 minutes[4]. However, studies have found that complete setting as demonstrated by indentation, or optimal resistance in push-out testing, may in fact take longer time; 36-72 hours [5, 6].

EndoSequence (Brasseler, Savannah, GA, USA) is a new, bioceramic material, introduced in different formulas for usage either as a sealer or for root repair, including root-end fillings. Containing calcium silicates, the composition of Endosequence is similar to MTA. It differs in the lack of aluminium

(tricalcium aluminate and tetracalcium aluminoferrite), and addition of calcium phosphate[7, 8].

There is currently a limited amount of studies on these materials. So far, results are promising but inconclusive in respect of comparability to MTA. A PubMed search on 'endosequence root repair' in March 2014, yielded a handful studies comparing properties of MTA and Endosequence Root Repair Material (ERRM). In a bacterial leakage study, Nair found no significant differences between ProRoot MTA and ERRM[9]. However, Hirschberg demonstrated significant more bacterial leakage though simulated retrograde fillings obturated by ERRM (93%), compared to ProRoot MTA (20%)[10]. Regarding cell viability or cytotoxicity, similar results are found in most studies[8, 11-14]. One study yielded more promising results for ERRM compared to MTA[15], another the opposite[16]. After exposure to PBS, precipitation of apatite crystals were found on surfaces of both MTA and ERRM. This may be viewed as an indication of bioactivity[17]. Surface pH was found to be higher on MTA than ERRM on simulated resorption defects filled with the materials[18]. Concerning antibacterial activity, equal reduction of viable *E. faecalis* counts were obtained after direct contact with both ERRM and MTA[19]. Finally, the setting time of both materials have been investigated. Charland found inferior setting of ERRM in presence of blood or minimal essential media or saline (> 48 hours, whereas MTA set completely within 36 hours)[5].

Thus, it appears that with an exception for setting time (and possibly leakage), comparable *in vitro* properties were found for ERRM and MTA. The bioceram is claimed to have superior handling properties and cost-effectiveness. If future clinical results are convincing, ERRM may challenge the current dominant position of MTA as a root-end filling material.

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