UNIVERSITY OF OSLO

FACULTY OF DENTISTRY

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Postgraduate Program in
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Case Book
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UiO : Det odontologiske fakultet

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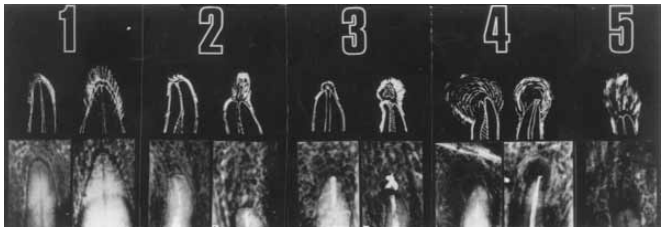
- Sealers: AH Plus, Epiphany/Real Seal Core materials: Gutta-percha, Resilon
- Temporary IRM top filling with a 2 mm IRM plug in the canal orifice.
- Removal of rubber dam
- Final radiograph

Treatment of Tooth with Apical Periodontitis:

- The same treatment as for teeth without apical periodontitis, but two-appointment treatment is the standard procedure: 2-3 weeks between 1st and 2nd appointment is the standard (this is mainly for practical reasons:
- Two-layered temporary top filling: Cavit G and IRM
- *In retreatment cases*: Final irrigation with 17% EDTA and 2% chlorhexidine

- The periapical index (PAI) Ørstavik *et al* 1986, is used for radiographic evaluation.



Reference set of radiographs with corresponding line drawings and their associated PAI scores (Ørstavik et al 1986).

Endodontic Surgery:

- All relevant radiographs mounted on viewer or screen
- Anaesthesia.
- 1 minute mouth rinse with Corsodyl®(Chlorhexidine 2mg/ml)
- full mucoperiosteal flap
- *Incision*: A horizontal incision extending one to several teeth mesial and distal of the involved tooth and one vertical-releasing incision, usually placed at the mesial end of the prospective flap.
- *Retraction:* To hold the flap away from the surgical site, providing maximum access and visibility, without causing harm to the flap or the surrounding tissues.
- *Osteotomy:* Involves removal of cortical and cancellous bone to gain direct access to the apical portion. This is achieved routinely by using rotary instruments.
- *Surgical curettage*: To remove all pathologic tissue, foreign bodies, and root and bone particles from the periradicular area.
- *Biopsy:* any soft tissue lesion removed during the surgical procedure should be submitted for biopsy.

- *Microbiological sample*: with paper point directly in the periapical smple, placed in prereduced anaerobic transportmedium, or periapical tissue placed in 4% formalin for scanning electron microscopy.
- *Root end resection*: by sectioning the apical segment of the root and/or bevelling it to the line of sight. 3 mm.
- *Ultrasonic root end preparation*: To provide a clean, well-shaped class I cavity
- *Haemorrhage control*

Local anaesthetic solutions possessing vasoconstrictor properties - Xylocain-Adrenalin® 10mg/ml + 5µg/ml
Stryphnon gauze (Adrenalonchlorid 0,33 mg/cm2)
Ferric sulfate (Fe2[SO4]3 with 15.5% astringet and 21% stasis)

- *Root-end filling:* using either IRM or MTA. Use of the MAP system (Micro-Apical Placement) or the MTA pellet-forming block will ease the application of MTA.
- *Cleaning of surgical site:* Saline solution to remove debris, and tissue edges are re-approximated in their correct position to promote healing by primary intention. Compression of the repositioned tissue with a saline-moistened piece of gauze will reduce the coagulum to a thin fibrin layer between the repositioned tissue and cortical bone. Tissue margins should rest passively in the desired place before suturing.
- *Wound closure:* using non-absorbable suture material in sizes 4-0 and 6-0. Postoperative radiograph is taken for control of procedures and as reference for follow-up.
- *Postsurgical care*: A disposable ice pack is covered with soft towelling, and the patient instructed on where and how to hold the ice pack firmly in position against the facial tissues approximating the surgical site. Unless contraindicated for some reason, the patient is instructed to take IBUPROFEN 400 mg every 4 to 6 hours for the first 48 hours. The patient is advised to rinse with Corsodyl® twice daily until suture removal.
- *Suture removal*: The epithelial seal at the wound edges is evident within 2 days; suture removal can take place earliest after 48 h but not later than 4-5 days.
- *Prescription* of: IBUPROFEN 400 mg. NO 30. Every 6 hours for 3 days.

Case 1

Treatment of the mandibular left second molar with acute irreversible pulpitis and cracked tooth syndrome

Fig 1. Frontal view

Fig 2. Tooth 37, several craze lines are visible in the distal, lingual and buccal aspect of the tooth

Patient: 65 year old Caucasian male
Chief complaint: He complained about strong pain left side. He felt sudden pain when chewing and biting, and he had problems with sleeping because of pain
Medical record: Non-contributory
Dental history: The patient was referred to the post-graduate endodontic clinic from the graduate clinic for retreatment of tooth 36 because of sudden pain.
Clinical findings 26 th of October 2010:
Soft tissue: normal findings
Dental:
Tooth 35 MOD amalgam filling
Tooth 36 MODL composite filling
Tooth 37 occlusal and buccal amalgam filling. Several craze lines can be seen in the enamel (Fig. 2 , 3).

Fig 3. Occlusal view

Clinical test 26 th of October 2010

	35	36	37
EPT (0-80)	17	-	14
Cold	+	-	+
Percussion Vertical/horizontal	-	v	v and h
Palpation	-	-	+
PPD	3	3	3
Mobility	-	-	-
Biting			+

Table 1

The patient has an intense reaction upon coldtest and EPT on tooth 37. He could tell

about lingering pain that lasted about 30 seconds. Using a FracFinder on the different cusps of tooth 37, the patient felt sharp pain when dl cusp was tested. He felt the pain most when he opened his mouth.

Radiographic findings 26 th of October 2010:
Dental: Tooth 36 has an insufficient root canal treatment and an MOD radiopaque restauration.
Tooth 37 has a curved mesial root and radiopaque restauration
Periodontal: 36 within normal limits,
37 attachement 2/3
Apical: 36 apical radiolcency mesial root, (PAI 3). 37 normal PDL (PAI 2)

Fig. 4 Periapical radiograph 26.10.2010

Diagnosis 26 th of October 2010:
Tooth 37
Pulpal: Acute irreversible pulpitis (K04.01)
Tooth 36
Pulpal: Root filled tooth (K04.19)
Periapical: Chronic apical periodontitis (K04.50)

Treatment plan:
Tooth 37: Treatment of inflammed pulp
Tooth 36: Retreatment of root filled tooth

Problem list:
Anatomical considerations (curved mesial root) 37

Treatment 26 th of October 2010
Cuspal reduction, especially dl cusp. Access opening and location of three canals. Bleeding from all canals. Distal crack is visible in the pulp chamber with extension mesially.
The patient was told that the tooth would have a less favourable prognosis unless a dental crown was made.
Mechanical: Bur,
NiTi hand instrumentation
-MB 17mm/R40
-ML 17mm/R40
-D16mm/R55
Chemical:1% NaOCl, 16% EDTA
Intracanal medicament: $Ca(OH)_2$
Temporary filling: IRM

Fig. 5 Working length radiograph

Treatment 16th of November 2010:
Less symptoms. Not tender to percussion, but he had exsperiensed sporadic pain from the area.
Filled with gutta-percha and AH plus, sealed with IRM. The graduate student was informed about the cracked tooth and the importance of permanent rastauration with cusp coverage.

Fig. 6 Masterpoint radiograph

Fig. 7 Final radiograph 16.11.2009

Prognosis:

Endodontic: the prognosis seemed to be good

Tooth: uncertain (a semipermanent composite restauration was palaced i December 2009, (one month after endodontic therapy)

The patient had started at a private dental practitioner.

Follow up examination 10 th of November 2011 (12 months):

The patient had still symtoms from the area from time to time. Retreatment of 36 was startet by general practitioner some months ago, but the patient had canselled the last appointment. The patient wanted to complete root-canal treatment of tooth 36 at the postgraduate endodontic clinic.

Fig. 8 Follow up radiograph 10.11.2011

Clinical findings 10 th of November 2011:

Soft tissue: normal findings

Dental:

Tooth 35: MOD amalgam filling

Toth 36: composite filling, occlusal IRM

Tooth 37: composite filling with cuspal coverage, but not covering completely at the distal aspect. The previous buccal amalgam filling was placed with an IRM

Fig. 9 Lingual view

Fig. 10 Buccal view

Clinical test 10 th of November 2011

	35	36	37
EPT (0-80)	23	-	-
Cold	+	-	
Percussion Vertical/horizontal	-	v	v
palpation	-	-	+
PPD	3	3	4
Mobility	-	-	-
Biting		-	+

Table 2

Radiographic findings 10th of November 2011:

Dental:

Tooth 36: insufficient root filled, radiopaque filling MOD

Tooth 37: root filled, radiopaque filling MO

Periodontal:

Tooth 37 attachment =2/3, bifurcation involvement

Apical :

Tooth 36: apical radiolcency (PAI 3)

Tooth 37: normal PDL (PAI 2)

Diagnosis 10th of November 2011
Tooth 36
Pulpal: Root filled tooth (K04.19)
Periapical: Chronic apical periodontitis (K04.50)
Periodontal: Within normal limits

Treatment plan:
Retreatment of root filled tooth 36

Progress notes 11th of November 2011:
Access opening and location of three canal orifices with guttapercha
Mechanical: Bur, IrriSafe
NiTi handinsrumentation, BioRace
-MB 20/#40
-ML 20/#40
-D 20/55#
Chemical: 1% NaOCl, 16% EDTA,
2% chlorhexidinedigluconate
Intracanal medicament: $Ca(OH)_2$

Fig 11. Working length radiograph

Treatment 12th of December 2011
Sporadic symptoms from area 37
Filled with gutta-percha and AH plus, sealed with IRM.

Fig. 12 Masterpoint radiograph

Fig13. Final radiograph 12.12.2011

Follow-up: The patient had an appointment for follow-up which he canselled. He could tell on the phone that he still had some discomfort from the tooth 37 from time to time. Especially in the morning, but no pain when chewing or biting.

Discussion
The patient in the present case had an incomplete fractured tooth.
Historically, there have been a number of terms used to describe cracked teeth as well as a number of different classification schemes. The incompletely fractured tooth has for example been defined as ‘a tooth with a demonstrable fracture, but no visible separation of thesegments’(1)
Cracked tooth syndrome was first described by Cameron (2) in 1964. He defined a cracked tooth as “an incomplete fracture of a vital posterior tooth involving dentin and possibly pulpal tissue.” He also stated that these dentinal injuries may be asymptomatic, or associated with symptoms and even orofacial pain (2).
The American Association of Endodontists (AAE) has identified five types of cracks in teeth (3); craze line, fractured cusp, cracked tooth, split tooth, and vertical root fracture. Craze lines are visible fractures that only involve enamel. Fractured cusps originate in the crown of the tooth, extend into dentin, and the fracture terminates in the cervical region. A cracked tooth is defined by the AAE as a crack extending from the occlusal surface of the tooth apically without separation of the two segments. A split tooth is a crack that

extends through both marginal ridges usually in a mesiodistal direction, splitting the tooth completely into two separate segments. Vertical root fractures originate in the root, and are generally complete, although they may be incomplete.

The diagnosis of CTS has been based in the past exclusively on tooth symptomatology: localized pain during chewing or biting, unexplained sensitivity to cold, and pain on release of pressure (2,4). A clinical study by Rattcliff et al. (5) showed that a larger preparation, presence of an amalgam restoration, and parafunctional habits increased the risk for the occurrence of crack lines. However, in another study 60% of teeth with cracks had no restoration (6). In a recent review of the the literature the aim was to establish what evidence exists regarding the risk factors for cracked teeth and their prevention, diagnosis, and treatment (7). They found that there is no current evidence demonstrating which treatment option has the greatest success rate both from a restorative perspective and from a pulpal health standpoint.

Only one clinical trial conducted to support the use of extracoronal restorations in the treatment of cracked teeth was found in this review of the literature. The study by Krell and Rivera (8) reported the outcomes of symptomatic cracked teeth that were initially diagnosed with reversible pulpitis and treated with full coverage restorations. The outcomes of this study suggest that, if a crack is identified early in cases with a diagnosis of reversible pulpitis and a crown is placed, root canal treatment will be necessary in about 20% of the cases. One hundred and twenty seven teeth were followed after crown placement for 6 years. From a restorative standpoint, the full coverage treatment was successful for all 127 teeth because symptoms resolved (with or without RCT) and the teeth were retained. T his study did not compare success rates of crowns versus other restorative treatments so it is impossible to interpret the results to say that full coverage is the best treatment for CTS. In another clinical study, 40 patients diagnosed with reversible pulpitis and CTS and treated with direct composite restorations were followed for 7 years (9). The patients were divided into two groups and the restorations placed were either direct composite intracoronal restorations or direct composite cuspal overlay. After 7 years, no teeth were extracted due to restorative failures; therefore, all treatments were considered successful. However, three patients needed RCT, 50% of all restored teeth were still symptomatic after 6 months, and 25% were still symptomatic after 7 years. There were no restorative failures for the group with cuspal coverage, but the intracoronal group did have a mean annual failure rate of 6.0%. This difference was significant.

The authors suggested that bonded composite can be an effective treatment for painful cracked teeth, resulting in more than 90% of the teeth maintaining pulp vitality.

In a study by Brynjulfsen *et al.*,(10), thirty-two patients with poorly localized orofacial pain, were finally diagnosed with 46 incompletely fractured teeth. They found that the longer the duration of pain before the diagnosis of an incompletely fractured tooth was established, the more diffuse was the distribution of pain. Endodontic or restorative treatment relieved the symptoms in 90% of the patients, whilst persisting symptoms in 10% were considered part of an orofacial pain complex of obscure aetiology.

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Case 2

Treatment of the maxillary right second molar with irreversible pulpitis and the mandibular right second molar with primary infection in a patient with myelomatosis

Fig. 1 Frontal view

Patient: 62 year old Caucasian male
Chief complaint: Pain upper and lower jaw right side. High CRP without knowing the reason for this
Medical record: The patiend had myelomatosis (myeloplastic/myeloproliferating with eosinofili and trombocytemie) that was diagnosed in August 2010. He had been medicated with Glyvek since then, but had to stop this treatment because of joint necrosis.

- He gets easily infections
- High blood pressure
- Dry mouth (frequent waterdrinking at night)
- Dental fear
- *Medicaments:* Celosok 50 mg and Atacand 4 mg

Dental history: The patient himself contacted the dental clinic because of pain and discomfort upper and lower jaw right side. He had been troubled with fever for some period of time and high CRP. The doctors did not know the reason for this.
When he first came to the clinic, caries removal and IRM restaurations both teeth were made by general practitioner the day before endodontic treatment started. He had not visited any dentist the last years because of dental fear.

Clinical findings 28 th of February 2012
Soft tissue: normal finding, pale gingiva
Poor dental hygiene upper and lower front
Dental:

Tooth 17: temporary filling
Tooth 16: MOD composite filling
Tooth 37: temporary filling
Tooth 36: MOD amalgam filling

Fig. 2 Occlusal view tooth 17

Fig. 3 Occusal view tooth 47

Clinical tests 28 th of February 2012

	16	17	46	47
EPT (0-80)	15	-	20	45
Cold	+	-	+	+
Percussion Vertical/horizontal	-	+	-	-
palpation	-	-		-
PPD	3	3		3
Mobility	-	-		-

Table 1

Radiographic findings 28 th of February 2012 (the radiographs were taken before placement of IRM)

Dental: Deep caries teeth 17 and 36.
Tooth 17 has an MO amalgam filling
Tooth 35 has an insufficient root filling with radiopaque occlusal filling, with a gap between tooth and filling distally.
Periodontal: Within normal limits
Apical: Normal findings (PAI I)

Fig. 4 Periapical radiograph 28.02.2012

Fig 5 Periapical radiograph 28.02.2012

Diagnosis 28 th of February 2012

Pulpal: Tooth 17: Infected necrosis (K04.11)
Tooth 36: Chronic irreversible pulpitis (K04.03)
Periapical: normal
Periodontal: within normal limits

Treatment plan

Treatment of infected pulp tooth 17
Treatment of pulpitis tooth 36

Problem list

Anatomical considerations mesial and distal roots tooth 17

Treatment 28 th of February 2012

Tooth 17: Because of fear of bacteremia the patient was given antibiotic prophylaxis one hour before treatment. Before endodontic treatment, his doctor was contacted, who confirmed that this was advisable because of his poor health and risk of infections. The patient was after endodontic treatment referred to a dentist at the same clinic, and adviced to see a general practitioner regularly. He was also told about the importance of maintaining a good oral hygiene (see discussion). He wantet to do as much as possible in one visit, and thought long treatment time was better than more visits. Access cavity opening and location of first three, then the forth (mp) canal was located after preparation of the mb canal.
Mb and mp had one foramen apically.

Mechanical:
Because of S shaped mb and ml canals, extensive NiTi hand instrumentation were performed with #06-10 files in mb and ml canals until the files were no longer deformed and twisted
-MB 21/30#,-MP 20/30#-DB 21/40#
-P 21/50#
Chemical: 1% NaOCl, 16% EDTA, chlorhexidine-di-gluconate 2%
Filled with gutta-percha and AH plus, sealed with IRM

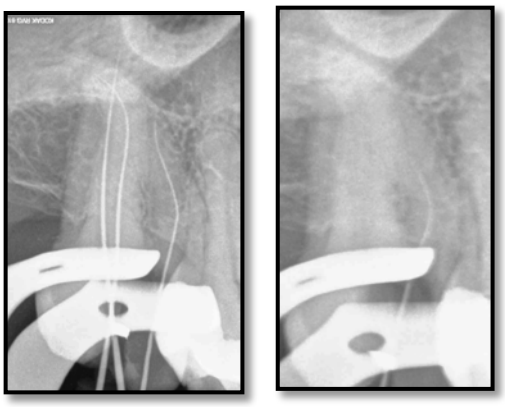

Fig. 6 Working length radiographs 28.02.2012

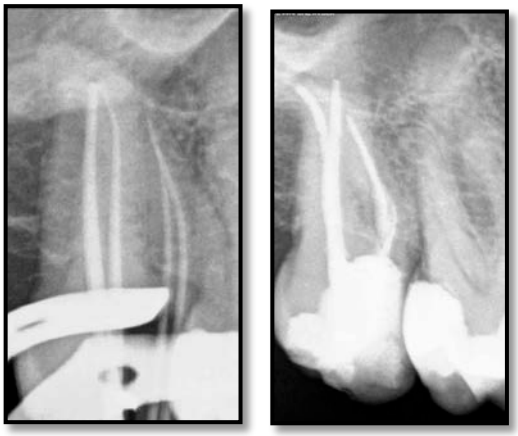

Fig 7. Masterpoint and final radiographs 28.02.2012

Prognosis

Endodontic: the prognosis seemed to be good

Tooth: the prognosis seemed too be good

Treatment 5 th of March 2012

Tooth 46: The patient had no symptoms from any teeth. His general health was better, and he had no fever.

Access opening and location of two mesial and one distal canals. Vital tissue apically in all canals.

Mechanical:

Bur . NiTi handinstrumentation. BioRace -MB 21/40#, -ML 20/40#, -D 21/50#

*Chemical:*1% NaOCl, 16% EDTA

Filled with gutta-percha and AH plus, sealed with IRM

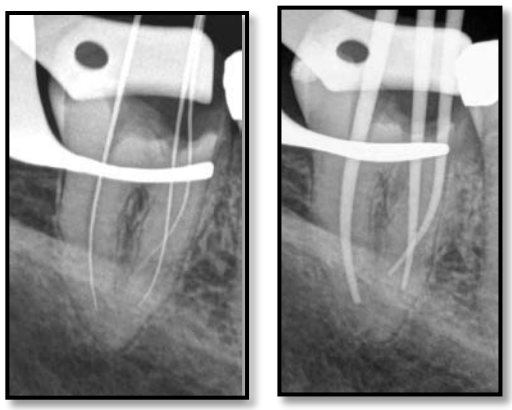

Fig 8. Working length and masterpoint radiographs 05.03.2012.

Fig. 9 Final radiograph 05.03.2012

Disussion

The patient in the present case had myelomatosis and immunosuppressed host defences. He was therefore given antibiotic prophylaxis before treatment, after consultation with his medical doctor.

The only established use of antibiotic prophylaxis in dentistry is in the attempt to reduce the potential consequences of bacteremias induced by dental treatment in certain medically at-risk patients. The principle indication for antibiotic prophylaxis for dental patients is the preven-tion of infective endocarditis in patients with specific medical conditions that are receiving specified dental treatments (1). Controversial indications

include dental patients with orthopedic prosthetic devices, indwelling catheters and impaired (immunosuppressed) host defenses.

Dental patients presenting for treatment with impaired host defenses (chemotherapy, organ transplant or tissue graft recipient, insulin-dependent diabetes, alcoholics) or patients with indwelling catheters (hemodialysis) may benefit from antibiotic prophylaxis if their white cell count is below 2,500 (normal = 4,000-11,000)

The most recent published guidelines from The American Heart Association for the prevention of infective endocarditis in medically at risk patients was published in April 2007 and represent a significant change from the previous guidelines (1). One of the stated reasons for the development of the current revised guidelines was that the risk of antibiotic-associated adverse events exceeds the benefit, if any, from prophylactic therapy.

The host defence against bacteria in the blood may be weakened by various diseases and conditions, e.g., neutropenia, asplenia, poorly controlled diabetes, end stage of renal disease, organ trans- plant, HIV infection and prosthetic joints or implants (2).

Debelian et al. (3, 4) found that when the root canal reamer ended 2 mm outside the root canal, 54% of the patients had cultivable microorganisms in the blood. When the instrumentation ended inside the root canal, i.e., 1 mm from the apical foramen, only 31% of the patients had bacteraemia. All these patients suffered from asymptomatic apical periodontitis. Phenotypic and genotypic profiles were provided from the bacteria and yeast recovered from root canal and blood. The methods used demonstrated identity between root canal isolates and blood isolates, suggesting that the root canal was the source of the blood isolates.

Savarrio et al. (5). found bacteraemia after root canal treatment in 30%. Pulse field gel electrophore sis showed that two pairs of strains from blood and root canal were identical.

The patient in the present case was advised to improve his oral hygiene to achieve better oral healt. An infected area is more due to cause bacteraemia than an uninfected area (6) and in the case with periodontitis, bacteraemia may be a common event induced by chewing and tooth brushing.

Oral bacteria have been demonstrated in atherosclerotic plaques, heart valves, aorta aneurysms, brain abscesses and joints (7a, b) .This particularly relates to periodon-topathogens such as Aggregatibacter actinomycetem- comitans, Treponema denticola and Porphyromonas gingivalis. While the main interest previously was directed against bacteraemia after dental surgery, authors have started to suspect that daily events such as chewing and tooth brushing can contribute to the cumulative exposure over time of the cardiovascular system to oral bacteria causing inflammation and thereby promoting atherosclerosis.

Both teeth in the present case were treated in one visit each without inlay of calcium hydroxide. The one-visit treatment in infected teeth, especially in teeth with apical periodontitis, is debated. The antimicrobial effects of calcium hydroxide have been evaluated by clinical studies where calcium hydroxide has been shown to successfully disinfect root canals following 1 month dressing in 97% of treated cases (8). In a later study by the same group, the effectiveness was confirmed even when the dressing was retained in the root canal for only 1 week (9). However, these results have not been reproduced in other studies. Cvek et al.,(10) Ørstavik et al.,(11) and Peters et al.,(12) demonstrated in clinical studies that calcium hydroxide did limit bacterial growth but did not totally eliminate the bacteria from the root canals. Waltimo et al.,(13) evaluated the clinical efficacy of

chemomechanical preparation of the root canals with sodium hypochlorite and interappointment medication with calcium hydroxide in the control of root canal infection and healing of periapical lesions. They found that calcium hydroxide dressing between the appointments did not show the expected effect in disinfecting the root canal system and in treatment outcome. Similar results had also been shown by Tang et al. ,(14) in a clinical study on the antimicrobial effect of calcium hydroxide used as interappointment dressing. Using an infective dentine model, (15) a study by Saleh et al.,(16) found that the use of AH plus and Grossmans sealer in root fillings in vitro was effective in killing *E. faecalis* in experimentally infected dentinal tubules within the zone of 300 mm around the root canal. Other endodontic sealers, as well as calsiumhydroxide, reduced the numbers, but did not effectively kill bacteria in infected dentinal tubules. Anather study by Özcan *et al.,(*17*)*confirmed that the AH Plus root canal sealer was effective in rendering the test *E. fae calis* cells in this ex vivo dentine infection model uncultivable or dead, whilst GuttaFlow was ineffective in achieving this.

An exhaustive literature search combined with specified inclusion criteria was performed to identify randomized or quasi-randomized controlled trials (RCTs or quasi-RCTs), comparing root canal treatment in single and multiple appointments (2 or more visits) in patients with infected root canals (18). According to the results from the present review, the healing rate of single- versus multiple-visit root canal treatment was similar for infected teeth. They also found that the prevalence of post-obturation pain was significantly lower in single-visit approach at short-term follow-up time. However, because the number of studies included in this review was limited, it might be preliminary to conclude that there is no difference between single- and multiple-visit root canal treatments in terms of postoperative complications for teeth with infected root canals

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Case 3

Treatment of the the mandibular right first molar with chronic apical periodontitis in a patient with anxiety disorders

Fig 1. Frontal view

Patient: 32 year old Asian female
The clinical photos are taken at recall, one year later, when she felt much better than the first visit one year earlier.
All upper front dental crown and dental crowns 46 shown in Fig 1. are made after the first visit to the post graduate clinic (Fig. 1, 8 and 9)
Chief complaint: No symtoms. She says she has has anxiety, she has dental fear, and she feels strong discomfort in the dental chair.
Medical record: The patient is a cathegory Group C patient (long-time sick with home care twice a week). She has anxiety and bipolar disorders
Medication: Rivotril (benzodiazepine)., Abilify (antipsychotic), Lamictal (anticonvulsant), Remeron (tetracyclic antidepressant), Afipran (against nausea and vomiting)
Dental history: The patient was referred from a specialist in pedodontics to the post-graduate endodontic clinic from the public dental service for treatment of tooth 46.

Clinical findings 04 th of September 2010

Soft tissue: Nomal findings
Dental: Tooth 48 has a composite filling
Tooth 46 has a MOD composite filling and an occlusal amalgam filling
Tooth 45 has a dental crown (clinical photos were not taken because of her condition).

Clinical tests 4 th of September2010

	45	46	48
EPT (0-80)	-	-	not taken
Cold	+	-	yes
Percussion Vertical/horizontal	-	both	-
Palpation	-	yes	-
PPD	3	4	3
Mobility	-	I	-

Table 1

Radiographic findings 04 th of September 2010

Fig. 2 Periapical radiograph

Dental:
Tooth 46 has an MOD radiopaque filling material and anatomically discrepancy distal root. The root canals seem to be calsified apically.
Tooth 45 is insufficient root filled, and has a dental crown
Periodontal: Within normal limits
Apical: Apical radiolucencies mesial and distal roots 46 (PAI 4) and tooth 45 has a widened PDL

Diagnosis 04 th of September 2010
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.5)
Periodontal: within normal limits

Treatment plan
Treatment of infected necrosis tooth 36

Problem list
The patient has problems having her mouth open during long periods of time. She has problems with vomiting during endodontic treatment because of her medication

Treatment 13th of October 2010
Access opening and location of three canals. Efforts were made to calm the patient. The canals negociated because of calsification. The patient felt sick and had to throw up during the endodontic treatment.
Mechanical: Bur, Irrisafe®
*Chemical:*1% NaOCl, 16% EDTA
Intracanal medicament:$Ca(OH)_2$
Temporary filling: IRM

Treatment 20 th of October 2010
Access opening. This time the DL canal was found just obove midroots..
The patient was calmer this time, but again she had to throw up during treatment. The treatment visit had to be as short as possible. Working lengths were achieved this time with EndoLift
Mechanical:
Irrisafe®, Endolift
NiTi handinstrumentation
MB24,5/30#, ML24,5/30#, DB 24,5/40#, DL24/40#
Chemical:
1% NaOCl, 16% EDTA
Chlorhexidine-di-gluconate 2%
Intracanal medicament: $Ca(OH)_2$
Temorary filling: IRM

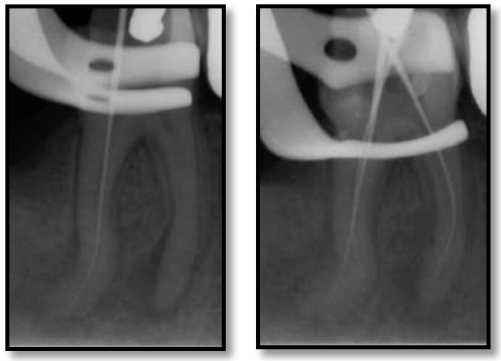

Fig. 3 and 4 Working length radiographs

Treatment 09 th of December 2010
No symptoms, not tender to percussion
filled with gutta-percha and AH plus, sealed with IRM

Fig. 5 Masterpoint radiograph

Fig. 6 Final radiograph 09.12.2010

Prognosis

Endodontic: The prognosis seemed to be good

Tooth: The prognosis seemed too be good

Follow up examination 06 th of September 2011 (12 months)

Radiograph showed healing of the periapical radiolucencies (PAI 2)

The patient was asymptomatic and Experienced no sensitivity to percussion or palpation tests. The PDL on 45 was widened, but there was no progression since last radiograph. She had new dental crown 36 and upper front.

Fig.7 Follow- up radiograph 06.09.2011

Fig. 8 Occlusal view

Fig. 9 Buccal view

Discussion

In the present case, instrumentation of the root canals was done with a #30 file as the last instrument.

Mechanical instrumentation of the root canal has been shown to reduce bacterial count even without irrigants or dressings (1). A combination of mechanical instrumentation and irrigation (2, 3) further reduced the number of microorganisms by 100 to 1000 times.

Shuping et al. (4) and Siqueira et al. (5) later confirmed the findings that larger file sizes are needed to allow the irrigating solution to reach the apex.

Larger instrumentation sizes not only allow proper irrigation of the canals but studies have shown a trend towards a decrease of remaining bacteria in the canal system. In a study by Ørstavik et al. (6) they found that root canals instrumented to larger-size reamers at the first appointment appeared to be rendered bacteria-free more easily.

Later studies confirmed this tendency (8, 9, 10,11), and Dalton et al. (7) also showed that with increasing file size, there was an increasing reduction of bacteria. Yared and Dagher (12) on the other hand, reported that a #25 file was as efficient as a #40 file for reducing residual microorganisms. However, in this study as well, no statistically significant difference was noted between the size 25 and 40 file groups after instrumentation, and after l-wk calcium hydroxide dressing.

Longitudinal studies have shown instrumentation to larger files sizes doesn't contribute significantly to the enhanced

statistical success for endodontic therapy. (13, 14, 15). However, many of these studies do not specifically evaluate the impact of a significant enlargement of the canal or of apical region with regards to clinical success.

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Case 4

Treatment of the mandibular right first molar with chronic apical periodontitis

Fig. 1 Frontal view

Patient: 52 year old Caucasian female

Chief complaint: Episodes of dull pain and discomfort when chewing.

Medical record: Non contributory
Dental history: The patient is referred to the post-graduate endodontic clinic by her general dental practitioner for endododontic treatment of tooth 46.
Clinical findings 20 th of January 2010:
Soft tissue: normal findings
Dental: Tooth 45 has an MOD composite filling. Tooth 46 has an MODBL composite builtup and caries can bee seen and along the composite margins.
Tooth 47 has a dental crown

Fig. 2 Buccal view

Fig.3 Lingual view

Clinical tests 20 th of January 2010

	45	46	47
EPT (0-80)	14	-	-
Cold	+	-	+
Percussion Vertical/horizontal	-	v +h	v
palpation	-	-	-
PPD	3	4	4
Mobility	-	-	-

Table 1

Radiographic findings 29 th of January 2010:
Dental: Tooth 46 has an MOD radiopaque filling material and calsified canals in the apical 1/3 of the root.
Periodontal: Within normal limits
Apical: Tooth 46 has apical radiolucencies mesial and distal roots (PAI 5) and lateral radiolucency mesial root

Fig 4. Periapical radiograph

Diagnosis 20 th of January 2010:
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50) and chronic lateral periodontitis (K.04.51)
Periodontal: Within normal limits

Treatment plan:
Remove caries and composite restauration
Treatment of infected pulp/apical periodontitis

Problem list:
Negotiating calcified root canals

Treatment 29 th of January 2010:
Removal of old composite filling and caries revealed that not much tooth substance was left. Access opening and location of three canals. It was not possible to access optimal working length because of calsified canals.
The patient was told that the endodontic treatment and tooth would have a poor prognosis. She did not want to extract the tooth before trying treatment.

*Mechanical:*Bur,Irrisafe®
NiTi handinstruments
MB: #40/13 mm, ML: #40/13 mm
D: #55/ 15 mm
*Chemical:*1% NaOCl, 16% EDTA
Chlorhexidine- di-gluconate 2%
Intracanal medicament:$Ca(OH)_2$
Temporary filling: IRM

Fig 5. Working length radiograph

Treatment 03 rd of February 2010:
Still pain and tender to percussion.
Instrumentation of lateral
canal in the mb canal to #25 with preconturated stainless steel instrument; pus when patency
Intracanal medicament:$Ca(OH)^2$
Temporary filling: IRM

Fig.6 instrument in lateral canal

Treatment 05 th of May 2010:
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

Fig. 4 Masterpoint radiograph

Fig. 5 Final radiograph 05.05.2010

Prognosis:

Endodontic: The prognosis seemed to be poor

Tooth: The prognosis seemed too be poor

Follow up examination 16th of September 2011 (16 months):

The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests. She had got a dental crown restauration.

Radiograph showed evidence of healing of the periapical radiolucencies apically both roots and lateral radiolucency mesial root (PAI 2). Radiograph shows placement of post in the distal root.

Fig. 6 Follow up radiograph 16.09.2011 (16 months)

Discussion

Strindberg (1) stated in 1956 that the highest success rate is obtained when the root filling is confined to 1 mm from the radiographic apex. In the study by Sjögren et al in 1990 (2) they found that the outcome in teeth with preopertive apical periodontitis was 68% when the root filling was more than 2 mm short of the radiographic apex, compared to 94% when the filling reached within 2 mm of the apex. Subsequently, most prognosis studies confirmed the practice of staying short of the apex to obtain the best treatment outcome.(3-9) In a study by Åkerblom et al (10), looking at the outcome of obliterated root-filled teeth that were rootfilled only one third of the rootlength,. Cinical and radiographic follow-up examinations were performed for 2 to 12 yr. The criteria for obliteration were: (a) The root canal was not patent for more than one-third of the root length. (b) No root canal lumen was visible on the radiographs apical to the instrumented portion. In teeth with preoperative periapical radio-lucencies, a success rate of 62.5% was noted.

An accessory canal is any branch of the main pulp canal that communicates with the external surface of the root. A lateral canal is an accessory canal located in the coronal or middle third of the root, usually extending horisontallyfrom the main root canal (11).

Ramifications can be observed anywhere along the length of the root, but they occur more commonly in the apical portion and in posterior teeth (12). In 73.5% of the cases, ramifications are found in the apical third of the root, in 11% in the middle third, and in 15% in the coronal third (13).

Ricucci et al. (14) reviews and reports on the histopathologic and histobacteriologic status of the tissue in lateral canals and apical ramifications (LC/AR) in diverse clinical conditions as well as in response to endodontic treatment. Serial sections from 493 human tooth specimens obtained by extraction or apical surgery. They found that LC/AR were observed in about 75% of the teeth. Chemomechanical preparation partially removed necrotic tissue from the entrance of LC/AR, whereas the adjacent tissue remained inflamed, sometimes infected, and associated with periradicular

disease. In cases in which lateral canals appeared radiographically ‘‘filled,’’ they were actually not obturated, and the remaining tissue in the ramification was inflamed and enmeshed with the filling material.

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Case 5

Treatment of the maxillary right lateral incisor and canine in patient with keratocystic odontogenic tumor

Fig 1. Frontal view

Patient: 65 year old Caucasian male

Chief complaint:

Pain and discomfort maxilla right side after surgery and removal of keratosystic odontogen tumor 2 months ago. Complains about palatal swelling right side

Medical record: Albyl E 160 mg

Dental history: first referred from private practinioner for implant treatment. Radiolucent area was observed maxilla right of postgraduate student in orthodontics September 2009. Tooth 12 did not respond to sensitivity test and the patient was referred to the Department of Oral Surgery and Oral Medicine.

CT scan showed a radiolucent area from tooth 11 to 15, expanding into the nasal floor and into sinus maxillaris. No buccal bone was evident. The patient was operated in October 2009, and histology confirmed the diagnosis odontogenic keratocystic tumor.

In December 2009 the patient had still symptoms and was referred to the post - graduate endodontic clinic for endodontic treatment of tooth12.

Clinical findings 15 th of December 2009:

Soft tissue: Palatal, fluctuating, soft swelling region 12,13

Dental:

Tooth 12: mesial composite filling
Tooth 13: sound
Tooth 14: amalgam filling
Tooth 15: MOD composit filling
Teth 16,17: dental crowns

Fig 2. Buccal view

Fig 3. Palatal view: palatal swelling region 13

Clinical tests 15 th of December 2009

	11	12	13	14
EPT (0-80)	-	-	-	15
cold	-	-	-	-
Percussion Vertical/horizontal	-	h	-	-
palpation	-	+	+	-
PPD	3	4	4	3
mobility	-	-	-	-

Table 1

Radiographic findings 15 th of December 2009

Dental: Tooth 12 has a mesial radiopaque

filling material
Tooth 13 is sound, Tooth 14 has an MO amalgamfilling , while tooth 15 has an MOD radiopaque filling material
Periodontal: Attachment 2/3-1/2
Apical : radiolucency from midline to mesially tooth 15, with extension to the nasal floor and into the maxillary sinus (PAI 5) (see description below)

OPG, CT maxilla fra 07.10.09:

I fremre, høyre del av maxillen, fra midtlinjen og omtrent til 2. premolar sees en osteolytisk prosess som ekspanderer både buccalt og palatinalt; særlig buccalt er det vanskelig å identifisere corticalisavgrensningen. Prosessen fyller ut hele alveolarprosessen og fremre del av maxillarsinus. Den ekspanderer også inn i nesekaviteten hvor det er vanskelig å se den corticale avgrensningen. Det er ingen påvisbar resorpsjon av røttene.

R: Stor benign cystisk prosess i høyre del av maxillen. Keratocystisk odontogen tumor?

Supplerende **MR på RH (22.10.09)** v/drs. Hopp og Smith viser en cystisk ekspansiv prosess som beskrevet på CT; signalmønsteret antyder en sannsynlig keratocystisk odontogen tumor (keratocyste).

Tore A. Larheim
professor
spes. kjeve- og ansiktsradiologi

Radiological description (in Norwegian) from the Department of Maxillofacial Radiology

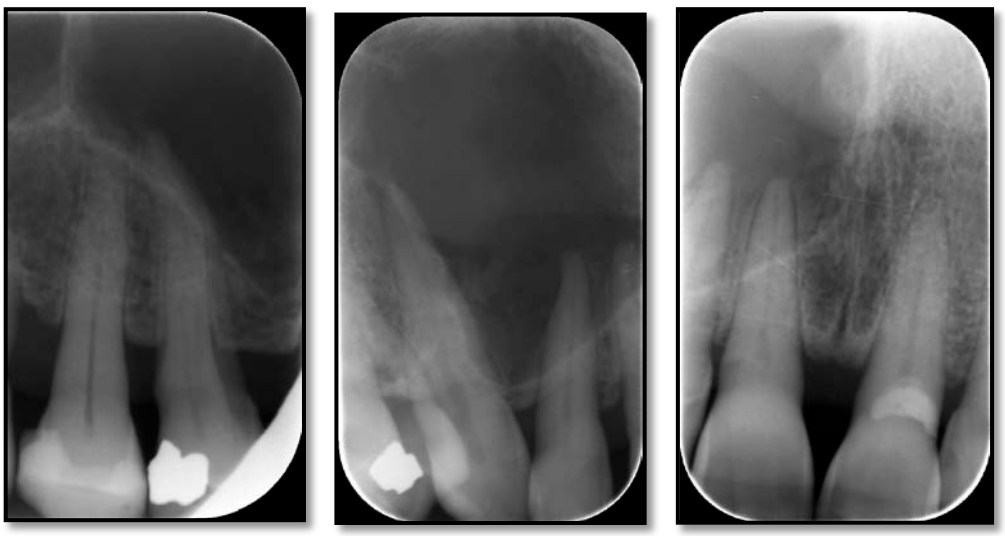

Fig. 4 Periapical radiographs taken at the post graduate clinic 15.12.2009

Fig 5 Panoramic view shows expansion of osteolytic process into the nasal floora and sinus maxillaris 20.10.2009.

Fig. 6 Axial CT scan demonstrates destruction of alveolar bone region 12-15

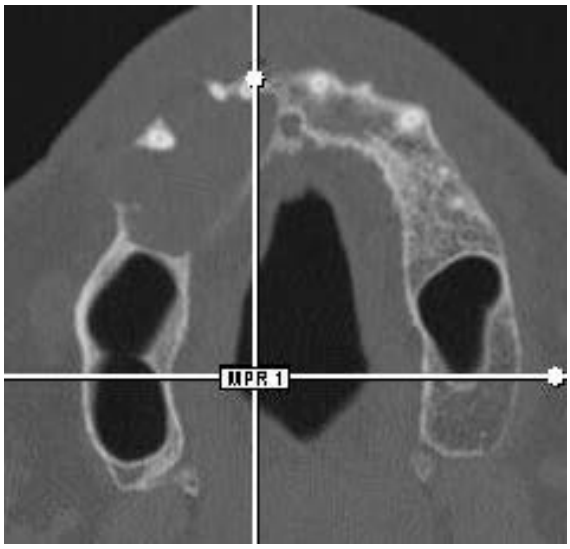

Fig. 7 Axial CT scan demonstrates expanded and thinned buccal and palatal cortex, with the buccal cortex being perforated (more cranial view than Fig 6)

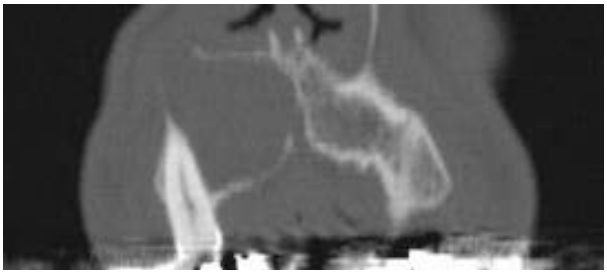

Fig. 8 Coronal CTscan demonstrates corticated process occupying part of maxillary sinus

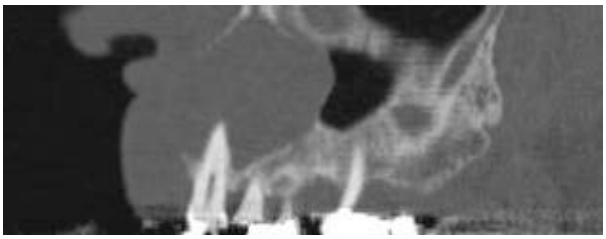

Fig. 9 Sagittal CT scan shows expansion of the tumor in maxillary sinus

Diagnosis 15 th of December 2009:
Pulpal: necrosis (K04.11)
Periapical: Chronic apical periodontitis tooth 12 (K04.50).
Keratocystic odontogenic tumor (K.09.0)
Periodontal: Chronic marginal periodontitis (K05.03)

Treatment plan:
Treatment of necrotic pulp tooth 12 and observation; especially tooth 13

Treatment 15 th of December 2009:
Access opening and location of one canal 12. No bleeding, but pale pink liquid in the canal (cystic liquid?). Length measurment on radiograph only.
Mechanical: Bur, Irrisafe®, NiTi hand instrumentation- 22 corr 22,5/#45
Chemical: 1% NaOCl,16% EDTA
Intracanal medicament: $Ca(OH)_2$
Temporary filling: IRM

Fig. 10 Working length radiograph

Treatment 20 th of January 2010:
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

Fig. 11 Masterpoint radiograph tooth 12

Fig. 12 Final radiograph 20.01.2010

Treatment 10 th of February 2010:
The patient had an appointment regarding control tooth 13. This time it did not respon to sensivity tests.

Clinical tests 10 th of February 2010

	11	12	13	14
EPT (0-80)	35	-	-	50
cold	-	-	-	-
Percussion Vertical/horizontal	-	-	-	-
palpation	-	-	+	-
PPD	3	3	3	3
mobility	-	-	-	-

Access opening and location of one canal tooth 13. No bleeding, but like 12; pale pink liquid in the canal

Mechanical:
Bur and IrriSafe
Stainless steel hand instrumentation
27,5mm/ #50
*Chemical:*1% NaOCl,16% EDTA
Intracanal medicament: $Ca(OH)_2$

Fig. 13 Working length radiograph tooth 13

Treatment 04 th of March 2010
No symptoms, not tender to percussion, the palatal swelling has dissapeared.
Filled with gutta-percha and AH plus, sealed with IRM

Fig. 14 Masterpoint radiograph

Fig. 15 Final radiograph 04.03.2010

Follow up examination 02nd of February 2011 and 12th of April 2012 (13 and 27 months):

After 13 months: The patient was asymptomatic and experienced no sen sitivity to percussion or palpation tests. Radiograph showed evidence of healing of the periapical radiolucency (PAI 3), although a mottled appearance in the bone structure can be seen.
After 27 months: Continuous PDL (PAI 2), and the Department of Oral Surgery and Oral Medicine confirm healing after resurgey 6 months earlier (Fig. 21).

Fig. 16 Follow up radiograph 02.02.2011

The patient have had 6 months recalls at the department of oral and maxillofacial surgery, and in March 2011 a recidiv was observed in the same region. A CT scan was taken. Biopsi was taken during surgery and histology confirmed that the radiolucency was a recidive of the odontogenic keratocystic tumor.

Fig. 17 Panoramic view; before re-surgery in March2011

Fig. 18 Panoramic view; After re-surgery October 2011

Fig. 19 Follow up radiograph 12.04.2012

Fig 20: Follow up radiograph 12.04.2012

Fig. 21 Panoramic view shows healing of the lesion, most likely with scar tissue12.04.2012

Discussion

The odontogenic keratocyst has been one of the most controversial pathological entities of the maxillofacial region since Philipsen first described it in 1956 (1).The World Health Organization recent classification of Head and Neck Tumors reclassified the keratocyst as a benign neoplasm, recommending the term ‘‘keratocystic odontogenic tumor” (KCOT) (2).

The surgical treatment method for KCOT is categorized as conservative or aggressive. Conserva-tive treatment is cyst-oriented and includes marsu-pialization or enucleation with or without curettage. Its advantage is preservation of anatomical structures, including teeth, which is advocated because KCOTs commonly present in younger patients. It has been asserted that a conservative approach is applicable to all age.

Aggressive treatment addresses the ―neoplastic nature‖ of KCOT and includes peripheral ostectomy, chemical curet- tage with Carnoy’s solution or en bloc resection. The aggressive modalities have been recommended for large KOCTs and recurrent lesion (3).

In a clinopathological study (4), three hundred and twenty-two patients (192 male and 130 female) with cystic lesions of the jaw were successfully diagnosed and treated. One hundred and fifty-five (48%) were radicular cysts, 80 (25%) were dentigerous cysts, 23 (7%) were odontogenic keratocyst (=keratocystic odontogenic tumor), 19 (6%) were eruption cysts, 16 (5%) were traumatic bone cysts, and 29 (9%) were non-odontogenic cysts. The angiogenesis was assessed in KCOT, DC, and normal oral mucosa using CD-105 antigen . It was demonstrated that CD-105 antigen is strongly expressed in microvessels of KCOT compared to DC and normal oral mucosa, suggesting that the cyst wall of KCOT plays a role in the neoplastic behavior of the lesion. These finding can further support the WHO decision (3) recommending the term KCOT, as it better reflects its neoplastic nature.

Although prognostic factors based on clinico-pathologic and immunohistochemical findings for deter-

mining the potential for recurrence of KCOT still remains unclear, its use for determining the potential for recurrence of KCOT after surgical treatment may become important to successfully manage this neoplasm's aggressive behavior.

The key element for future management of KCOTs will probably be based on thorough knowledge of the biological basis of this tumor, thereby enabling a more tailored treatment approach (5).

KCOTs are benign but locally aggressive, it is generally accepted that they arise from the remnants of the dental lamina which persist in subepithelial tissues including bone after the completion of odontogenesis (6).

Since the KCOT is a relatively uncommon lesion, epidemiological data vary considerably. KCOTs probably account for between about 2% and 11% of all jaw cysts and can occur at any age; many data suggest a bimodal age distribution around the third and sixth decades (7).

One of the clinical features of the KCOTs that causes difficulty in management is their tendency to recur after treatment. Reported rates of recurrence range from 3% to 60% (7). Many theories have been proposed to account for the high level of re- currence of these lesions. Firstly, the cyst lining is delicate and remnants can be left behind after surgical removal, satellite cysts (from odontogenic epithelial residues) or daughter cysts (from out pouching's of the main cyst lining) may develop into new cysts after removal.

A Cochrane systematic review (8) sought high level evidence on the effectiveness of managing keratocystic odontogenic tumours by comparing the effectiveness of surgical interventions and adjuncts for their treatment. No eligible studies for inclusion were found.

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Case 6

Treatment of a maxillary left first molar with iatrogenic furcal perforation

Fig. 1 Frontal view

Patient: 47 year old Central Asian male

Chief complaint: Episodes of dull pain

Medical record: Non contributory
Dental history: The patient was referred to the post-graduate endodontic clinic from the student clinic for endododontic treatment of tooth 26 and treatment of furcal perforations
Clinical findings April 29 th 2010
Soft tissue: Gingival recession mandibular firs centrals
Dental: Tooth 24: amalgam filling
Tooth 25 is sound
Tooth 26 has a temporary filling and distal composite filling.

Fig. 2 Buccal view

Fig. 3 Palatal view

Clinical tests 29 th of April 2010

	24	25	26
EPT (0-80)	26	16	-
Cold	+	+	-
Percussion Vertical/horizontal	-	-	v + h
palpation	-	-	-
PPD	3	3	4
Mobility	-	-	I

Radiographic findings 29 th of April 2010
Dental: Tooth 25 is sound
Tooth 26 has a radiopaque filling material
Periodontal: Attachment is 2/3
Apical: Tooth 25 has continuous PDL while 26 has widened PDL apically and mesially along the mesial root (PAI 3)

Fig. 4 Periapical radiograph 29.04.2010

Radiographic findings: history (graduate clinic)

Fig. 5 Periapical radiograph before treatment at graduate clinic17.03.2009

Fig. 6 Instruments in perforation in the furcal area 01.02.2010

Diagnosis 29 th of April 2010
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50)
Periodontal: Chronic marginal periodontitis (K05.03)

Treatment plan:
Treatment of infected pulp/apical periodontitis and closure of perforations with MTA

Problem list : Perforations

Treatment 29 th of April 2010:
Access opening and location of mb and mp canals. These canals were previously not instrumented. Perforations were seen two places in the furcal area close to the mesial canals. These perforations had happened during instrumentation and not access preparation, and the perforations were discovered when the student should obturate the tooth at the last visit.

Bleeding from both perforations was stopped with $Ca(OH)_2$. MTA was placed over both perforations. A moist cotton pellet was placed over the MTA for 15 min., so the cement could set.

Mechanical: Bur, Pre RaCe, Irrisafe® , NiTi handinstrumentation
MB: #40/20 mm, MB2: #40/19 DB: #40/19 (corr. from 20)mm
P: #50/ 19 (corr. from 20)mm
Chemical: 1% NaOCl, 16% EDTA, Chlorhexidine- di-gluconate 2%
Intracanal medicament: $Ca(OH)^2$
Temporary filling: IRM

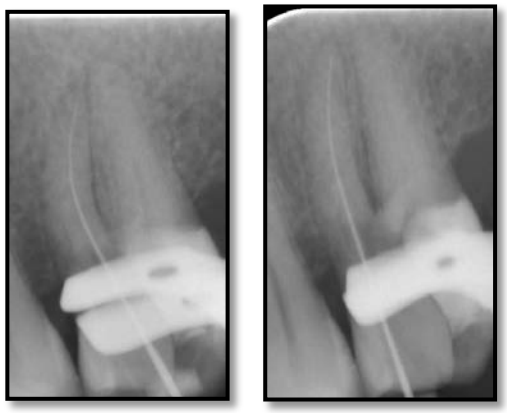

Fig. 7 Working length radiographs

Fig 8. Working length radiograph

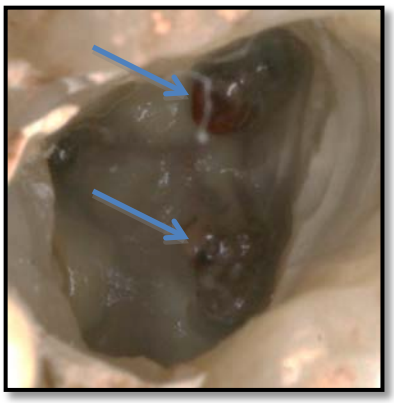

Fig 9 shows two perforations (arrows) close to the mb and mp canals

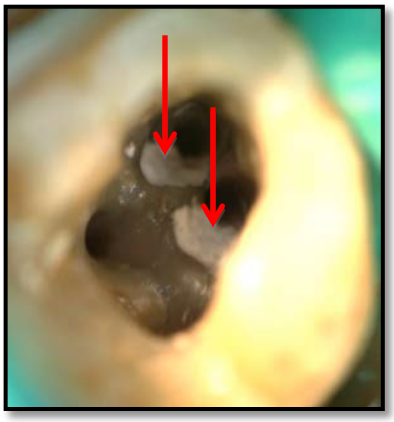

Fig. 10 MTA in perforations (arrows)

Fig. 11 MTA over perforations and Cavit in mb, mp and db canals

Treatment 10 th of June 2010

No symptoms, not tender to percussion

Filled with gutta-percha and AH plus, sealed with IRM

Fig. 12 Masterpoint radiograph

Fig 13. Final radiograph 10.06.2010

Prognosis

Endodontic: the prognosis seemed to be good

Tooth: the prognosis seemed too be poor because of large perforations near the crestal bone, time delay before restauration, (see discussion) and little remaining tooth substance.

Follow up examination 10th of June 2011 and 10th of April 2012 (12 and 22 months)

Radiograph showed periapical healing mesial root. (PAI 2)

The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests. The tooth had been restored with a composite filling

Fig. 14 Follow up radiograph 11.06.2011 (12 months)

Fig. 15 Follow up radiograph 10.04.2012 (22 months)

Discussion

Over a period of 11 years, 55 root perforations were recorded at the Department of Cariology and Endodontics, School of Dentistry, University of Bergen (1). Forty-four of these perforations were diagnosed during endodontic or prosthodontic treatment, seven during routine endodontic recall and four prior to endodontic retreatment. Perforations occurred in all tooth groups, but were more common in the maxilla than in the mandible. Of the perforations occurring during endodontic treatment, nine were located in the midroot level and 11 in the apical third of the roots. Further analysis of specific procedures related to perforations demonstrated that attempts to negotiate calcified canals accounted for 11 out of 26 perforations (42%). Nine out of 26 (35%) occurred during root canal instrumentation, and the remaining six (23%) resulted from attempts to locate calcified canal orifices.

Factors that affect treatment prognosis of perforation repair include the level, location and size of the perforation, the time delay before perforation repair and the material used to seal the perforation.(2). According to Tsesis and Fuss (3) the prog- nosis of old and large crestal perforations is questionable. Perforations near the crestal bone are susceptible to epithelial migration and rapid pocket formation and treatment of these has a low success rate.(4,1).

Pitt Ford et al (5) were the first investigators who used MTA for repair of furcal perforations. They showed that cementum was generated underneath the material in most treated teeth, in contrast to the teeth whose furcation perforation sites were repaired with amalgam. These authors reported that when a perforation is left untreated for a period of time and becomes contaminated, the healing rate after perforation repair with MTA is significantly reduced.

Mineral trioxide aggregate (MTA) has later been successfully used to repair perforations (6,7,8,9).

MTA has not only been shown to be biocompatible but has also demonstrated the ability to allow regeneration of dental hard tissues (10,11). Main et al (7) showed that MTA had the ability to promote regeneration of cementum, thus facilitating regeneration of the periodontal apparatus.

Torabinejad et al (12) claimed that the reduction of bacterial leakage when MTA was used as the repair material was result of its sealing ability rather than an antimicrobial property of the material. However, Eldeniz et al.,(13) evaluating the antibacterial activity of leachable components from selected root- end filling materials, found that set samples of ProRoot MTA cement completely inhibited Pseudomonas aeruginosa and delayed or limited growth of Enter. faeca-lis. Similar results were obtained in other studies where the antimicrobial properties of MTA were evaluated.(14,15).

In a dye leakage study, Lee et al (16) investigated the sealing ability of MTA in lateral perforations and reported that MTA allowed significantly less leakage than IRM or amalgam. In a study by Lodiene et al., (9), the percentage of leaking samples was significantly higher in resin composite than in the other groups and the negative control group ($p < 0.05$). SEM inspection revealed the presence of bacteria in all leaking specimens. Bacteria were observed along the filling-dentine interface as well as in dentinal tubules at some distance from the filling. They concluded that the resin composite material leaked significantly more than the MTA and glass ionomer cements when used to repair large furcation perforations. Bacteria could penetrate into dentine even at a distance from the perforation filling. Bacterial contamination may occur through furcation canals or dentinal tubules as well as along the material-dentine interface.

A recent case series investigated the prognosis of teeth with perforations in the furcation or within the cervical third of roots repaired with GMTA. They reported that 9 of 10 teeth healed after 5 years (17).

A two year follow-up in the present case shows satisfactory conditions, both clinically and radiographically. It appears that MTA repair in the furcal area has made a bacteria-tight seal, so no reinfection has taken place.

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Case 7

Treatment of a mandibular right lateral incisor with two canals

Fig. 1 Frontal view

Patient: 42 year old Asian female

Chief complaint: No complains

Medical record: Non contributory

Dental history: The patient was referred to the post-graduate endodontic from the graduate clinic for endododontic treatment of tooth 41. Teeth 31,32 and 33 were endodontically treated in 2007.

Clinical findings 07 th of September 2011:

Soft tissue: Normal findings

Dental: Dental crowns all teeth upper and lower jaws and teeth 31, 32,33 had occlusal composite fillings

Fig. 2 Buccal view. Tooth 41 blue arrow

Clinical tests 07 th of September 2011

	41	42	31
EPT (0-80)	-	-	-
Cold	-	-	-
Percussion Vertical/horizontal	-	-	-
palpation	-	-	-
PPD	4	4	4
Mobility	II	II	I

Radiographic findings 07 th of September 2011

Dental: Tooth 42 has a dental crown, the root canal appears to be calsified. Teeth 41, 31,32 are all root filled

Periodontal: attachment is 2/3-1/2

Apical: Tooth 42 has an apical radiolucency (PAI 4)

Teeth 32,31 have widened PDL (PAI 2)

Fig 3. Periapical radiograph

Radiographic findings: history

Teeth 31, 32 and 33 were endodontically treated in 2005 by post graduate student.

Old radiographs show that the periapical radiolucensies have healed (PAI 3 in 2005 and PAI 2 in 2007). In the radiograph from 2007, it is evident that tooth 41 has widened PDL apically.

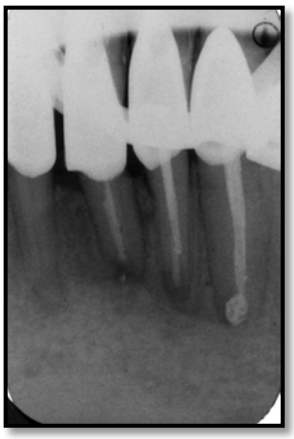

Fig. 4 Periapical radiograph 31.05.2005

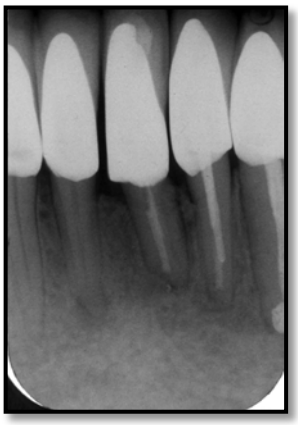

Fig. 5 Periapical radiograph 29.01.2007

Diagnosis 07 th of September 09.2011
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50)
Periodontal: Normal

Treatment plan
Treatment of infected pulp/apical periodontitis

Problem list
Negotiating calcified root canals

Treatment 07 th of September 2011
Access opening and location of one canal. The canal is necrotic.The lingual canal was localised with preconturaed #15 after full instrumentation of the first canal. Working length was achived with EndoLift #08.Care was taken not to remove too much tooth substance because of fragile root and overextended crown.

Mechanical:
Burs, Irrisafe® ,EndoLift, NiTi handinstruments, BioRace B: #30/14 mm, L: #30/13 mm
Chemical: 1% NaOCl, 16% EDTA, Chlorhexidine- di-gluconate 2%
Intracanal medicament: $Ca(OH)_2$
Temporary filling: IRM

Fig 6 and 7. Working length radiographs, working length not achieved in lingual canal in Fig. 6.

Treatment 28 th of September 2011
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

Fig 9. Masterpoint radiograph

Fig 10. Final radiograph 28.09.2011

Prognosis

Endodontic: The prognosis seemed to be good

Tooth: The prognosis seemed too be good

Follow up examination 17th of April 2012 (6 months)

Radiograph showed widened PDL 41, but there was evidence of healing (PAI 2)

The patient was asymptomatic

And experienced no sensitivity to percussion or palpation tests.

Fig. 11 Follow up radiograph 17. 04.2012

Discussion

Several methods have been used to investigate the anatomy of root canals, for example, direct observation with the aid of a microscope (1), sectioning techniques (2) filling with an inert material and then decalcifying (3), and the examination of radiographs (4).

The incidence oftwo canals in the case of mandibular incisors has been reported to be as low as 4 per cent (5), and as high as 41.4 per cent (6), with an average value of approximately 30 percent. Madeira and Hetem found the incidence to be 11.5%. (7),Vertucci found it to be 25.7% (8a, b), Green 20% (9) and Kartal and Yanikuglo 45%(10) . In the study by Kartal and Yanikuglo (10) , it was found that two canals connect in the apical third in 37% of the cases and reach the apex as one canal.

In the present case the mandibular 41 had a type III (1-2-1) in Vertuccis classification.

In the study by Kartal and Yanikuglo (10), the found central and lateral mandibular incisiors with this classification in 20% of their samples.

Four hundred and fifty-five extracted mandibular incisor and 340 extracted mandibuiar premolar teeth were radiographed to assess the inci- dence of twin canals as visualized on radiographs taken in the mesio-distal direction. The ability to detect the presence of these twin canals by viewing radiographs taken in the sundard bucco-lingual direction was then assessed. Using the guideline that 'disappearance or narrowing infers division' when viewing these radiographs resulted in a failure to diagnose one-third ofthe twin canals (11).

In a study on two hundred extracted mandibular inisors undertaken to determine the most effective horizontal beam angulation for the diagnosis of twin canals, it was found that the 20° right and 30° left horizontal beam angulations showed significantly more accurate diagnosis of twin canals than the ortho-radial view (0°) (12).

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Case 8

Retreatment of a mandibular left canine with sinus tract and primary treatment of a mandibular right first premolar with acute apical periodontitis

Fig. 1 Frontal view

Fig. 2 Occlusal view

Fig. 3 Buccal view; gutta-percha point in sinus tract

Patient: 55 year old South American male

Chief complaint
Episodes of strong pain left side.
Medical record: Allergic to pollen.
Ulcerus
Medication: Nexium
Dental history: The patient was referred to the post-graduate endodontic clinic from the graduate clinic for surgical treatment of tooth 43. This tooth was treated at the post-graduate clinic 18.11. 2009 and the root-filling seemed dense and good.
The patient had been at a dental emergency clinic 01.05 2012 (three days before) because of pain and swelling in the same area. He got Apocilling 660 mg prescription for one week. No endodontic treatment was done.
Clinical findings 04 th of May 2011
Soft tissue: Sinus tract buccaly region 43
Dental: Tooth 43 has lost part of the occlusal filling.
Tooth 44 has a temporary filling IRM
Teeth 41 and 42 have composite fillings incisally
All teeth have erosive occlusal damages and dental crowns in upper jaw.

Clinical tests 04 th of May 2011

	42	43	44
EPT (0-80)	23	-	54
Cold	+	-	no
Percussion Vertical/horizontal	-	both	-
palpation	-	yes	-
PPD	3	4	3
Mobility	-	I	-

Radiographic history

Fig. 4 Periapical radiograph before endodontic treatment 22.09.2009

Fig. 5 Periapical radiograph after endodontic treatment 18.11.2009

Radiographic findings 04 th of May 2011

Dental: Tooth 43 is root filled, lack of top filling. Tooth 44 has a radiopaque filling material filling that appears to be close to the pulp.

Periodontal: Within normal limits

Apical: Apical diffuse radiolucency tooth 43 that extend masially towards the marginal bone (PAI 5). The radiopaque guttapercha point can be seen in the lesion

Fig. 6 Periapical fistulogram 04.05.2011

Diagnosis 04 th of May 05.2011

Pulpal: root filled tooth (K04.19)

Periapical: Chronic apical periodontitis with sinus tract (K04.62)

Periodontal: within normal limits

Treatment plan:

Retreatment tooth 43

Problem list:

Most likely a persistent infection

Treatment 04 th of May 2011:

Access opening and location of one canal. Pus in the canalspace

Mechanical: Bur, Irrisafe®, BioRace , NiTi handinstrumentation 24/60#

Chemical: 1% NaOCl, 16% EDTA

Chlorhexidine-di-gluconate 2%

Intracanal medicament: $Ca(OH)_2$

Temporary filling: IRM

Fig 7. Working length radiograph tooth 43

The patient had since last visit (16.05.2011) had an episode of acute pain in the same region. Another post graduate student had diagnosed tooth 44 with acute apical periodontitis (K.04.4). The radiograph that date showed apical radiolucency 44 (PAI 3). The tooth still tested positive on EPT (68). It was irrigated with 16% NaOCl, placement of eugenolpellet and IRM.

Treatment 31st of May 2011:
Mechanical:
Bur, Irrisafe®, BioRace
19 mm adjusted to18,5/50#
*Chemical:*1% NaOCl, 16% EDTA
Chlorhexidine-di-gluconate 2%
Intracanal medicament: $Ca(OH)_2$

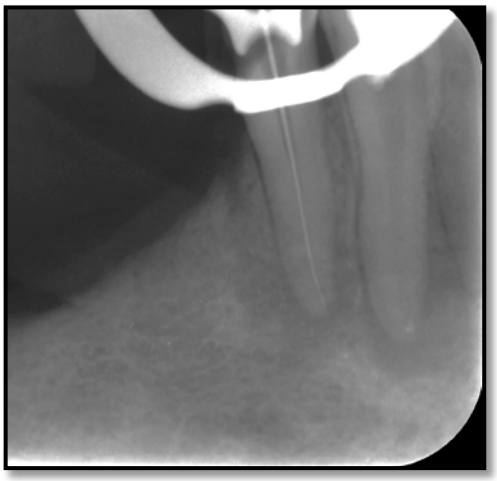

Fig. 8 Working length radiograph tooth 44

Treatment 15th of June 2011:
No symptoms, not tender to percussion
Teeth 43 and 44 were filled with gutta-percha and AH plus, sealed with composite

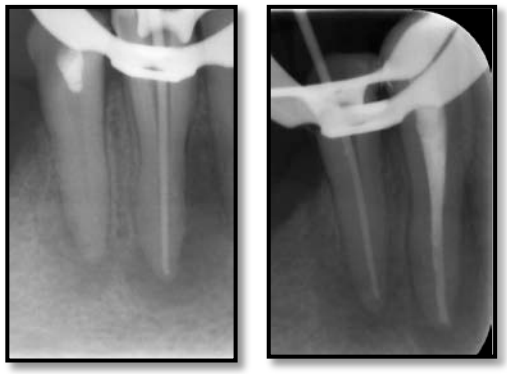

Fig. 9 Masterpoint radiographs 15.06.2011

Fig. 10 Final radiograph 15.06.2011

Prognosis:
Endodontic: The prognosis seemed to be good for 44 and uncertain for 43
Teeth: The prognosis seemed too be good for booth teeth if permanent restaurations are made, most preferably dental crown because of erosion problems.
Thee graduate student was informed about extensive erosions, further treatment and refunds

Follow up examination 16th of October 2011 and 13th of March 2012 (4 and 9 months):
The patint had not experienced pain, the sinus tract was closed and he had achieved dental crowns on all teeth lower jaw.
Radiograph showed evidence of

healing of the periapical radiolucency 43 (PAI 2 after 9 months) and healing of the periapical radiolucency 44 (PAI 1)
The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests.

Fig. 11 Follow up radiograph 16.10.2011

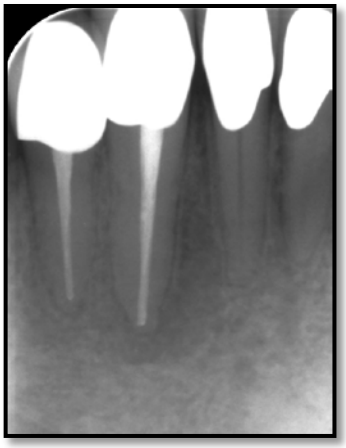

Fig. 12 Follow up radiograph 13.03.2012

Fig. 13 Buccal view 13.03.2012

Discussion

Discussion

One of the species of bacteria frequently retrieved from previously root-filled canals is *Enterococcus faecalis*, which is a Gram-positive facultative anaerobe. In previous studies, the prevalence of *E. faecalis* in failed endodontic cases ranged between 24 and 70%, when culture-based techniques were used (1-6).
Numerous studies demonstrate the hardy nature of enterococci, which are able to resist various intracanal medications and adapt to harsh environmental conditions (7). In a recent study (8), The genetic relationship between *E. faecalis* from root canals and isolates from the different host sources was determined using pulsed-field gel electrophoresis. In 16% (8⁄50) of the patients, enterococci were collected from the root canal samples. The genetic analysis showed that the isolates from the root canals were not related to those from the normal gastrointestinal microflora. None of these patients had enterococci in their saliva samples. They concluded that endodontic infections with *E. faecalis* are probably not derived from the patient's own normal microflora, which indicates that these infections are of exogenous origin.
Bacteria associated with persistent apical in- fections such as *E faecalis* are often more challenging to eradicate. Several studies have reported that calcium hydroxide is not effective in eliminating *E faecalis*, which is often associated with persistent endodontic infections.(9-13).Such bacteria can proficiently invade the dentinal tubules and have the ability to survive and buffer the high pH produced by calcium hydroxide.(14)
Kuruvilla and Kamath (15) compared the separate and combined effects of 2.5% NaOCl and 0.2% chlorhexidine in vivo. Teeth treated with chlorhexidine and NaOCl combined showed the greatest reduction in the number of micro-organisms. In a clinical study, it was reported that treatment of root canals with IKI prior to calcium hydroxide dressing

did not significantly reduce the amount of bacteria but that it might reduce the frequency of persisting strains of *E faecalis*. The ability of calcium hydroxide to kill *E faecalis* have been shown to be improved by combining it with IKI or chlorhexidine (16)
The impact of mechanical agitation of the hypochlorite solutions on tissue disso-lution was found to be very important by Moorer and Wesselink (17) who emphasized the great impact of violent fluid flow and shearing forces caused by ultrasound on the ability of hypochlorite to dissolve tissue. However, the mechanisms involved are not completely understood (18). Optimizing the concentration, temperature, flow, and surface tension can improve the tissue-dissolving effectiveness of hypochlorite even 50-fold (19).

The patient in the present case had a tooth with acute apical periodontitis.

In an early study by Haapasalo et al., (20) black-pigmented Bacteroides spp. were identified in acute and clinically asymptomatic symptomatic cases of apical periodontitis. They suggested that the presence of B. gingivalis and B. endodontalis in necrotic root canal is closely related to an acute infection and that fermentative BP Bacteroides species, including B. intermedius, are frequently present both in symptomatic and asymptomatic infections. It is also possible that the risk for persisting symptoms may be greater when BP Bacteroides species are part of the infective flora.

It has been demonstrated that many bacterial species are virulent or more virulent when in association with other species (21–24).

In a recent study using molecular techniques (25), samples were taken from necrotic root canals of teeth with symptomatic or asymptomat apical periodontitis in addition to teeth with chronic apical abscesses. They found that basically the same species were highly prevalent in the different clinical conditions evaluated, and none of these most prevalent taxa were positively associated with symptoms. However, the results revealed that the taxa identified formed different partnerships and associations in samples from cases with or without pain. They speculated in that some bacterial associations can result in a more virulent multispecies community.

The composition of the microflora might vary according to the geographic location of the study (26,27) .

Nine months follow-up in the present case shows healing of the periapical lesion, and no clinical symptoms. The intracanal infection appears to be controlled, most likely because of additional chemical (chlorhexidine-di-gluconate 2%) and mechanical cleaning of the root-canal , and a tight coronal restauration.

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Case 9

Retreatment of a mandibular left first molar with post and two separated instruments

Fig. 1 Frontal view

Fig. 3 Buccal view

Patient: 53-year-old Caucasian female

Chief complaint

Episodes of pain and discomfort. Periodically tender when chewing and biting. No symptoms at the moment.

Medical record: Elevated cholesterol blood level, depressive.

Medication: Symvastetin (hart) and Sypralex (antidepressant).

Dental history: referred to the post-graduate endodontic clinic from the graduate clinic for retreatmet of tooth 36**.**

Clinical findings 25 th of October 2011

Soft tissue: normal findings

Dental: Tooth 36 has a composite restauration and secondary caries. Several teeth have erosions, lost fillings and discolorations.

Fig. 2 Lingual view

Clinical tests 25 th of October 2011

	33	34	36
EPT (0-80)	34	26	-
Cold	+	+	-
Percussion Vertical/horizontal	-	-	-
palpation	-	-	-
PPD	3	3	3
Mobility	-	-	I

Radiographic findings 25 th of October 2011

Dental: Tooth 36 has an inadequate root filling. Separated instruments in mb and ml canals, post in distal root, radiopaque restauration

Periodontal: Within normal limits

Apical: Diffuse apical and lateral radiolucency mesial root tooth 34 (PAI 4) and widened PDL (PAI 3) distal root

Fig. 4 Periapical radiograph 25.10.2011

Diagnosis 25 th of October 2011
Pulpal: Infected necrosis (K04.11)
Root filled tooth (K4.19)
Periapical: Chronic apical periodontitis (K04.50) (PAI 5)
Periodontal: within normal limits

Treatment plan
Retreatment, removal/bypassing separated instruments and removal of post

Problem list
Removal/bypassing separated struments and removal of post

Treatment 25 th of October 2011
Composite and caries were removed. Removal of post in distal canalwith ultrasound (Fig. 5). Bypassed intrsument in ml canal. Not possible to negociate distal canal due to calsification

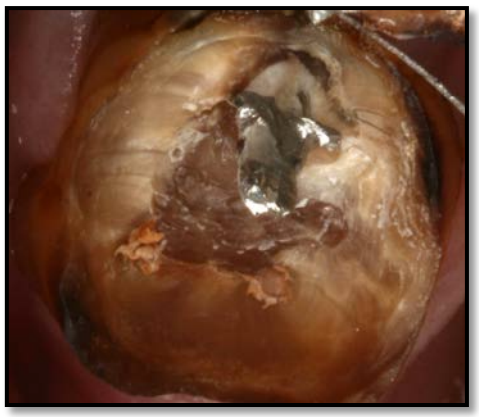

Fig. 5 Post in distal canal, old guttapercha in ml and mb canals

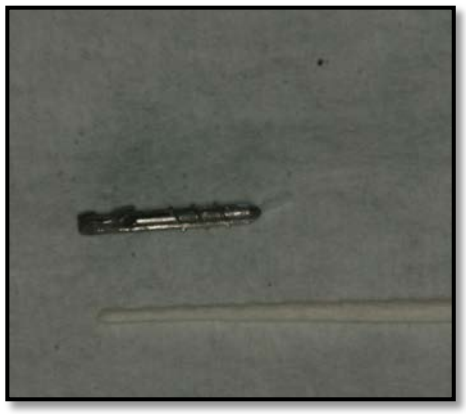

Fig. 6 Removed post

Fig. 7 Removed separeted instrument (arrow), paperpoint and cotton pellet in ml and d canals respectively

Mechanical: Bur, Irrisafe®, Ultrasound in distal canal. Prebent stainless steel in ml canal because of step -ML 14/35#
Chemical:
1% NaOCl
16% EDTA
clorhexidine-di-gluconate 2%
Intracanal medicament:
$Ca(OH)_2$

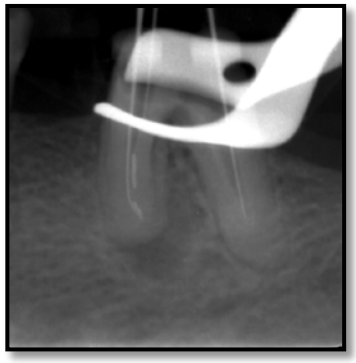

Fig 8. Working length radiograph ml canal

Treatment 22 th of November 2011
Since last vistit, the patient had an espisode of exacerbation while she was in Denmark. She got Apocillin 660 mg prescription for one week.
Access preparation and removal of separated instrument in mb canal was performed with modified Gates glidden and ultrasound.
During ultrasound removal of instruments, the separated instrument in the ml canal moved more apically in the canal. Mesial root had one apical foramen.

Mechanical: Bur, Irrisafe®
Prebent stainless steel because of
Step in mb canal -MB 13/35#
*Chemical:*1% NaOCl, 16% EDTA, clorhexidine-di-gluconate 2%
Intracanal medicament:
$Ca(OH)_2$

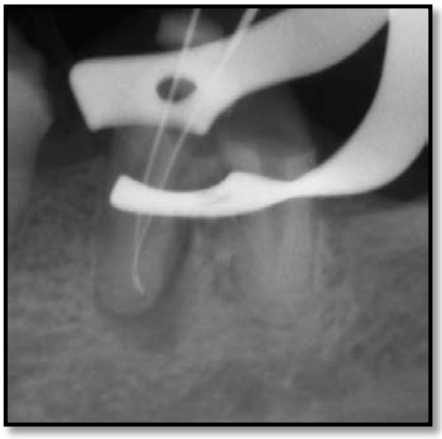

Fig. 9 Working length radiograph mesial canals

Treatment 10 th of January 2012
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, warm guttapercha in distal canal. Sealed with IRM

Fig. 10 Masterpoint radiograph

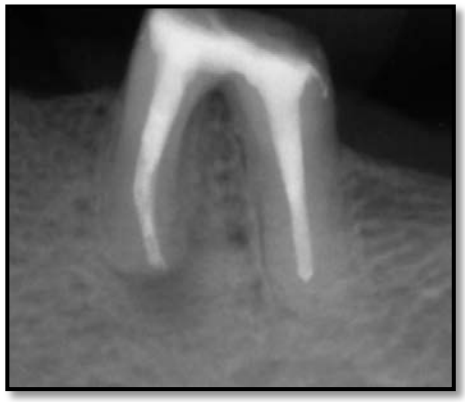

Fig. 11 Final radiograph 10.01.2012

Prognosis
Endodontic: The prognosis seemed to be good
Tooth: The prognosis seemed too be uncertain

Follow up examination 13 March 2012 (2 months)
Radiograph was taken by graduate student before placement of dental crown. No clinical tests were done.

Radiograph showed evidence of healing of the periapical radiolucency (PAI 3)

Fig 12.Follow up radiograph 13.03.2012

Discussion

The use of vibrating mechanical energy has been embraced by endodontists as a highly efficient method of dislodging and removing metallic posts from the cemented or bonded interface of the root canal wall (1–4).

It is generally accepted that external root temperature increases that exceed 10 ^{0}C produce irreversible bone and attachment damage as well as dehydration effects on dentin, often resulting in resorption and necrosis (5, 6).

As evidence accumulates in the literature for the considerable heat buildup and transfer that occur during ultrasonic vibration in the removal of posts, a number of studies during the past few years have cautioned the practitioner regarding the need for adequate coolant to counter heat buildup (7-9).

Ruddle (10) proposed a technique for the removal of broken instruments using Gates Glidden drills (size 3 or 4) to prepare a circumferential “staging platform” at the coronal aspect of the ob- struction. Attention must be paid during preparation of a staging platform, because a size 3 or 4 Gates Glidden may perforate or weaken a root, for instance the mesial (11, 12) and distal root (13) of mandibular molars,

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Case 10

Non surgical - and surgical endodontic treatment of a maxillary right central incisor with radicular cyst

Fig. 1 Frontal view

Fig 2. Occlusal view

Patient: 23 year old African male

Chief complaint:

Tender when chewing and biting
Sensation of slight mobility

Medical record: Non contributory

Dental history: Referred to the post-graduate endodontic clinic from his private practitioner.

Endodontic treatment was started earlier by another postgraduate student when I saw the patient for the first time. Calsium - hydroxide inlay in the canal.

The patient thought that he might have had a trauma 15 years ago, but was not sure.

Clinical findings 19 th of May 2009:

Soft tissue: Normal findings

Dental: Tooth 21 has a yellowish discoloration and a palatal temporary filling. All other teeth are sound except for tooth 15, which are root filled and fractured to the gingival level.

Fig. 3 Frontal view

Fig. 4 Palatal view

Clinical tests 19 th of May 2009

	11	21	22
EPT (0-80)	24	-	22
Cold	+	+	+
Percussion Vertical/horizontal	-	v	-
palpation	-	+	-
PPD	3	5	4
Mobility	-	I	-

Radiographic findings 19 th of May 2009:
Dental: Tooth is 11 sound, tooth 21has a radiopaque filling material coronally and 1/3 into the canalspace.
Pulpal: Tooth 21 has a wide canal, open apex?
Periodontal: Attachment 2/3
Apical: Tooth 11 has a continuous PDL while 21 has apical circumferencial radiolucency (PAI 5)

Fig 5. Periapical radiograph 19.05.2009

Diagnosis 19 th of May 2009:
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50)
Periodontal: Normal

Treatment plan:
Treatment of infected pulp/apical periodontitis and most likely apical surgery

Problem lis:t
Open apex?
Large lesion and the tooth has already had an intracal inlay with $CaOH_2$ for one month; compromised healing?

Treatment 19 th of May 2009:
Access opening and location of a wide canal. Liquid in the canal (cystic?).
Mechanical: Irrisafe®, NiTi handinstruments/Haedstrøm
One canal: #70/23 mm,
*Chemical:*1% NaOCl, 16% EDTA
Chlorhexidine- di-gluconate 2%
Intracanal medicament: $Ca(OH)_2$

Fig 6. Working length radiograph

Treatment 18 th of June 2009:
Less symtoms, less liquid in the canal,
but too much liquid that final treatment could be done

*Mechanical:*Irrisafe®
*Chemical*1% NaOCl, 16% EDTA
Chlorhexidine- di-gluconate 2%
Intracanal medicament:
longterm $Ca(OH)_2$

Treatment 03rd November 2009:
Less symptoms, but still tender to percussion. To make surgery easier, it was decided to put an apical plug with MTA angulus. Moist cotton pellet. IRM.

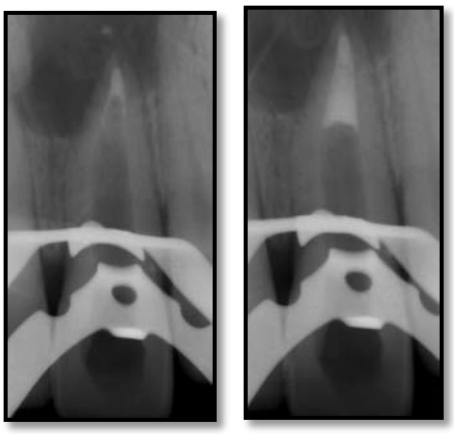

Fig 7. MTA apically

Treatment 10 th of November 2009

Still tender to percussion, not tender to palpation. Filled with warm gutta-percha and AH plus, sealed with composite restauration. It was decided to perform surgery

Fig. 8 MTA apically and warm gutta-percha above

Fig. 5 Final radiograph 10.11.2009

Prognosis

Endodontic: The prognosis seemed to be poor

Tooth: the prognosis seemed too be uncertain due too thin root canal walls

Follow up examination 07 th of September 2010 (9 months)

Due to studies abroad, he was not able to come for surgical treatment before the date above. Radiograph showed no healing of the periapical lesion. The patient was asymptomatic and experienced no sen sitivity to percussion or palpation tests. The radiographs showed no sign of healing. (PAI 4.) A lamina dura was evident around the lesion

Fig. 6 Panoramic view 07.09.2010 shows distinct radiolucent area from 11 distally to 23 mesially extending to the nasal cavity

Fig. 8 Follow up radiograph 07.09.2010

Surgical treatment 11 th of November 2010:

1. Anaesthesia 2. Sulcular flap from 13M-24D, with a buccal releasing incision 13D 3. Osteotomy 4. Biopsy and microbiological sample 5. Apical root resection without retrograde filing 6. Haemostasis 7. Sutures 8. Prescription of analgesics and post-op information.

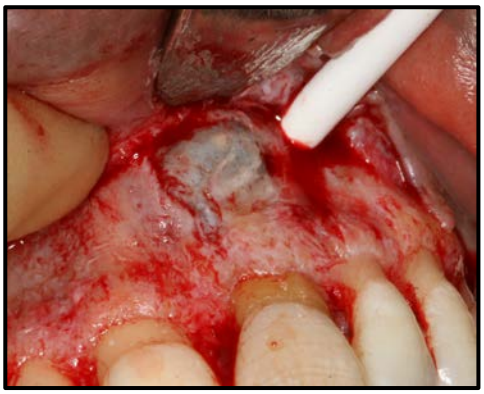

Fig. 9 Elevation of flap reveals destruction of buccal bone and bluish cystic cavity wall

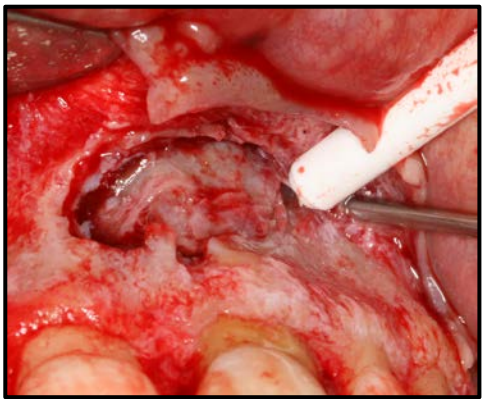

Fig. 10 Elevation of flap reveals fenestration of buccal bone and a bluish cystic cavity wall

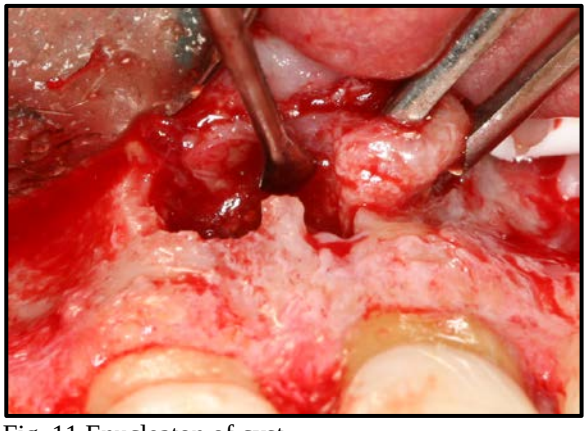

Fig. 11 Enucleaton of cyst

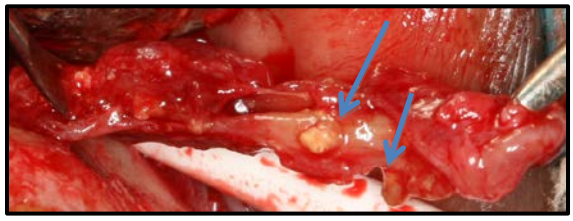

Fig. 12 Enucletion of cyst reveals several sulfur granules (arrows)

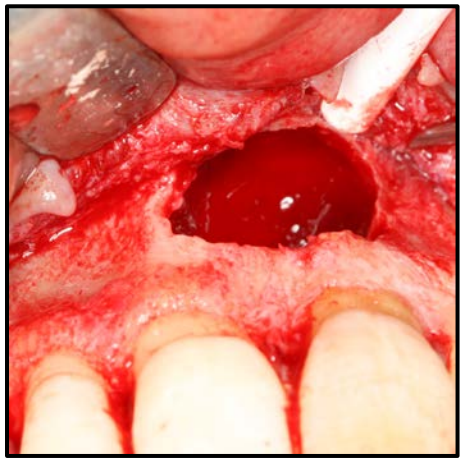

Fig. 13 Cavity after cleaning

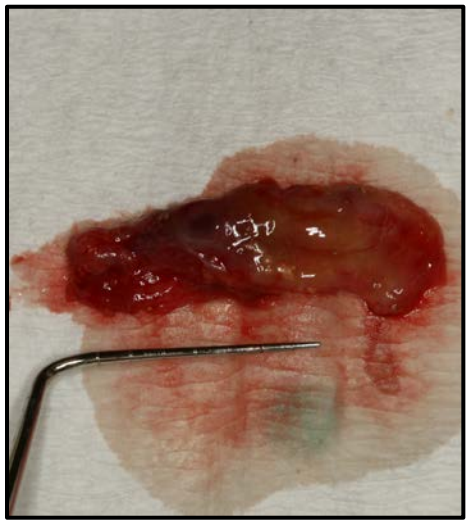

Fig. 14 Cystic lesion

Fig. 15 Lesion on formalin. Sulfur granules can be seen

Fig. 16 After suturing

Fig. 17 Treatment result11.11.2010

Prognosis

Endodontic: seemed to be good

Tooth: seemed to be uncertain

Treatment 16 th of November 2010:

The patient returned after one week for suture removal. He could tell about extraoral swelling and colorchange of the skin (blue). He was symptom-free at suture removal,

Fig. 18 Removal of sutures after one week

Histological findings (Hanna Strømme Koppang)

Epithelized lumen (15 mm in diameter), with fibrous capsula. Inflammatory cells. Foreign body material. Histological findings show most likely a radicular cyst

Bacteriological findings (with DNA-DNA hybridization): Diagnostic service, Institute of Oral Biology

- *Streptococcus intermedius*
- *Actinomyces israelii*
- *Actinomyces viscosus*
- *Treponema denticola*
- *Prevotella intermedius*
- *Eubacterium sabbureum*

Result SEM (scanning electron microscopy). Institute of Oral Biology- Steinar Stølen

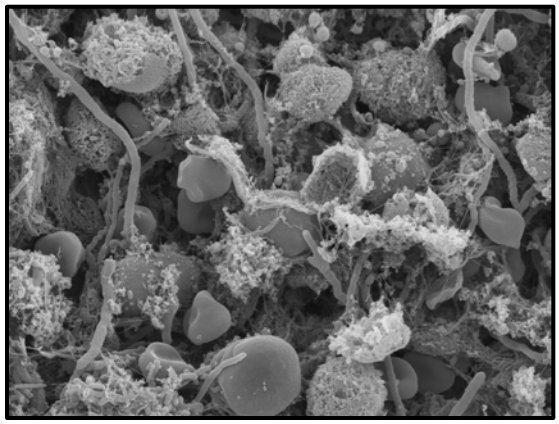

Fig. 19 SEM photo shows microorganisms, mostly long rods, and extracellular material

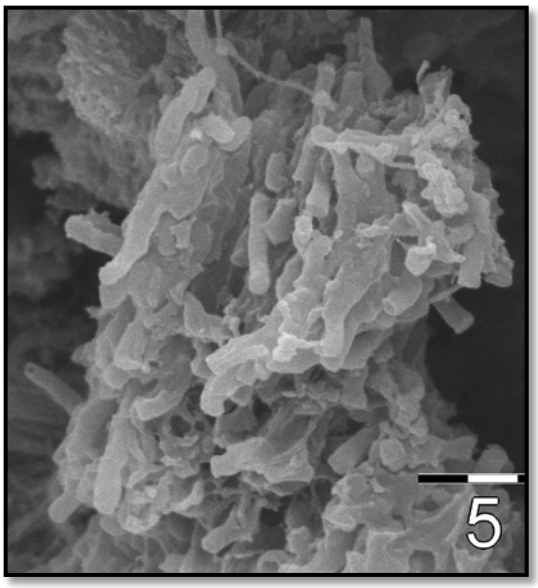

Fig. 20 SEM photo shows microorganisms embedded in extracellular material

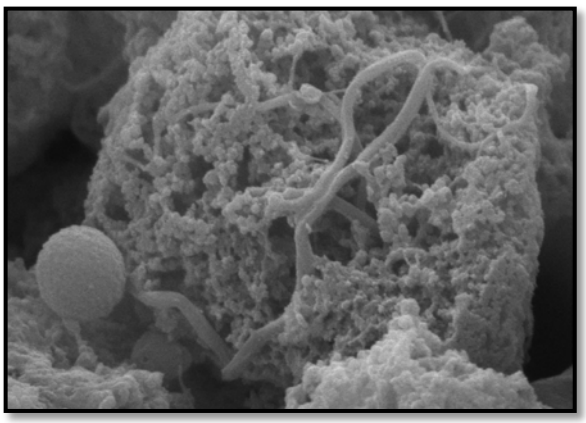

Fig. 21 SEM photo shows microorganisms; cocci and rods embedded in an extracellular material

Prognosis:

Endodontic: The prognosis seemed to bee uncertain

Dental: the prognosis seemed to be uncertain due to thin root canals walls

Follow-up examination 17 th of February and 11th of October 2011 and 12th of April 2012 (3 , 11 and 16 months)

The radiographs showed healing of the periapical lesion. The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests.

Fig. 22 Follow up radiograph 17.02.2011 (3 months)

Fig. 23 Follow up radiograph 11.10.2011 (11 months)

Fig. 24 Follow up radiograph 12.04.2012 (16 months)

Discussion

Occasionally, strands of epithelial cells are present within the periapical lesion. These cells are thought to originate from strands of Malassez in the periodontal ligament. In the inflammatory process, cytocines and growth factors are released that bring the epithelial cells to proliferate. It is estimated that about 50% of longstanding lesions contain epithelium (1, 2). However, serial

sections revealed that only 15% of these lesions were actually periapical cysts (2).

Radicular cysts develop as a sequel to apical periodontitis. A cyst is a cavity with epithelial lining filled by fluid or semisolid material surrounded by a dense connective tissue.
Radicular cysts are devided into true cysts and pocket cysts (2). In a pocket cyst there is direct continuity between the cyst cavity and the root canal space, whereas no such direct communication is present with the true cyst.
Apical periodontitis cannot be differentially diagnosed into cystic and non cystic lesions based on radiographs alone (3, 4).
The radiologic appearance of the periapical tissues after surgical therapy has some unique features (5).
Surgical treatment of periapical lesions may sometimes result in the formation of scar tissue, leaving a permanent defect in the bone that is visible on the radiograph (Fig 23,24 in the present case).
Typical findings indicative of scar tissue are - according to Molven et al (6): the reduction of the bone defect but persistence of a widened periodontal membrane. A pattern of irradiating fine bone trabeculae in contact with the root end. A solitary defect surrounded by compact bone but without root contact.

In the present case, several sulfur granules were seen in the periapical lesion (Fig. 12, 15). Bacteria in the sulfur granules seem to live in biofilm, and in the SEM pictures microorganisms are seen embedded in- and interspersed between an extracellular matrix. (Fig. 10-21 present case).
The biofilm community lifestyle provides microorganisms with a series of advantagesand skills that are not observed for individual cells living in a free-floating (planktonic) state including establishment of a broader habitat range for growth; increased meta- bolic diversity and efficiency; protection against competing microorganisms, host defenses, antimicrobial agents, and environmental stress; and enhanced pathogenicity (7, 8).
Apical periodontitis is a biofilm-induced disease (9). In situ investigations using optical and/or electron microscopy have allowed observations of bacteria colonizing the root canal system in primary or persistent/secondary infections as sessile biofilms covering the dentinal walls (11–16). Extraradicular bacteria have been found in biofilms adhered to the apical root surface (17, 18, 19) or located within the body of the inflammatory lesion, usually forming cohesive actinomycotic colonies (20).So far, it has been suggested that the main bacterial species implicated in independent extraradicular infections are Actinomyces species and Propionibacterium propionicum in a pathologic entity named apical actinomycosis (20-23). However, tudies have reported the extraradicular occurrence of a complex microbiota (24, 26).

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Case 11

Surgical endodontic treatment of the right maxillary first incisor with sinus tract

Fig. 1 Frontal view

Patient: 64-year-old Caucasian female
Chief complaint:
Tender when chewing and biting. Sporadically swollen with pus
Medical record: Rheumatisms (Lupus)
High blood pressure
Prednisolon 2,5 mg/week
Aprovel 150 mg (for hypertension)
Carredilol 25 mg (α og β bloccant)
Efexor Depot 75 mg (for depression)
Dental history: endodontically treated by specialist in endodontics. Referred to the postgraduate clinic for surgical treatment because of persisting suppurative sinus tract
Clinical findings 08 th of February 2011
Soft tissue: Sinus tract buccaly 11
Dental: dental crowns

Fig. 2 Occlusal view

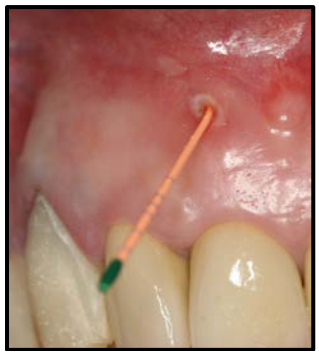

Fig. 3 Occlusal view. Guttapercha in sinus tract

Clinical tests 08 th of February 2011

	12	11	21
EPT (0-80)	65	-	-
Cold	+	-	-
Percussion Vertical/horizontal	-	v and h	-
palpation	-	+	-
PPD	4	4	4
Mobility	-	I	-

Radiographic findings 08 th of February 2011
Dental: Teeth 21,11 are root filled and have dental crowns
Periodontal: Attachment 2/3
Apical: Tooth 11 apical radiolucency (PAI 3).

Fig. 4 Periapical fistulogram

Diagnosis 08 th of February 2011:
Pulpal: Root filled tooth (K04.19)
Periapical: Apical periodontitis with sinus tract (K04.62)
Periodontal: normal

Treatment plan:
Apical surgery 11 with retrograde filling

Problem list:
Esthetic wound healing

Surgical treatment 09 th of March 2011:
1. Anaesthesia 2. Sulcular flap from 13D-22D, with a buccal releasing incision 13D 3. Osteotomy 4. Biopsy and microbiological sampling 5. Apical root resection 6. Retro-preparation 7. Haemostasis 8. Retrograde filling with M T A 9. Sutures 10. Prescription of analgesics and post-op information.

Inspection of the root apex before root resection revealed extrusion of gutta-percha, most likely because of resorptions. It was also evident that here was a crack in the root surface starting apically with extension coronally but which ended at the distal aspect some mm from the apex.

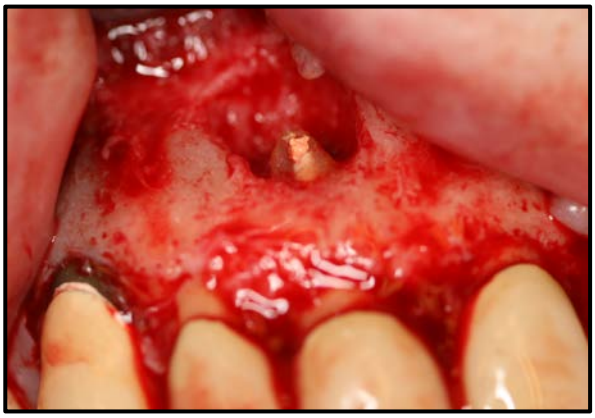
Fig. 5 After ostetomy


Fig. 6 removal of granulation tissue

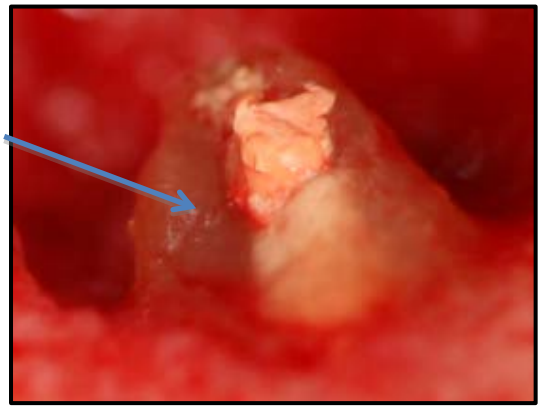
Fig. 7 Root apex with extrusion of gutta-percha, Arrow shows crack line apically.

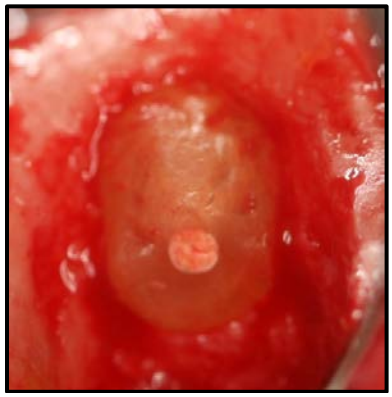
Fig. 8 After root resection

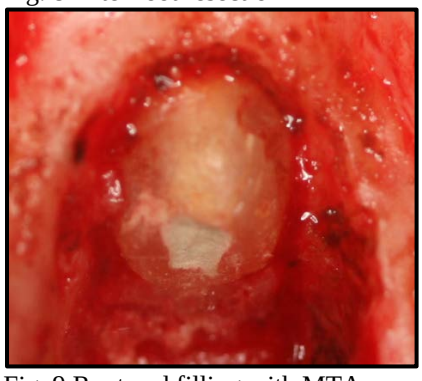
Fig. 9 Root end filling with MTA

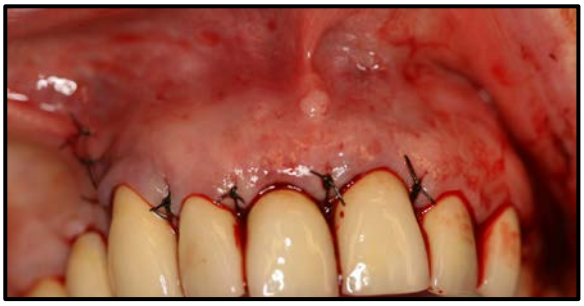
Fig. 10 After suturing


Fig. 11 Treatment result 12.03.2011

Treatment 17 th of March 2011

The patient returned after one week for suture removal. She had not experienced any post-op problems. The sinus tract was closed, but there was irritation in the papillas and retraction of the periodontium (Fig.12).


Fig. 12 Removal of suteures after one week

Histological findings (Hanna Strømme Koppang)

Granulation tissue with intense chronic inflammation. Connective tissue with moderate chronic inflammation and foreign body material

Bacteriological findings (with DNA-DNA hybridization): Diagnostic service, Institute of Oral Biology

Treponema denticola
Prevotella nigrescens
Campylobacter rectus
Treponema socranskii spp. socranskii
Fusobacterium nucleatum spp. vincentii
Porphyromonas gingivalis

Result SEM (scanning electron microscopy). Institute of Oral Biology- Steinar Stølen

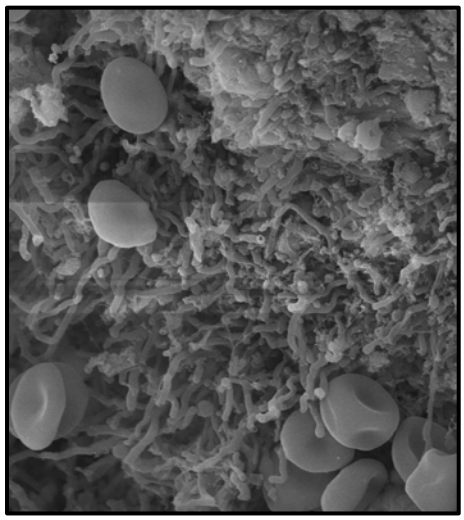
Fig. 13 SEM photo shows branching filamentous rods and extracellular material

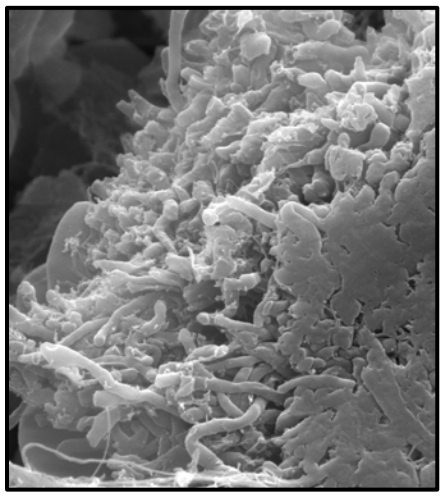
Fig. 14 SEM photo shows rods and extracellular material

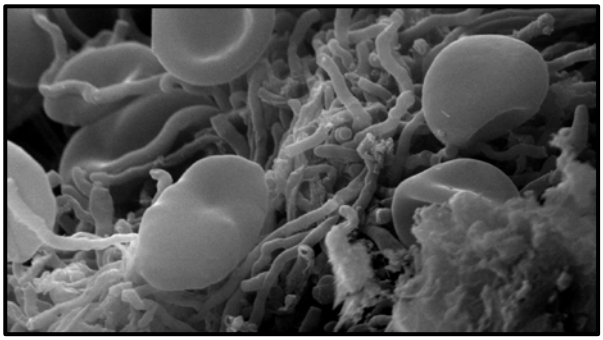

Fig 15 SEM photo shows filamentous organisms, blood cells

Prognosis

Endodontic: The prognosis seemed to bee good

Dental: The prognosis seemed to be good

Follow-up examination 6 th of September 2011 and 13 th of March 2012, (6 and 12 months)

The tooth was asymptomatic, but in September 2011 clinical gingival conditions were not satisfactory because of gingival retractions. In March 2012, twelve months after surgery, the clinical conditions were satisfactory, and the patient was pleased. Radiographic signs of healing with a continuous PDL surrounding the apex. No sinus tract is evident.

Fig. 16 Healing; 6 months

Fig. 17 Healing; 12 months

Fig. 18 Follow up radiograph 6 months

Fig. 19 Follow up radiograph 12 months

Dicussion

In the present case, a crack line was seen in the apical 1/3 of the root starting apically and extendet only about 5 mm coronally. Vertical root fracture (VRF) originate from the apical end of the root and propagate coronally and is one of the frustrating complications of root canal treatment, which often results in tooth extraction (1).

The root fracture might occur as result of a microcrack or craze line that propagates

with repeated stress application by occlusal forces. Bier *et al* (2) showed dentinal damage (microcracks) in teeth that were prepared with several nickel-titanium (NiTi) rotary instruments with the exception of S-Apex rotary files. They found the highest defect ratio when ProTaper was used, whereas no defect was observed with hand files. In another study by Yoldas et al (3) they found that all rotary files created microcracks in the root dentin of extracted teeth, whereas the SAF file and hand instru- mentation presented with satisfactory results with no dentinal microcracks.

It has been shown that root canal filling procedures could also create cracks (4). Shemesh et al (5) observed significantly more dentinal defects (microcracks) in teeth that were obturated with spreader than when no spreader was used.

The size of the initial spreader may be important to prevent extra loading. In the study by Piskin et al., (6) they revealed that spreader size equal to the master apical file decreased the fracture resistance of maxillary incisor roots. A size 25 spreader on the other hand, which reached to within 1–2 mm of the working length, did not influence the fracture resistance of maxillary roots.

Kim *et al* (7) have found a potential relationship between the design of NiTi instruments and the incidence of vertical root fractures. They concluded that file design affected apical stress and strain concentrations during root canal instrumentation.

The flap design is important for the healing of the wound after apical surgery and many factors play an important role when the choice of design is considered. Amongst them are marginal gingival recession, necrosis of the gingival papilla, ease of access, maintenance of an adequate blood supply, trauma to the wound margins, and scarring (8).

It is crucial that the submarginal incision is used only when there is a broad zone of attached gingiva with a minimum of 2mm In addition; the underlying apical lesion or surgical bony access must not extend to the flap margins. (9). This is because a sufficient amount of marginal attached gingiva in place is important to avoid deprivation of blood supply to this unreflected tissue and risk its necrosis. Such tissue breakdown will lead to a major recession with a compromised aesthetic result

Kamper et al. (8) looked at three different flap designs used in apical surgery where the purpose was to evaluate the clinical and histological features of healing of three types;

They found that the intrasulcular incision showed some level of alveolar bone loss and gingival recession, which could be avoided using the submarginal incision. Scar formation was the biggest problem with the submarginal flap.

It might be that a papilla preserving incision could have been the incisiontechniqu to shoose in the present case. The evaluation of healing patterns of the papilla base incisions after 3 months revealed mainly completely undetectable or only partially detectable incision lines and generally demonstrated ex- cellent healing. None of the operated sites displayed any measur- able loss of papilla height, or other complications (10). Another study analyzed degrees of papilla shrinkage, when papilla base flaps and sulcular full-thickness flaps were raised (11). The comparison revealed significant loss of papilla height, when the papilla was mobilized during the surgical procedure. In contrary, the papilla base incision resulted in rapid and predictable recession-free healing. To avoid opening of the interproximal space in esthetically relevant areas, the use of the PBI was recom- mended for periradicular surgical procedures. A further study (12) investigated surgical outcomes concerning main vertical loss of height during 12 months.. In cases of full thickness flap elevation, mean papilla loss of height after 1 yr was 0.98 mm. In contrast to full

mobilization of the papilla, the PBI resulted in significantly lower recession depths of only 0.21 mm after one year.

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Case 12

Surgical endodontic treatment of a maxillary left canine with sinus tract

Fig 1. Frontal view

Patient: 52-year-old African male

Chief complaint
Experience of dull pain before and after endodontic treatment performed in February 2010.
Medical record: Non contributory
Dental history: Graduate student endodontically treated tooth 23 in December 2009. The sinus tract persisted even after 3 months long-term inlay with calsiumhydroxide. The tooth was then obturated and the patient was referred to the post-graduate clinic for apical surgery.

Clinical findings 28 th of April 2010
Soft tissue: sinus tract buccaly regio 23, 24
Dental: All teeth sound
Discoloration (dental fluorosis?)

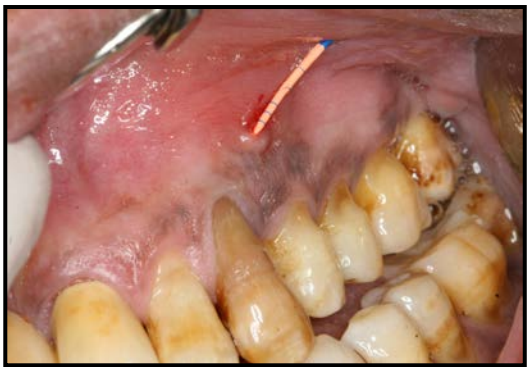

Fig. 2 Buccal view. Guttapercha point in sinus tract

Fig 3. Palatal view

Clinical tests 28 th of April 2010

	22	23	24
EPT (0-80)	14	-	-
Cold	+	-	+
Percussion Vertical/horizontal	-	v and h	-
palpation	-	+	-
PPD	3	6	4
Mobility	-	-	-

Radiographic findings 28th of April 2010
Dental: Tooth 23 has an homogenous root flling, 1 mm from radiological apex
Periodontal: attachment ½-2/3
Apical: tooth 23 apical radiolucency (PAI 4).

Fig. 4 Before endodontic treatment 01.12.2009

Fig 5. Periapical radiograph 28.04. 2010

Diagnosis 28th of April 2010

Pulpal: Root filled tooth (K04.19)
Periapical : Apical periodontitis with sinus tract (K04.62)
Periodontal: Chronic marginal periodontitis (K05.03)

Treatment plan

The root-filling appeared dense and good, and it was decided to do apical surgery 23 with retrograde filling without re-treatment

Treatment 12th of May 2010

1. Anaesthesia 2. Sulcular flap from 11M-25D, with a buccal releasing incision 11D 3. Osteotomy 4. Biopsy and microbiological sampling 5. Apical root resection 6. Retro-preparation 7. Haemostasis 8. Retrograde filling with MTA 9. Sutures 10. Prescription of analgesics and post-op information.

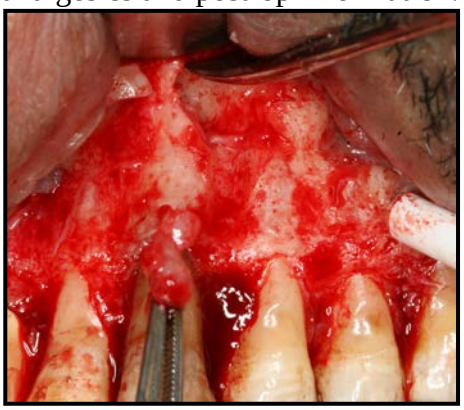

Fig. 6 Fenestration of buccal bone. Enucleation of lesion

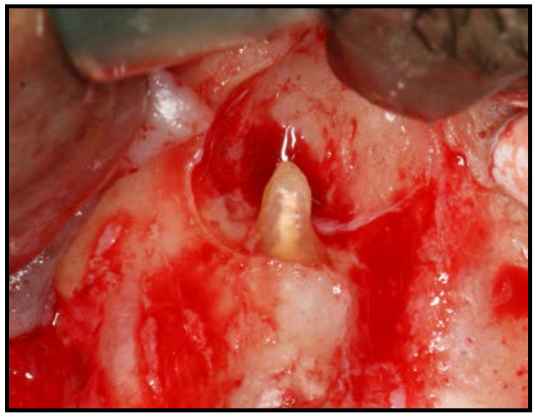

Fig. 7 Apex is visible after ostetomy

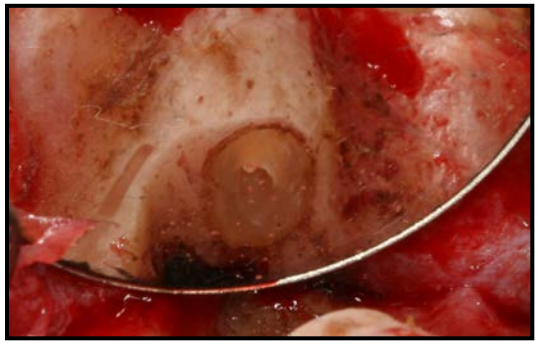

Fig. 8 After rootresection and retropreparation

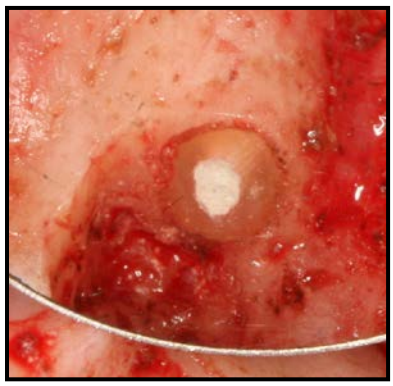

Fig. 9. Rerograde filling MTA

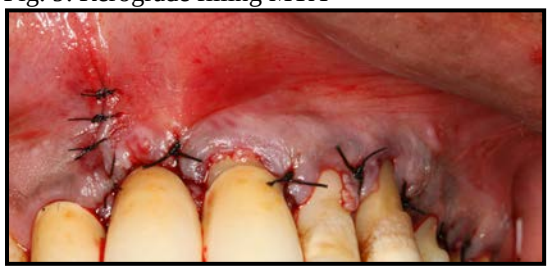

Fig. 10 After suturing

Fig. 11Treatment result 01.05.2011

Treatment 5th of May 2010:
The patient returned after one week for suture removal. He had not experienced any post-op problems. The sinus tract was closed (Fig. 11).

Fig 12. Healing one week after surgery

Fig. 13 Healing one week after surgery

Bacteriological findings with DNA-DNA hybridization): diagnostic service, Institute of Oral Biology
Actinomyces israelii
Treponema denticola
Prevotella nigrescens
Fusobacterium nucleatum
Porphyromonas endodontalis
Eicenella corrodens

Result SEM (scanning electron microscopy). Institute of Oral Biology-Steinar Stølen

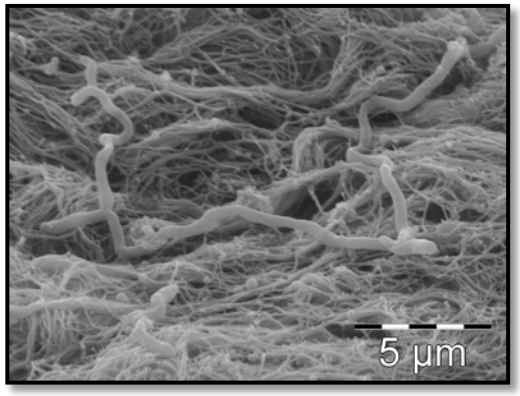

Fig. 14 Branching filamenteous rods

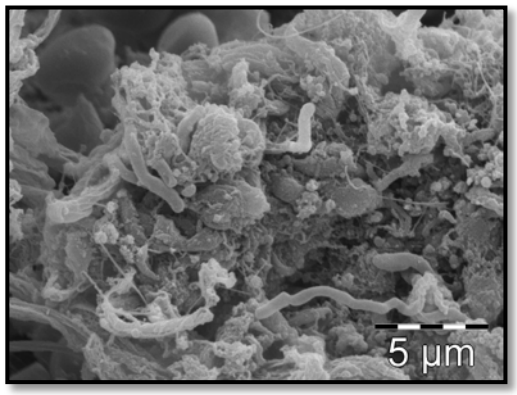

Fig. 15 Rods and extracellular material

Prognosis:
Endodontic: The prognosis seemed to bee good
Dental: the prognosis seemed to be uncertain

Follow-up examination 24th of January 2012 (22 mnd):
The tooth is asymptomatic and the clinical conditions ae satisfactory with no sinus tract (Fig. 16, 17). Radiographic signs of healing with a continuous PDL surrounding the apex (PAI 2) (Fig. 18).

Fig. 16 Buccal view. Healing; 22 months

Fig. 17 Buccal view. Healing; 22 months

Fig. 18 Follow up radiograph 24.01.2012

Discussion

Microbiological sampling detected *Actinomycs israelii* in the periapical tissue in the present case. It is well known that *A. israelii* can be established in periapical tissues (1). In addition, SEM photos (Fig 14,15) showed microorganisms interspersed between an extracellular matrix. These extraradicular findings confirmed that apical surgery was the right treatment choice. A retreatment procedure would most likely not have eradiated the infection.

The patient in the present case had a persistent sinus tract. A sinus tract is defined as a passage of pus from an abscess cavity to and external environment through a tissue membrane such as the oral mucosa or the skin. The reason that a sinus tract develops is not fully understood. Mortensen et al. (2) investigated 1600 teeth with periapical lesions; 136 (9.0%) teeth had sinus tracts. They found that the size of the radiolucency seemed to matter, because teeth with periapical lesions smaller than 5 mm had sinus tracts in 5% of cases, whereas teeth with periapical lesions greater than or equal to 5 mm had sinus tracts in 19%.

Some studies have suggested that the odontogenic sinus tract is lined with epithelium (3, 4). Valderhaug (5) studied experimentally induced sinus tracts in monkeys. He found that most of the sinus tracts were completely or partly lined with epithelium. However, studies by Grossman (6) and Bender and Seltzer (7) state that the sinus tract is not lined with epithelium, but lined with granulation tissue. According to Grossman (6) sinus tracts can heal and closure occurs after root-canal treatment. In this case the sinus tract persisted after treatment of the tooth. It was an indication of treatment failure and a surgical approach had to be done.

For non surgical endodontic treatment, it has been shown that the presence of a sinus tract will not influence the long-term outcome of endodontic treatment (8-12).

However, another study fond that the presence of sinus was found to be a significant prognostic indicator (13).

In surgical endodontic, studies have reported a comparable treatment otcome (14,15).

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Case 13

Surgical endodontic treatment of a maxillary right first premolar

Fig. 1 Frontal view

Patient: 44 year old Caucasian female

Chief complaint
Experience of dull pain after-treatment of tooth 14 done by post graduate student in 2008

Medical record:
High blood pressure
Fibromyalgia
Marevan 3,5 mg, 2 tablets daily
Cozaar (blood pressure)
Sarotex 10 mg (antidepressive)
Zoloft 100 mg (antidepressive)

Dental record: Tooth 14 was endodontically treated by graduate student in 2006 and retreated by post graduate student in 2008

Clinical findings 17th of February 2010
Soft tissue: Normal findings
Dental: Tooth 13 sound, tooth 14 has a dental crown, tooth 16 MOD amalgam filling while tooth 17 has caries and tooth fracture

Fig. 2 Buccal view

Fig. 3 Occlusal view

Clinical tests 17th of February 2010

	14	16	17
EPT (0-80)	-	45	40
Cold	-	+	+
Percussion Vertical/horizontal	-	v and h	-
palpation	-	+	-
PPD	2	2	2
Mobility	-	-	-

Radiographic findings 17th of February 2010
Dental: Tooth 17 has loss of tooth substance, no filling. Tooth 16 MOD amalgam filling, tooth 15 has a dental crown and is root filled
Periodontal: normal
Apical: Tooth 16 has a continuous PDL and tooth 15 has apical radioluceny (PAI 4).


Fig. 4 Periapical radiograph 17.02.2010

Radiographic findings; history
Root filled in 2006 by graduate student and in 2008 by postgraduate student. Radiograph 2009 showed no radiographic healing of the lesion


Fig. 5 September 2006


Fig. 6 September 2008


Fig. 7 September 2009

Diagnosis 17th of February 2010
Pulpal: Root filled tooth (K 04.19)
Periapical : Chronic apical periodontitis (K04.50)
Periodontal: normal

Treatment plan
Apical surgery 14 with retrograde filling

Treatment 14th of April 2010
1. Anaesthesia 2. Sulcular flap from 13M-17D, with a buccal releasing incision 13M 3. Osteotomy 4. Biopsy 5. Apical root resection 6. Retro-preparation 7. Haemostasis 8. Retrograde filling with MTA 9. Sutures 10. Prescription of analgesics and post-op information

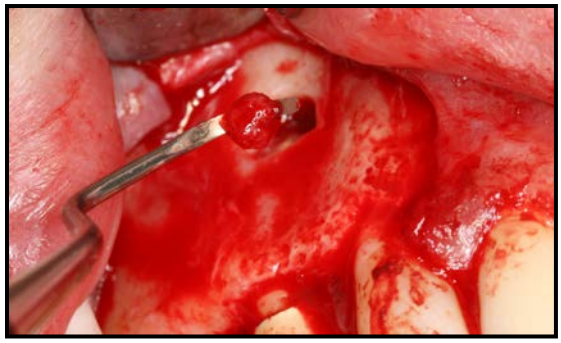
Fig. 8 Enucleation of lesion

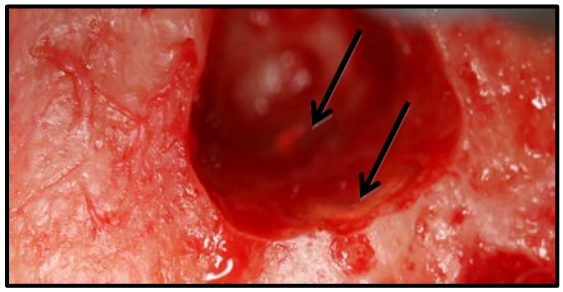
Fig. 9 Buccal and palatinal roots after root resection (arrows)

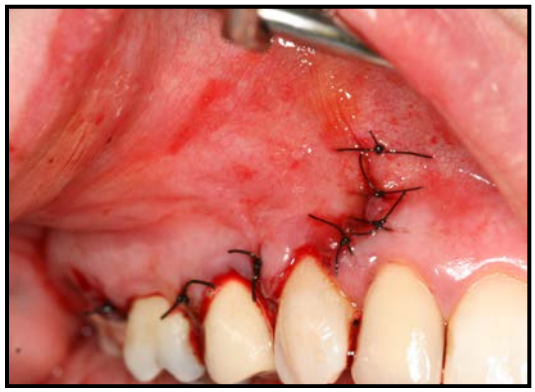
Fig. 10 After suturing

Fig. 11 Before retrograde filling in palatinal canal

Fig. 12 Retrograde fillings in buccal and palatinal canals

Treatment 21th of April 2010

The patient returned after one week for suture removal. She had not experiencedany post-operative problems.

Fig. 13 Before suture removal

Fig 14. Removal of sutures

Prognosis

Endodontic: The prognosis seemed to bee good

Dental: the prognosis seemed to be good

Follow-up examination 30th of January 2012 (21 months)

The radiograph was taken by graduate student. Radiographic healing was evident with a continuous PDL surrounding the apex. Tooth 17 had been extracted since last visit.

Fig. 15 Follow up radiograph 30.01

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Dicussion

In a review by Friedman (1), where selected studies on apical surgery were included, the succees rate was reported to be 37-91% while up to 33% can still be healing several years after surgery. This unsually high and low rates (2, 3) were found to fall considerably outside the range of rate reported in the remaining studies, and the change for complete healing after surgical endodontics is 60-78%(4-7). Lesions >10 mm do show a lower rate of complete healing and a greater incidence of incomplete healing by scar tissue formation (8, 9).

The root anatomy of the maxillary first molar can vary depending on whether one, two, or three roots are present. Prominent root concavities are present on both the mesial and distal surfaces of the root. The mesial root concavity is more prominent and extends onto the cervical third of the crown (10) The palatinal aspect of the

buccal root tip of the two-rooted maxillary first premolars usually has a deep longitudinal depression along its length.(11). Joseph et al.(12) found an incidence of buccal furcation groove in 62% of teeth with bifurcated roots.

The majority of anatomical studies have reported that the most common form of the maxillary first is the two-rooted form in a Caucasian material (13), whereas in studies of the Asian population, single-rooted maxillary premolars are the dominant form (14).

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Case 14

Non surgical retreatment and later surgical retreatment of a mandibular right first molar with carbon post and separated instrument

Fig. 1 Frontal view

Fig. 2 Occlusal view

Patient: 60 year old Caucasian male

Chief complaint:No complains, but he wants restaurations maxilla right side for esthetical reasons. He has never experienced pain from tooth 46

Medical record: He has high blood pressure but he takes no medication.
Dental history: Tooth 46 was previously endodontically treated but the date is unknown.
The patient was referred from the graduate clinic for retreatment of tooth 46 before dental crown therapy

Clinical findings 02nd of September 2009
Soft tissue: Normal findings
Dental: He has a well conserved dentition.
Tooth 46 has a composite filling, caries mesially and carbon post in distal root
Tooth 45 has an unsatisfactory composite filling

Clinical tests 02th of September 2009

	43	44	46
EPT (0-80)	35	21	-
Cold	+	+	-
Percussion Vertical/horizontal	-	-	v
palpation	-	-	-
PPD	3	3	3
Mobility	-	-	-

Radiographic findings 02th of September 2009
Dental: Tooth 44 has a radiopaque filling material. Tooth 45 has an incomplete root filling and post. Tooth 46 has an incomplete root filling, caries mesially, post in the distal canal and separated instrument in the mesial canal
Periodontal: attachment >⅔
Apical: Tooth 46: apical radiolucency (PAI 4)
Tooth 45: continuous PDL space

Fig 3. Periapical radiograph 02.09.2009

Diagnosis 02th of September 2009
Pulpal: Endodontically treated tooth (K04.19)
Periapical : Chronic apical periodontitis (K04.50)
Periodontal: normal

Treatment plan
Retreatment of endodontically treated tooth

Problem list
Removal of post, and removal of separated instrument?

Treatment 16th of September 2009
Carbonpost in distal canal was removed with LN bur and long burs.
Separated instrument in ml canal was removed with modified Gates Glidden to make a shelf, and ultrasound in a counterclockwise direction around the instrument

Mechanical: Bur, Irrisafe®
NiTi handinstruments, BioRace
MB: 17mm/40#
ML: : 17mm/40#
D: : 16mm/50#
Chemical
1% NaOCl, 17% EDTA, 2% chlorhexidine digluconate
Intracanal medicament
$Ca(OH)_2$

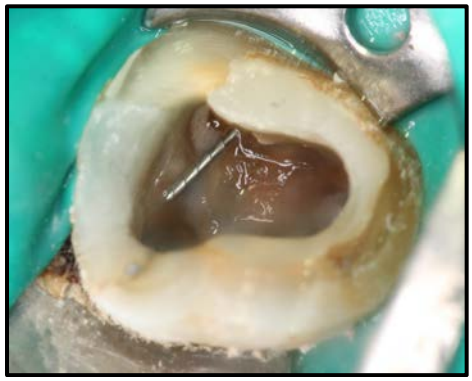

Fig. 4 Separated instrument in cavity after removal

Fig. 5 Separated instrument in ml canal

Fig. 6 Working lenght in ml canal achieved after removal of separated instrument

Progress notes 10th of October 2009
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

Fig. 7 Masterpoint radiograph

Fig. 9 Final radiograph 10.10.2009

Prognosis

Endodontic: the prognosis seemed to be good

Tooth: the prognosis seemed too be uncertain

Follow up examination 12th of October 2010 (12 months)

Radiograph showed no healing of the apical radiolucency (PAI 4). The Patient was asymptomatic and experienced no sen sitivity to percussion or palpation tests. He was under treatment at the student clinic, but had still an IRM filling (although written instructions in the journal about the importance of permanent filling or at least an semipermanent solution like composite). The reason for IRM was that they would not make any permanent restauration unless healing occurred. The patiens graduate student was contacted and asked to make at least a composite restauration, which was done.

Tooth 45 had a new dental crown without endodontic retreatment of unsatisfactory rootfilling and widened periodontal ligament apically. The patient was informed that apical surgery on tooth 46 was advisable. He wanted to wait for some months because of vacation, and then contact the clinic.

Follow up examination September 2011 (23months)

No apical healing. Composite filling. The patient wants surgical endodontic treatment to be done.

Fig. 10 Follow up radiograph 12.10.20

Fig. 11 Follow up radiograph 09.2011

Progress notes 19th of October 2011

1. Anaesthesia 2. Sulcular flap from 43M-regio 47D, with a buccal releasing incision 43M 3. Osteotomy 4. Biopsy 5. Apical root resection 6. Retro-preparation 7. Haemostasis 8. Retrograde filling with MTA 9. Sutures 10. Prescription of analgesics andpost-op information.

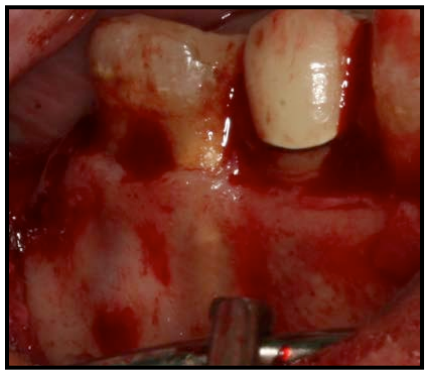

Fig. 12 Reflection of mucoperiosteal fla

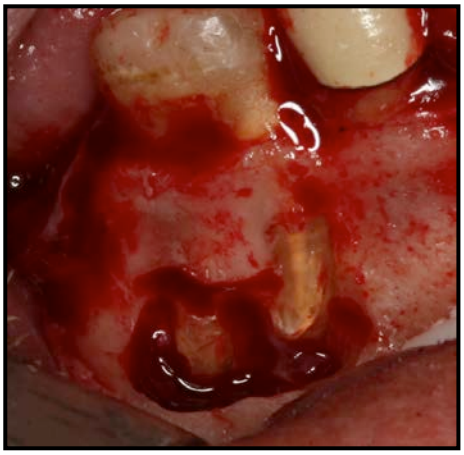

Fig. 13 After ostetomy

Fig. 14 Root resection of mesial root

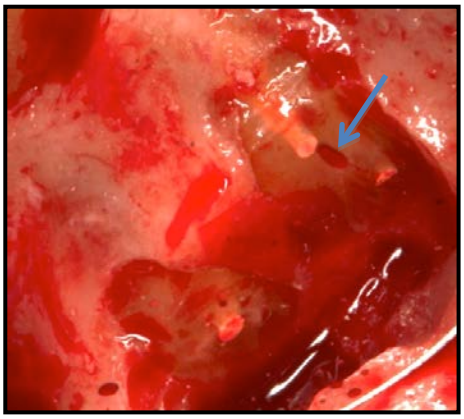

Fig. 15 Close up of root resected roots, an isthmus can be observed in the mesial root (arrow)

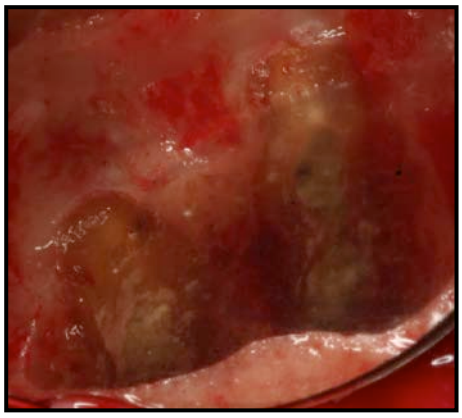

Fig. 16 MTA in distal, ml and mb canals and in the isthmus between ml and mb canals

Fig. 17 After suturing

Fig. 18 Treatment result

Treatment 25th October 2011: Removal of sutures. Soft tissue healing and the patient had no symptoms

Fig. 19 Before removal of sutures; one week after surgery

Microbiological findings (diagnostic service, Institute of Oral Biology)

- *Porphyromonas gingivalis*
- *Prevotella nigrescens*
- *Treponema socranskii spp. Socranskii*
- *Eubacterium saburreum*
- *Streptococcus intermedius*
- *Treponema denticola*
- *Prevotella melaninogenica*
- *Porphyromonas gingivalis*
- MCK plate: *Sphmon. paucimobilis*

Result SEM (scanning electron microscopy). Institute of Oral Biology- Steinar Stølen

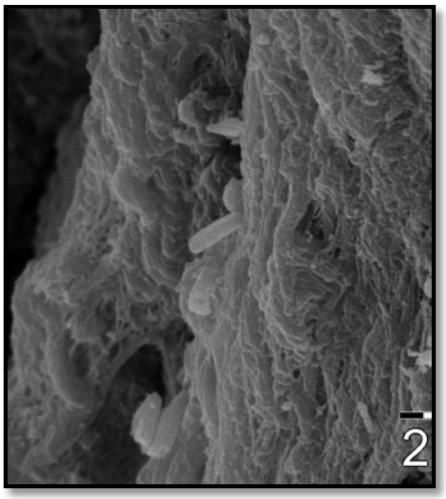

Fig. 20 Microorganisms embedded in an extracelluar material

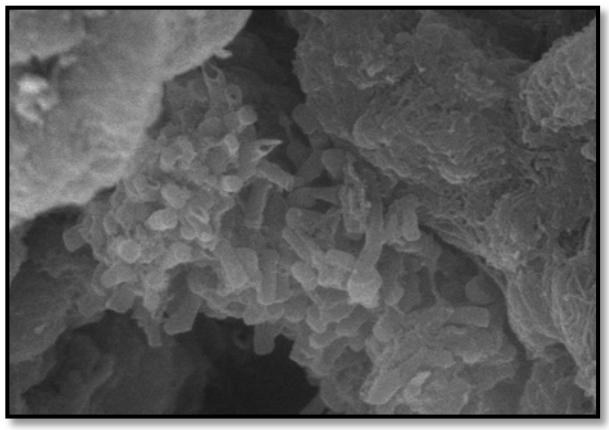

Fig. 21 Microorganisms embedded in extracellular matrix

Follow up examination 7th of March 2012 (6 months after surgery)

No symptoms on palpation and percussion. No pathological pocket depths. Signs of apical healing

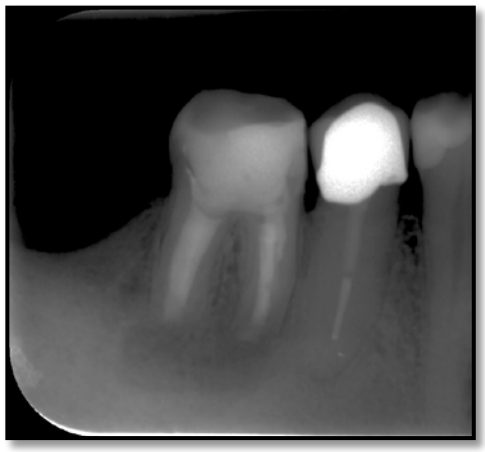

Fig. 22 Follow up radiograph 06.03.2012

Fig. 23 Buccal view; 6 months after surgery

Discussion

The patient in the present case had not got a permanent filling at the one year recall.

Multiple in vitro studies have shown that even the best root canal treatment can allow leakage of bacteria and their byproducts through an apparently well-filled canal system (1). The work of Ray and Trope (2) was a paradigm shift in endodontic treatment philosophy when they found that the coronal restoration had a greater impact on success than the quality of root canal treatment.

Yet, other studies have shown that even in the presence of obvious contamination, periapical disease does not necessarily develop in all patients (3,4,5). Whilst most laboratory studies found microbial leakage through filled root canals to occur within weeks (1), histological observations on unrestored, adequately root filled teeth that were extracted from patients have indicated otherwise (4,6,). In a recent study by Rechenberg et al., (7) they assess the routes of bacterial leakage in a commonly used two-chamber model designed to evaluate root fillings. Under the current conditions, bacterial leakage occurred through the sticky wax seal separating the two chambers of a standard laboratory set-up and through the root canal filled with gutta-percha and an epoxy resin sealer. Bacteria permeated tubular aspects of dentine in root filled teeth, whilst sclerotic or atubular dentine and the interface between sealer and dentine devoid of patent tubules remained bacteria-tight. Bacterial leakage through secondary dentinal tubules (i.e. lateral branches connecting the main tubules) was observed. This suggests that bacteria do not necessarily penetrate filled roots at the immediate interface between root filling material and the canal wall, but also deeper in the (tubular) dentine. They concluded that the current experimental method proved to be unsuitable to compare root fillings.

In a recent systematic review and metaanalysis (8) on the impact of the quality of coronal restaurations versus the quality of root canal filing on periapical healing, they concluded that odds for healing of apical periodontitis increase with both adequate root canal treatment and adequate restorative treatment. Although poorer clinical outcomes may be expected with adequate root filling–inadequate coronal restoration and inadequate root filling–adequate coronal restoration, there is nosignificant difference in the odds of healing between these 2 combinations.

The prevalence of root canal isthmuses in the apical 5 mm of the mesial root of mandibular molars were investigated by means of micro-computed tomography (MCT) (9). They found that isthmuses were present in the vast majority of roots observed. The third millimetre from the apex showed more isthmuses than expected. The results of clinical and surgical endodontic procedures performed in the mesial root of mandibular molars may be affected by this aspect of the root canal anatomy.
In a recent study a three-dimensional analysis of the isthmus area of the mesiobuccal root canal system in mandibular molars were obtained using high-resolution micro–computed tomography (m-CT) scanning (10). They measured the amount of debris and root filling material in the isthmus after instrumenta-tion/irrigation and root filling. A considerable amount of dentin debris is produced and packed into the isthmus area during rotary instrumentation of mesial canals of lower molars despite continuous irrigation during and after instrumentation. The average percentage of volume of filling material in the isthmus areas was significantly lower (57.5%) than in the main root canals (98.5%, $p < 0.001$).

Sphingomonas paucimobilis was in he present case identified with DNA-DNA hybridization in the periapical lesion.

Sphingomonas paucimobilis, formerly known as Pseudomonas paucimobilis, is a, strictly aerobic, Gram-negative bacillus with a single polar flagellum. It was initially reported to be a human pathogen in 1979. The natural habitat of this organism has not been fully defined but it is known to be widely distributed in the natural environment, particularly in water and soil, and has also been isolated from hospital settings, including hospital water systems, distilled water, dialysis fluid, nebulizers, and other respiratory
therapy equipment. S. paucimobilis has been associated with a variety of infections in humans, including bacteremia, pneumonia, catheter- related infections, meningitis, peritonitis, osteomyelitis, septic arthritis, postoperative endophthalmitis, lung em- pyema, splenic abscesses, urinary tract infections, and bil- iary tract infections (12).

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Case 15

Treatment of maxillary central incisors with external inflammatory resorptions

Fig. 1 Frontal view

Patient: 27 year old Caucasian female

Chief complaint: The patient had vague symptoms from the upper front teeth. She was referred from her GP for endodontic treatment 11 and 21

Medical record: non-contributory

Dental history:
She had a bicycle accident and dental trauma in june 2008 and was treated at Ullevål hospital: teeth 11 and 21 had complicated crown fractures and were mobile, no splinting, soft food for 2 weeks, there was no information about the type of injury of the tooth in the journal from Ullevål hospital. Teeth 11 and 21 got composite restaurations at her private dentist and the teeth were after that observed. Root resorptions were discovered in November 2011 and the patient was referred to the post -graduate endodontic clinic for endodontic treatment.

Clinical findings 07 th of January 2009
Soft tissue: normal findings

Dental
Tooth 11 has a composite filling mesially, sign of complicated crown fractur
Tooth 21 has a composite filling distally, and sign of complicated crown fracture.
Tooth 22 has a small enamel fracture while tooth 12 is normal
The patient is caries free with few restaurations

Fig. 2 Palatal view

Fig. 3 Buccal view

Clinical tests 07th of January 2009

	12	11	21	22
EPT (0-80)	65	-	-	15
Cold	+	-	-	+
Percussion	-	v +h	v + h	-
Vertical/horizontal palpation	-	+	+	-
PPD	3	4	4	3
mobility	-	-	-	-

Radiographic findings 07th of January 2009
Dental: Tooth 12 is normal
Tooth 11 has a composite filling mesially and a pulpstone can be observed in the pulpchamber.
Tooth 21 has a composite filling distally,

Tooth 22 is normal
Periodontal:
3 mm bone destruction between 11 and 21
*Apical:*Tooth has an expanded PDL space and diffuse radiolucent area, external root resorption in middle and apical 1/3 of the root , (PAI 3)
Tooth 21 has widened PDL space and diffuse radiolucent area, external root resorption in apical 1/3 (PAI 3).
Teeth 12 and 22 have continuous PDL

Fig. 4 Periapical radiograph 20.11.2008

Diagnosis 07th of January 2009
Teeth 11 and 21:
Pulpal : Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50)
Pathologic resorption: External inflammatory resorption (K03.30)
Periodontal: Within normal limits

Treatment plan
Treatment of infected pulp/apical periodontitis teeth 11 and 21
Follow up

Problem list:
External inflammatory resorption – perforation during instrumentation ?

Treatment 07 th of January 2009
Access cavity preparation and location of one canal, pulpstone in canal removed with ultrasound

Mechanical:Bur, IrriSafe, ultrasound
NiTi haninstrumentation
#70/23mm adjusted to 22 mm
Chemical:1% NaOCl, 16% EDTA, 2% chlorhexidine digluconate
Intracanal medicament: $Ca(OH)_2$

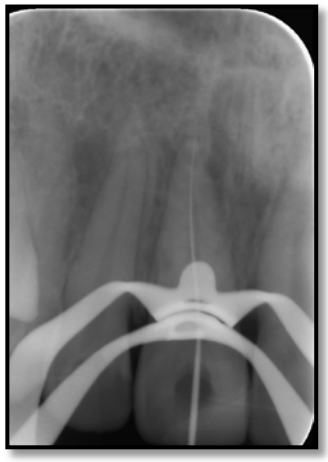

Fig 5. Working length radiograph tooth 11

Treatment 08th of January 2009
*Mechanical:*Bur, IrriSafe
NiTi haninstrumentation #70/22mm
Chemical: 1% NaOCl, 16% EDTA, 2% chlorhexidine digluconate
Intracanal medicament:
$Ca(OH)_2$

Fig. 6 Working length radiograph

Treatment 22th of January 2009
Tooth11: Slightly tender to palpation buccaly

Tooth 21: Tender to palpation buccaly, tenderness to percussion

*Chemical:*Bur, IrriSafe
1% NaOCl, 16% EDTA,
2% chlorhexidine digluconate
Intracanal medicament
$Ca(OH)_2$

Treatment 11th of February 2009
No symptoms, not tender to palpation /percussion.
Kofferdam and instruments changed after final treatment of tooth 21.
MTA plug apically both teeth,
Filled with gutta-percha varm technique, AH plus, sealed with IRM and composite

Fig. 7 Apical plug with MTA tooth 21

Fig. 8 Apical plug with MTA tooth 11

Fig. 9 After treatment11.03.2009
MTA, warm guttapercha, IRM and composite

Follow up examination 07th of September 2010 and 06th of December 2011(18 and 32 months)
Radiograph showed evidence of healing of the periapical radiolucency, but the lamina dura is widened (PAI 2) Cement repair in resorptions is evident (Fig. 10, 11.1 and 11.2) . She has new porcelain laminates on teeth 11 and 21 (Fig. 12). The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests. 12 and 22 were sensitive to ice and EPT

Fig. 10 Follow up radiograph 07.09.2010
(18 months)

Fig. 11.1 Follow up radiograph 06.12.2011 (32 months)

Fig. 11.2 Follow up radiograph 06.12.2011 (32 months)

Fig. 12 Frontal view (32 months aftre treatment)

Discussion

Inflammatory root resorption (external infection related resorption) is a combination of disruption to the outer cementum layer of the root surface and an intracanal infection that triggers osteoclastic activity. This type of resortion can affect all parts of the root and is a rapidly progressing progress that may result in total resorption of the root within a few months. It is usually a sequelae to acute trauma and is especially common after intrusion and replantation of avulsed teeth (1, 2).

When the resorption has penetrated the cementum and exposed dentinal tubules, toxins from bacteria in the root canal and dentinal tubules can diffuse to the PDL. This results in continuation of the osteoclastic process and an associated inflammation in the PDL, leading to resorption of alveolar bone. If bacteria are eliminated from the root canal by endodontic therapy, the resorptive process will be arrested (3, 4). The resorption cavity will then be filled with cementum or bone.

The patient in the present case had most likely a subluxation injury combined with a complicated crown fracture in both of central incisors

The reported risk of pulp necrosis (PN) is generally low in teeth with subluxation injuries. In a recent article by Lauridsen et al. (5) they report that a concomitant crown fracture may increase the risk of PN in subluxated injured teeth, also in teeth with mature root development. They concluded that a concomitant crown fracture and no response to EPT at the initial examination may be used to identify teeth at increased risk of PN following subluxation injury.

The goal in treatment of a severely luxated, intruded, or avulsed tooth is to endodontically treat the tooth before it becomes infected and to halt osteoclastic activity on the outer root surface (6).

Long-term CH placement is recommended for teeth in which external resorption is already radiographically visible (7).

However; root fracture could be a consequence of long-term treatment of CH (8, 9)

MTA, as with with CH, shares a similar highly alkaline pH when freshly mixed (10) and demonstrates some antimicrobial activity (11).

Beyond showing that CH can maintain a high pH over time in the root canal, several

studies have also confirmed hydroxyl ion diffusion through dentin by detecting elevated pH levels on the external root surface (12-14). Similarly, calcium ions from intracanal MTA have been shown to diffuse from the root canal through radicular dentin into simulated external root surface cavities (15).

The reason for an MTA plug in the present study was primarly to get practice.

However, the alkaline pH of MTA found in simulated resorptive lesions combined with its good sealing ability warrant further studies as a treatment option in cases with external inflammatory root resorp tion, particularly in cases in which root fracture could be a consequence of long-term and short-term treatment with CH (16).This is a problem in immature teeth, but the problem apparently does not exist in mature teeth. (4).

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Case16

Endodontic treatment in conjunction with surgical treatment of maxillary right first central, mandibular left second premolar and first molar in patient with multiple inflammatory cervical resorptions

Fig. 1 Frontal view

Fig. 2 Occlusal view

Fig. 3 Lingual view

Fig. 4 Buccal view

Patient: 56 year old Caucasian female

Chief complaint
She complains about lots of problems with her teeth. She is afraid of not to be taken seriously. For example about her muscular problems and allergies she thinks she has to metals.

Medical record: Muscular problems not diagnosed. Low blood pressure.

Dental history: The patient was referred to the post-graduate endodontic clinic from the student clinic because of discovery of a resorption on tooth 36 below a crown made 1 year earlier. It was observed on a routine x ray.
The student came to the postgraduate endodontic clinic for discussion, and CT were ordered from the Department of Maxillofacial Radiology

Clinical findings 15th of February 2011
Soft tissue: normal findings
Dental:

Tooth 35 has a composite filling
Teeth 36 and 37 have dental crowns

Fig. 5 Frontal view

Clinical tests 15th of February 2011

	12	11	21	35	36	37
EPT (0-80)	17	7	12	25	-	44
Cold	+	+	+	+	+	+
Percussion Vertical/horizontal	-	-	-	-	v	-
palpation	-	-	-	-	-	-
PPD	4	7	4	4	5	4
Mobility	-	I	-	-	-	-

Radiographic findings: history (radiographs taken at the graduate clinic)

Fig. 6 Periapical radiograph 23.02.2009

Fig. 7 Bite wing taken after placement of dental crown 11.02.2010

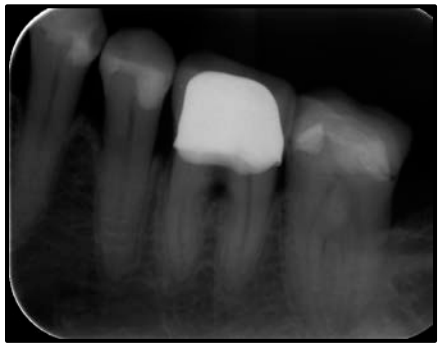

Fig. 8 Periapical radiograph taken 03.02.2011 by student

Fig. 9 Periapical radiograph 15.02.2011

Radiographic findings 15 th of February 2011

After examination of the radiographs that followed the referral 03.02.2011, it was decided to order a CT- scan of both jaws. That would provide valuable information about the extension of the defect s, and also give information about resorptions elsewhere.

In addition to cervical resorptions on teeth 35 and 36, a cervical resorption on tooth 11 was identified on CT scan.

Dental: Tooth 36 has a dental crown, radiolucent area in furcation

Tooth 35 MOD radiopaque filling material and radiolucent area mesially in the cervical area

Tooth 11 has a radiolucent area mesially in the middle 1/3 of the root, located at the bone level.

Periodontal: attachment ½-2/3

Apical : normal PDL teeth 35, 36 and 11

Svarrapport:

Intraorale bilder og OPG fra 03.02.11, CT mandibula og maxilla 0.625 mm fra 08.02.11 til sammenligning:

36: Kronebesatt. Det sees lingualt en cervical resorpsjon på mesiale rot, mot furkasjonsområdet. Resorpsjonen synes å gå helt inn til pulpa.

35: Fyllinger mesialt og distalt. Det sees mesiobuccalt en mindre cervical resorpsjon, noe mer enn 1/2 inn mot pulpa; ingen påvisbar perforasjon.

11: Ingen tidligere behandling. Det sees en buccal, cervical resorpsjon. Resorpsjonen strekker seg i cervical-incisal retning mot pulpa og perferer denne. Det er noe utvidet periodontalspalte buccalt som synes å være direkte relatert til den cervicale resorpsjonen.

R: Multiple cerviale resorpsjoner; inn i pulpa på 36 og 11, konf. beskrivelse.

Margareth Kristensen
spes. kand. kjeve- og ansiktsradiologi

Tore A. Larheim
professor

Tore A. Larheim

Radiologic description (in Norwegian) from the Department of Maxillofacial Radiology

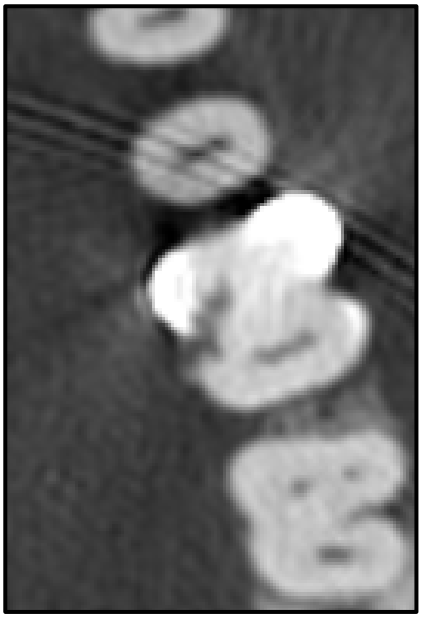

Fig. 10 CT scan axial view of left lower first molar with cervical resorptions. The resorption can be seen in the lingual furcal area at the level of marginal bone. The perforation seemed to perforate into the pulp

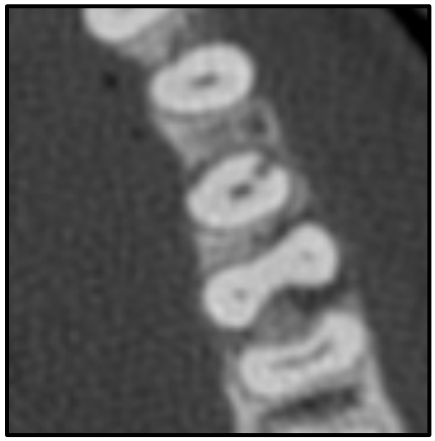

Fig. 11 CT scan axial view of vital left lower second premolar with cervical resorption. The resorption can be seen mesiobuccal to the pulp chamber at the level of marginal bone. The resorption has not perforated into the pulp.

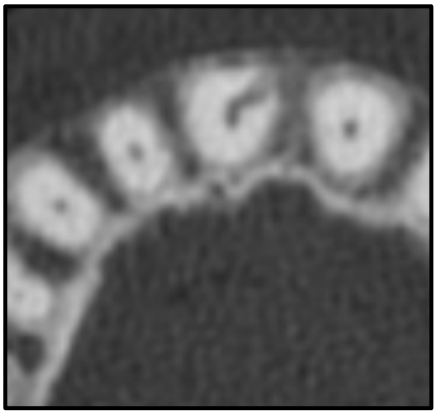

Fig 12 CT scan-axial view-of upper right first central with cervical resorption. The resorption can be seen mesiobuccal to the pulp chamber at the level of marginal bone with eextension to about half the root lenght .The resorption has perforated into the pulp.

Fig. 13 CT scan saggital view of upper right first central with cervical resorption. The resorption has extended below the marginal bone to about half the root length.

Diagnosis 15th of February 2011

Pulpal: Chronic irreversible pulpitis 11, 35 and 36 (K04.03)
Periapical: Normal
Periodontal: Chronic marginal priodontitis
Resorption: Cervical resorption (K03.38)
Multiple cervical resorptions (MIR)

Treatment plan

The patient was told about the poor prognosis for tooth 11 (see discussion), and treatment alternatives were discussed. She wanted under no circumstances to extract the tooth before treatment was tried. She did not want implant treatment because of her fear of metals.

-Pulpectomy teeth 11, 35 and distal root tooth36.
-Flap-elevation to fill the defects in teeth 11, 35 and 36 externally with composite.
-Hemisection tooth 36: the patient wanted to keep tooth 36 despite the uncertain prognosis, and tooth 36 was discussed with a prosthodontist who meant it was possible to preserve the distal root to be incorporated into a bridge later on.

Problem list

The extension of the lesions were large according to CT findings (grade IV on tooth 11 according to Haithersays classification) and it seemed difficult to get an acceptable outcome of the treatment according to the radiographic findings on 36 and 11

Progress notes 15th of February 2011

Tooth 36: Access opening and location of two distal canals.
Short of time. Cavit was placed below resorption communication with PDL in the furcation area (that could be observed in the miscroscope). Patient went on vacation for 3 weeks.

Mechanical: Bur and ultrasound
NiTi rotary intrsumentation BioRace
-DB 19,5/50#
-DL 19/50#
Chemical: 1% NaOCl, 16% EDTA
Intracanal medicament: $Ca(OH)_2$

Fig. 14.Working length radiographs

Treatment 31st of March 2011

No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

Fig. 15 Masterpoint radiograph

Fig 16. Final radiograph

Treatment 05th of April 2011

Tooth 35: Access opening and location of one canal.
Mechanical: NiTi rotary instrumentation
- 20,5/40#
Chemical: 1% NaOCl, 16% EDTA
Filled with gutta-percha and AH plus,
Sealed with IRM

Fig. 17 Masterpoint radiograph

Fig. 18 Removal of gutta-percha in
root canal to below the resorption process

Fig. 19 final radiograph 05.04.2011

Prognosis

Endodontic: The prognosis seemed to be uncertain
Tooth: The prognosis seemed too be uncertain

Treatment 6th of April 2011

Tooth 11: Access opening and location of one canal.
Mechanical: NiTi rotary instrumentation
-one canal 20,5/60#

Chemical
1% NaOCl, 16% EDTA
Filled with gutta-percha and AH plus,
Sealed with IRM

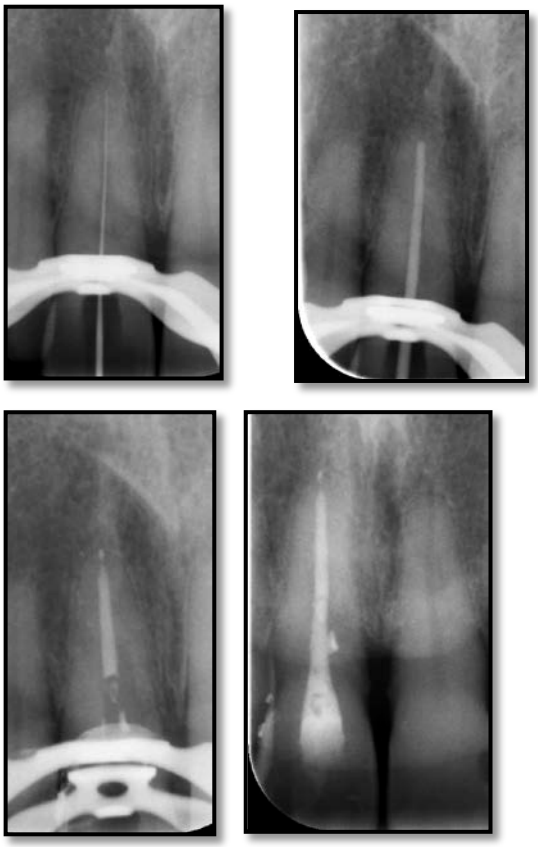

Fig. 20 Upper row shows working length and masterpoint radiographs. Removed guttaparcha in root canal to half the root length,and final radiograph below

Treatment 11th of May 2011
Surgery: Hemisection tooth 36
Marginal incision from 33d-37 d.
Cleaning of resorption lacunae and placement of composite restaurations
Lot of bleeding for unknown reason

Fig. 21 Elevation of flap

Fig. 22 Tooth 36 hemisectioned placement of composite in cavity tooth 35

Fig. 23 After sutures

Fig. 24 Radiograph after surgery

Treatment 18th of May 2011
Surgery: Treatment : Tooth 11
Marginal incision from 12d-21 d.
Removal of bone 11 buccaly 4 mm (Fig. 25). Cleaning of resorption lacunae and placement of composite restoration

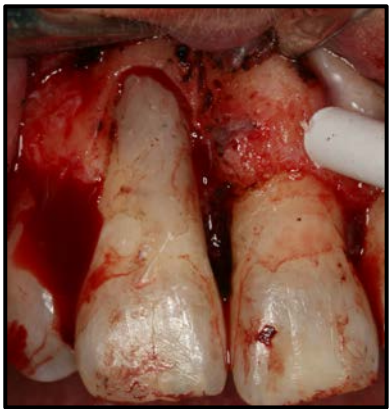

Fig. 25 Buccal bone removed, placement of composite in cavity after cleaning of resorption lacunae

Fig. 26 Radiograph after surgery

Removal of sutures. Healing. Discoloration of all teeth. No symptoms
Removal of sutures maxilla was done by graduate student

Fig. 27 Removal of sutures after one week.

Follow up examination 19th of October 2011 and 1st of March 2012 (5 and 10 months after surgery)

Radiograph showed no recurrence of resorption lesions. The patient was asymptomatic and experienced no sen sitivity to percussion or palpation tests. Dicoloration of all teeth, possibly due to tea drinking. Estethic conditions in front had improved from October 2011 to March 2012. The composite filling had a discoloration that might be a result from blood contamination in the surgical procedure It did not bother the patient.
She had a new prosthetic bridge 44-47

.Fig. 28 Follow up radiograph 19.10.2011 (5 months)

Fig. 28 Follow up radiograph 01.03.2012 (10 months)

Fig. 29 Follow up radiograph 19.10.2011 (5 months)

Fig. 30 Follow up radiograph 01.03.2012 (10 months)

Fig. 31 Healing (5 months)

Fig. 32 Healing (10 months)

Fig. 33 Healing (10 months)

Discussion

Pathologic root resorption can be classified based on the site, nature, and pattern of the process (1). Invasive cervical resorption is a form of external root resorption, characterized by its cervical location and invasive destructive nature (2). It runs an insidious and often aggressively destructive course that is characterized by invasion of the cervical region of the root by fibro vascular tissue derived from the periodontal ligament (3).

This pathological process appears to follow an injury to the cervical attachment apparatus, most importantly to an area of the cervical root surface (precementum), below the epithelial attachment. Progressively, the resorption process will involve cementum, enamel and dentin, to eventually involve the pulp space late in the process (3,4).

The aetiology of invasive cervical resorption is poorly understood, but several potential predisposing factors have been identified. Trauma, orthodontics, orthognathic and other dentoalveolar surgery and periodontal treatment has also been cited (4). A group of 222 patients with a total of 257 teeth that displayed varying degrees of invasive cervical resorptions have been analyzed (3), regarding a sole predisposing or a combination of factors. Of the potential predisposing factors identified, orthodontics was the most common sole factor identified in 47 patients (21,2%) with 62 affected teeth. Trauma was the second most frequent sole factor with 31 patients (14%) with 39 affected teeth (15,1%). Thirty-three (14,9%) of the patients who had a history of intra-coronal bleaching, 10 (4,5%) had bleaching as a sole factor, 17 (7,7%) a history of bleaching and trauma, 2 (0,9%) bleaching and orthodontics and 4 (1,8%) a combination of bleaching, trauma and orthodontics. Surgery, particularly involving the cemento-enamel junction area was identified in 13 patients (5,9%) as

a sole factor. The presence of an intracoronal restoration was the only identifiable factor in 15,3% of the patients and 14,4% of the teeth, while 15% of the patients and 16,4% of teeth showed no identifiable predisposing factors. Intracoronal bleaching in root-filled teeth has been the most widely documented factor and a direct cause-effect relationship is evident (5,6).

The clinical presentation of invasive cervical resorption varies considerably depending on the extent of the resorptive process. The condition is usually painless; a pink discolouration of the crown indicates the resorptive process and probing may result in profuse bleeding. Other teeth give no visual signs and diagnosis is usually the result of a routine radiologic examination. At advanced stages when the lesion involves the pulp, symptoms normally associated with pulpitis will be manifested, and periodontal infection adjacent to the infiltrating resorptive tissue can result in pain and swelling from an intrabony defect and a periodontal pocket formation (2).

A clinical classification has been developed by Heithesary for research purposes and also to provide a clinical guide in the assessment of cases of invasive cervical resorption (3):

Class 1- Denotes a small invasive resorptive lesion near the cervical area with a shallow penetration into dentine

Class 2- Denotes a well-defined resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extensions into the radicular dentine

Class 3- Denotes a deeper invasion of dentine by resorbing tissue, not only involving the coronal dentine but also extending into the coronal third of the root

Class 4- Denotes a large invasive resorptive process that has extended beyond the coronal third of the root

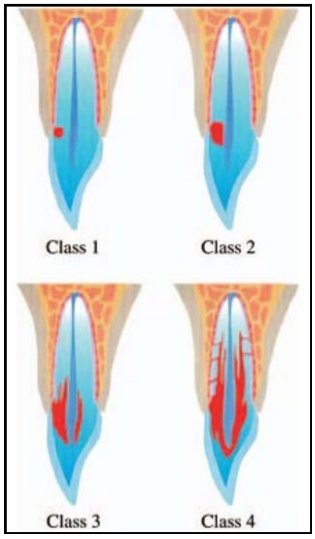

Fig Classes and extent of cervical root resorption according to Heithersay (3).

Regarding which teeth that are most commonly affected, Heithesary (3) showed that the maxillary central incisors came first, followed by maxillary canines, maxillary lateral incisors, mandibular first molars, maxillary first molars, mandibular second molars and mandibular incisors.

Histological, the defect appears similar to any external inflammatory root resorptive defect. There is usually a small opening to the root, with large amounts of fibrovascular, granulomatous tissue inside the defect and multinucleated giant cells resorbing the dentinal structure. In extensive defects, osseous tissue may be seen inside the granulomatous tissue. Resorption channels surrounding the root canal can be seen as they burrow deep into the dentin and interconnect more apically with the periodontal ligament. The predentin/ odontoblastic layer is unaffected and protects the pulp (7,8). If pulpal involvement occurs, the fibro- osseous tissue can be found deposited within the root canal system (2).

If the resorptive process occurs mesially or distally on the root surface it is common that the radiograph shows a small radiolucent opening to the root. It usually can be seen as radiolucency at the attachment level and from there it can spread further coronally or apically with a mottled appearance. Because the pulp is

not involved, its outline can usually be distinguished through the resorptive defect (7). A computerized tomography examination can give us valuable additional information, especially when the lesion is located buccally or lingually. Treatment depends on the various classes of invasive cervical resorption, as the process becomes more complex, differing between non-surgical or surgical treatment will be required. The basic aims though is the same, namely the inactivation of all active resorbing tissue and the reconstitution of the resorptive defect either by the placement of a suitable filling material or by the use of biological systems such as membranes (9). A non-surgical treatment regime were applied to 101 teeth from 94 patients displaying varying degrees of invasive resorption and followed up for a minimum of 3 years (3b). It involved the topical application of a 90% aqueous solution of trichloracetic acid to the resorptive tissue, curettage, endodontic treatment where necessary, and restorations with glass-ionomer cement. The result was complete success in Class 1 and Class 2 resorptions. Of the 63 teeth classified with Class 3, 61 (96,8%) showed resorption control. When all factors (resorption control, angular bone loss, periapical changes and extraction) were included in the assessment, the overall success rate of Class 3 treatment was 77,8%. In Class 4 resorptions 16 teeth were treated and the results showed a survival rate of 50% and a success rate as judged above 12,5%. This represents an unsatisfactory outcome for this treatment regimen when applied to Class 4 resorptions, and alternative prosthodontic replacement is generally suggested (9).

Using hemisection to preserve multirooted teeth that have furcation involvement has increased over the last years. A reported failure rate of 13.1% has been found over 5-10 years and comparing the results with implants the failure rates of the two treatments were not so different (10). Fugazzotto (11) reported success rates ranging from 95.2% to 100% for various root resected molars in function. Only resection of the distal root demonstrated a lower success rate of 75%. Implants used in molar position had a success rate ranging from 97% to 98.6%.

In a study by Blomhof et al., (12) they compared tooth mortality of root-resected molars with that of root-filled, single-rooted teeth. Survival rates were 68% for root-resected molars and 77% for root-filled single-rooted teeth over a 10-year period. This difference was not statistically significant.
They concluded that the prognosis of root-resection is not poorer than the prognosis of single-rooted teeth with an equal susceptibility to periodontitis, if endodontic conditions and maintenance care are optimal.

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Case 17

Non-surgical retreatment of the maxillary right lateral incisor with internal resorption

Fig 1: Frontal view

Patient: 56 year old Caucasian female

Chief complaint
Tooth 12 has the last month been tender when chewing and biting.

Medical record: Non contributory

Dental history: Tooth 12 was endodontically treated about 20 years ago. No history of trauma that she can recall.
The patient was referred from the graduate clinic for evaluation and treatment of the right maxillary lateral

Clinical findings 17th of June 2010
Soft tissue: Normal findings
Dental:
Tooth 13 has a distal amalgam filling.
Tooth 12 has a distal and occlusal composite filling and tooth
11 composite fillings mesially.

Fig 2: Buccal view

Fig 3: Palatal view

Clinical tests 17th of June 2010

	13	12	11
EPT (0-80)	14	-	25
Cold	+	-	+
Percussion Vertical/horizontal	-	v	-
palpation	-	-	-
PPD	3	3	3
Mobility	-	-	-

Radiographic findings 17th of June 2010
Dental: Tooth 12 has a radiolucent restorative material
Pulpal: Resorption internal in apical 1/3

Intraradicular radiopaque material
Apical: Tooth 12 has a widened PDL (PAI 2)
Periodontal: Attachment ½- 2/3

Fig 4. Periapical radiograph

Diagnosis 17th of June 2010
Pulpal: Necrotic pulp K04.11
Endodontically treated K04.19:
Internal resorption K03.32:
Periapical: normal
Periodontal: Chronic marginal periodontitis K05.03:

Treatment plan
Re-treatment 12 and filling of resorptive process with MTA

Problem list
Extension of resorptive process is unknown; communication externally?

Treatment 16th of June 2010
Access opening. Difficulties in finding the canal through the internal resorption (Fig. 5). Pus and bleeding in the canal; resorptive process penetrating to the periodontal ligament or it could be an external resorption.
Mechanical: Bur, Irrisafe
NiTi handinstruments #60/17- corr. 16mm
Chemical: 1% NaOCl*;* 17% EDTA
2% chlorhexidine digluconate
Intracanal medicament: Ca(OH)2

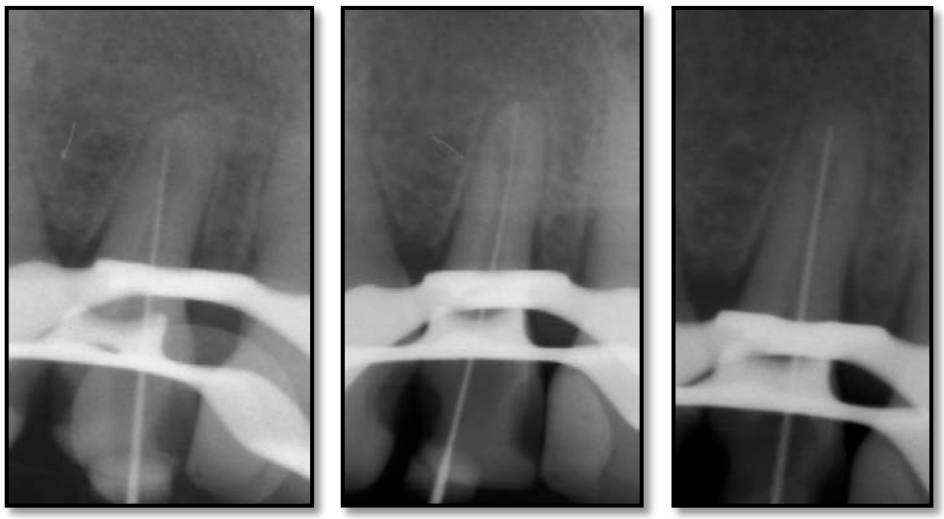

Fig 5. Working lengths radiographs

Treatment 25th of August 2010
No symptoms, not tender to percussion
Filled with MTA Angulus, warm gutta-percha and AH plus, sealed with composite restauration

Fig. 6 MTA in resortion and warm gutta-percha in canal

Fig. 7 Final radiograph

Prognosis

Endodontic: the prognosis seemed to be uncertain

Tooth: the prognosis seemed too be uncertain

Follow up examination 08th of June 2011 and 13th of March 2012 (10 and 19 months)

Radiograph showed no recidive of resorptionprocess

The patient was asymptomatic and experienced no sensitivity to percussion or palpation tests.

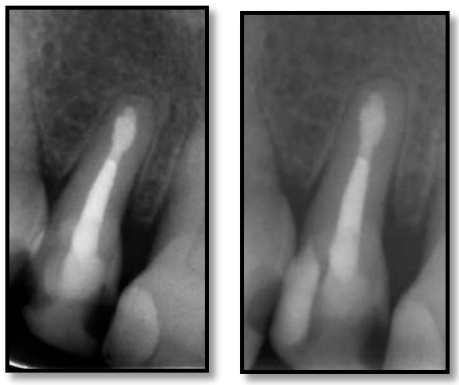

Fig 8.1 and 8.2 Follow up radiographs 08.06.2011 and 13.03.2012 (10 and 19 months)

Discussion

Internal inflammatory root resorption destroys dental hard tissue by odontoclast activity (1, 2). Internal resorption starts inside the root canal and requires at least partially vital pulp tissue. If the resorption is not detected and remains untreated, it can potentially grow larger and eventually perforate the root from inside. When internal resorption is detected early enough, the treatment is usually successful, and the long-term prognosis of the affected tooth is good. Only when a significant amount of tooth structure has been destroyed and/or the resorption is close to the marginal bone (coronal third of the root) will weakening of the tooth have a negative impact on the prognosis of the treatment.

Internal root resorption is regarded as rare, but the frequency of internal resorp- tion is not well known. Many of the published articles on internal resorption are case reports (3, 4), with an emphasis on the treatment of the resorption. In some studies, the occurrence of internal resorption has been estimated to be between 0.01% and 1% (1). Thoma (5) reported internal root resorption in 1 out of 1,000 teeth. However, Cabrini et al (6) detected internal resorption by histological examination in 8 out of 28 teeth (28%) 49 to 320 days after calcium hydroxide pulpotomy. In another study of 33 au- totransplanted maxillary canines, 17 (55%) developed internal resorption during the

follow-up time of 6 years (7). The results of these studies point to inflammation as an important factor in the etiology of internal resorption. Inflammation was also reported as the primary cause of internal resorption in the classic animal studies by Wedenberg and Lindskog (8–10), who exposed pulps of monkey teeth to Freund's adjuvant (inflammation) or to Freund's adjuvant and oral bacteria (inflammation + infection). A histological and scanning electron microscopic study on resorption in teeth with apical granulomas and cysts showed that apical resorption was a common finding and that intracanal resorption was also often found (11). The resorption zones of the external and intracanal sides of the foramina seemed to be interconnected. The study concluded that intracanal resorption may be quite common; however, exact numbers were not given (11).

In a recent study by Haapasalo et al.(12), they examined the occurrence of internal resorption in teeth with healthy and diseased pulps using a scanning electron microscope with high magnification.

Four of the 8 teeth with pulpitis (50%) and 10 of the 13 teeth with necrotic pulps (77%) had internal resorption ($P < .01$). The average number of resorptive lesions in the affected necrotic teeth was 2.4, whereas in teeth with pulpitis and internal resorption, the average number of lesions was 1.25. The amount of resorption was always <100-mm deep; the length of the lesions varied from 200 mm to >1 mm. Most lesions were detected in the middle third of the root, followed by the apical third. Only 1 internal resorptive lesion was detected in the coronal third of the root canal. They concluded that internal resorption was a frequent finding in teeth with pulp inflammation or necrosis.

In a systematic review and meta-analysis, they evaluated clinical outcome differences of root canal obturation by warm gutta-percha (GP) or cold lateral condensation (CLC) There were 10 clinical studies evaluated. Postoperative pain, long-term outcomes, obturation quality, and overextension were the characteristics investigated. The results suggest that the two obturation techniques are not significantly different except in overextention. Overextension was more likely to occur in the warm GP obturation group in comparison with the CLC group. Postoperative pain prevalence, long-term outcomes, and obturation quality were similar between the two groups

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Case 18

Patient with persistent pain maxilla left side


Fig. 1 Frontal view


Fig. 2 Occlusal view

Patient: 77 year old Caucasian male

Chief complaint
He had experienced pain from tooth 25 of various intensity since 2007

Medical record:
By-pass surgery 1991
Albyl E; 160 mg

Dental history:
Referred from private practitioner because of persisting pain 25 after endodontic treatment April 2009.
He has had symptoms from tooth 25 since 2007. In October 2009 he got Apocillin 660 mg for14 days because of maxillary sinusitis

Clinical examination 11th of February 2010
Extraorally:
He had skin rubor, nasal secretion and lacrimation on the left side
Intraorally:
No pathologica findings on mucosa
Dental:
Tooth 25 mod amalgam/composite filling,
26 missing (extracetd early childhood).


Fig. 3 Occlusal view


Fig. 4 Buccal view

Clinical tests 11th of February 2010

	24	25	27
EPT (0-80)	26	-	15
Cold	+	-	+
Percussion Vertical/horizontal	-	both	-
palpation	-	+	-
PPD	3	3	3
Mobility	-	-	-

Radiographic findings 11th of February 2010:

Dental: Tooth 24 has a dental crown, tooth 25 is root filled with radiopaque filling material MOD. Tooth 27 has a MO radiopaque filling material. Tooth 26 is missing.

Periodontal: Attachment ⅔

Apical: 24, 25, 26 continuous PDL space

Fig. 5 Periapical radiograph

Diagnosis 11th of February 2010

Pulpal: Endodontically treated (K04.19)
Periapical : normal
Periodontal: within normal limits

Treatment plan

Re-treatment

Problem list

Painmanagement was thought to be a problem

Treatment 11th of February 2010

The patient could tell about dull pain when he wakes up in the morning. He does not take any painkillers. The pain is of variable intensity.
Tooth 25 was tender to percussion in each blindtest, so a restinfection was suspected, even though the pain was not easy to interpret. The patient was informed that a retreatment might not resolve his painproblem. Access opening revealed unsealed guttapercha in the pulpchamber. The amalgam filling was removed and staining of the tooh revealed a crackline extending from the mesial to the distal aspect (Fig.8). During the second visit, the cusp became mobile and was removed (Fig.9).

Mechanical:
Bur, Pre RaCe, Irrisafe®
NiTi handinstruments, BioRace
B: #40/19 mm,
P: #40/17,5
Chemical: 1% NaOCl 16% EDTA
Chlorhexidine- di-gluconate 2%
Intracanal medicament: $Ca(OH)_2$

Fig. 6 Working length radiograph

Treatment 10th of March 2010

Still symptoms, still tender to percussion, but less symptoms since last vistit, according to the patient.
He had still nasal secretion and lacrimation.
Neuropatic pain and/or chronic maxillary sinusitis were suspected.
The patients doctor was contaced. CT was taken at the Department of Maxillofacial Radiology, Institute of Clinical Dentistry, UiO. MR caput was done at Ullevål hospital.
The tooth was filled with gutta-percha and AH plus, sealed with IRM

Fig. 7.1 and 7.2 Masterpoint and final radiographs

Fig. 8 Cracks stained with methylene blue (arrows)

Fig. 9 Removed fractured tooth substance at craze lines

Prognosis

Endodontic: the prognosis seemed to be good

Tooth: the prognosis seemed too be uncertain.

The patient did not want to come for follow-up, but he could tell on the phone that he had sinus surgery, which gave no painrelief. He had still symptoms from the tooth when he woke up every morning.

Radiologisvar ved spesialist Bjørn Bamse Mork-Knutsen, avdeling for kjeve - og ansiktsradiologi

”CT viser homogen sløring i hele venstre maxillarsinus, og også betydelig sløring i hele høyre maxillarsinus. Det er noe slimhinnefortykkelse også i etmoidalceller høyre side, men frontalsinus og sphenoidalsinus er klare.”

”I høyre maxillarsinus sees fortykket bakre benbegrensning, som uttrykk for (event. tidligere) kronisk sinusitt”

”Med tynne snitt sees helt normale periapikale bentegninger 1 og 2 kvadrant”

Fig. 10 Coronal CT scan shows homogenous opacification of right and left maxillary sinuses

Discussion

The nerve fiber density within human teeth is quite impressive. A number of ultrastructural studies have evaluated the type (as based on fiber diameter and

presence or lack of myelin) and number of axons that innervate anterior and posterior teeth. Comprehensive studies of nerve fibers within posterior teeth are limited to single-rooted premolars Nair (1) con- cluded that human premolar teeth contain 2300 axons at the apex; 87% of these are unmyelinated, and the remainder is myelinated. The vast ma- jority of the myelinated fibers are thinly myelinated and fall in the A-delta class, and the remaining 7% represent the more thickly myelinated A-beta nerve fibers. Even though the ‘‘average’’ premolar tooth has a significant nerve density, this can vary depending on the developmental stage and type of tooth [2] and can vary widely among individual samples. Other axons that enter the tooth pulp originate from post- ganglionic sympathetic neurons located in the superior cervical ganglion whose role involves vasoconstriction [3],

Some of the peptides identified in tooth pulp include the tachykinin SP neuropeptide Y (NPY) CGRP. Peptides as a group are important in nociception because the expression of some change considerably with injury or after inflammatory insults. Sprouting of CGRP fibers is seen in the rat tooth pulp after inflam- matory lesions [4], and similar results involving increased fibers with CGRP, NPY and SP have been described in human teeth with carious lesions [,5]. These same neuropeptides is especially implicated in inflammatory processes because sensory nerve stim- ulation can lead to their local release by way of an axon-reflex [6], or they may be released from nerves that innervate blood vessels, leading to vasodi- lation and protein extravasation, which results in a neurogenic inflammation [7]. Neurogenic inflammation and local tissue injury are associated with the release or activation of many different molecules that are involved in the sensitization of peripheral nociceptors, in- cluding their ability to further enhance the release of CGRP and SP. These substances include cytokines, prostaglandins, histamine, bradykinin, serotonin, lipids, nitric oxide, and hydrogen ions. The local release of CGRP and SP from peripheral terminals may bind to CGRP and SP recep- tors on immune cells, and this binding may be involved in the regulation of the immune response in a paracrine fashion. For example, SP released from nerve terminals can bind to mast cells, leading to degranulation and the release of histamine (8).

The International Association for the Study of Pain has introduced the concept of neuropathic pain, defined as “a pain initiated or caused by a primary lesion or dysfunction in the nervous system” [9]. Recently, experts have defined neuropathic pain as a direct consequence of a lesion or disease regarding the somatosensory system [10].

Proposed mechanisms for trigeminal neuropathy include peripheral or central sensitization, beta fibre reorganization and sympathetically maintained pain due to alpha receptor sprouting. (11)

Many patients with neuropathic pain exhibit persistent or paroxysmal pain that is independent of a stimulus. This stimulus-independent pain can be shooting, lancinating, or burning and may depend on activity in the sympathetic nervous system. Spontaneous activity in nociceptor C fibres is thought to be responsible for persistent burning pain and the sensitisation of dorsal horn neurons. Similarly, spontaneous activity in large myelinated A fibres (which normally signal innocuous sensations) is related to stimulus-independent paraesthesias and, after central sensitisation, to dysaesthesias and pain. Stimulus-evoked pain is a common component of peripheral nerve injury or damage and has two key features: hyperalgesia and allodynia. Hyperalgesia is an increased pain response to a suprathreshold noxious stimulus and is the result of abnormal processing of nociceptor input. Allodynia is the sensation of pain elicited by a non-noxious stimulus

and can be produced in two ways: by the action of low threshold myelinated A beta fibres on an altered central nervous system; and by a reduction in the threshold of nociceptor terminals in the periphery. Hyperalgesia is the consequence of an increased central response to A beta fibre input. Continual input to the dorsal horn as a result of spontaneous firing in C fibre sensory neurons causes sensitisation of dorsal horn neurons, which increases their excitability such that they respond to normal inputs in an exaggerated and extended way. Thus, stimuli that would normally be innocuous are now painful. (11) .

The patient in the present case had chronic sinusitis, that was discovered on CT scans after endodontic retreatment.

Maxillary sinusitis causes a constant boring pain with zygomatic and dental tenderness from the inflammation

of the maxillary sinus The character of the pain of maxillary sinusitis is dull, aching, boring and tender, of mild to moderate severity. The pain is triggered by bending forward, touching the area or biting on the upper teeth (12).

Since the patient had persisting pain also after sinus surgery, it was probably not chronic sinusitis. It might have been though, that having chronic sinusitis for some time could give rise to neuropathic pain due to nerve damage as discussed above.

The patient in the present case had for a long time an incompletely fractured tooth.

In a study by Brynjulfsen *et al.*,(13), thirty-two patients with poorly localized orofacial pain, were finally diagnosed with 46 incompletely fractured teeth. They found that the longer the duration of pain before the diagnosis of an incom- pletely fractured tooth was established, the more diffuse was the distribution of pain. Endodontic or restorative treatment relieved the symptoms in 90% of the patients, whilst persisting symptoms in 10% were considered part of an orofacial pain complex of obscure aetiology.

As discussed above, the patient in the present case had most likely several diagnosis; cracked tooth, infected tooth and chronic maxillary sinusitis that could explain his pain condition.

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Case 19

Endodontic treatment of a mandibular right first molar with C-shape anatomy

Fig. 1 Frontal view

Patient: 60 year old Caucasian male

Chief complaint:
Episodes of pain. Tender when chewing and biting.
Medical record: Non-contributory
Dental histoy: The patient was referred to the post-graduate endodontic clinic by his general dental practitioner.
The patient had the diagnosis acute apical periodontitis when he visited his dental practitioner in November 2008. The dentist had made an access cavity but he found the pulp chamber to have an "unusual" appearance and was not successful in finding the root canal orifices. Cavity was filled with eugenol pellet and IRM

Clinical findings 20th of January 2009
Soft tissue: normal findings
Dental:
Teeth 45 and 46 have dental crowns and tooth 46 has an occlusal temporary filling
Teeth 57 and 48 have amalgam fillings

Fig. 2 Occlusal view

Clinical tests 20.01.2009

	48	47	46
EPT (0-80)	15	20	-
Cold	+	+	
Percussion Vertical/horizontal	-	-	v
palpation	-	-	-
PPD	3	3	3
Mobility	-	-	-

Radiographic findings 20th of January 2009
Dental: Distal sweep of fused roots tooth 46. Fused root on tooth 47
Periodontal: Within normal limits
Apical : Diffuse radiolucencies mesial and distal roots tooth 46 (PAI 4)

Fig 3. Periapical radiograph

Diagnosis 20th of January 2009
Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50). PAI 3
Periodontal: Within normal limits
Treatment plan
Treatment of infected pulp
Problem list:
Anatomical considerations 46

Treatment 29th of January 2009
Access opening and location of first three, then the forth canal located with ultrasound
Mechanical: Bur and ultrasound
NiTi handinstrumentation, BioRace
-MB 20/50#, -ML 22/40#
-DB 20/50#, -DL 20/40#
Chemical: 1% NaOCl, 16% EDTA
Intracanal medicament: $Ca(OH)_2$

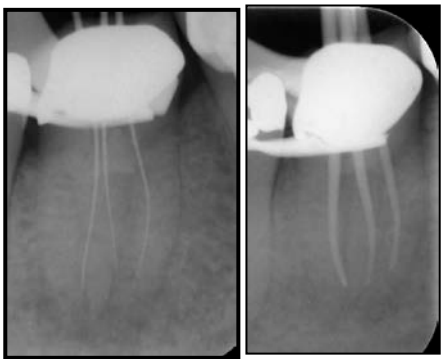

Fig 4. Working length radiograph 20.01.2009
Fig 5. Masterpoint radiograph 05.02.2009

Treatment 5th of February 2009
No symptoms, not tender to percussion
Filled with gutta-percha and AH plus, sealed with IRM

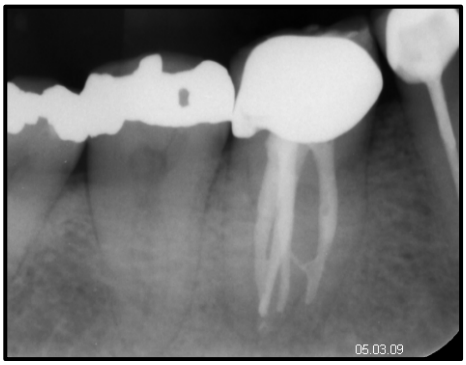

Fig 6. Final radiograph

Prognosis
Endodontic: the prognosis seemed to be good
Tooth: the prognosis seemed too be good

Follow up examination 14th of April 2010 and 20th of January 2012 (11 and 35 months)
Radiograph showed evidence of healing of the periapical radiolucency (PAI 1).The patient
was asymptomatic and experienced no sen sitivity to percussion or palpation tests.

Fig. 7 Follow up radiograph 14.04.2010

Fig. 8 Follow up radiograph 20.01.2012

Discussion
The first who reported c-shaped root canal configurations were Cooke and Cox in 1979 (1).
Manning (2) speculated that the failure of the Hertwig's epithelial root sheath to fuse on the lingual or buccal root surface was the main cause of a C-shaped root, which always contains a C-shaped canal. The main feature of this variation is the presence of a fin or web connecting the

individual root canals to form a letter ‘C’ shape at the root canal orifice. The C-shaped slit may end in two or three canals apically in the root, or the C-shaped may be continuous throughout the length of the root

This variation may occur in mandibular first molars, maxillary molars , mandibular first premolars , and even in maxillary lateral incisors, but it is most commonly found in mandibular second molars (3).

The C-shaped canal is most frequently found in the mandibular second molar. It is a significant ethnic variation that has a high prevalence in Asians (4,5). Melton et al. (6) first proposed the classification of C-shaped canals based on their cross-sectional shape. However, there was no clear description of how to differentiate between Category II and Category III. Fan et al. (7) analysed the morphology of C-shaped canals in mandibular second molars using microcomputed tomography (micro-CT) and modified Melton’s method (Fig. 1).

Category I (C1): the shape was an uninterrupted “C” with no separation or division

Category II (C2): the canal shape resembled a semicolumn result- ing from a discontinuation of the “C”

Category III (C3): two or three separate canals

Category IV (C4): only one round or oval canal in that cross- section (Fig. 1E).

Category V (C5): no canal lumen could be observed (which was usually seen near the apex only)

Fig. Classification of C-shaped canal configuration. Fan et al 2004. (a modification of Melton’s method from 1991):

Irregular areas in a C-shaped root canal system may house soft tissue remnants or infected debris, so thorough cleaning, shaping and a intracanal dressing with Ca(OH)2 is recommended. These teeth pose a considerable technical challenge but by the use of microscopy, ultrasonic instrumentation, and plasticized obturation techniques have made treatment successful1.

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Case nr 20

Endodontic treatment of the immature maxillary left lateral incisor with dens invaginatus

Patient: 10 year old Caucasian male

Chief complaint
Pain region 22.
Medical record: Non contributory
Dental history
Referred to the post-graduate endodontic clinic from the post-graduate clinic pedodontics for treatment of tooth 22 with dens invaginatus.
The patient had been treated at the dental public clinic. Because of an abscess region 22, the dens invaginated tooth was discovered. An incision of the abscess was made and prescription of Apocillin 660 mg. The endodontic treatment was started, but because of pulpanathomy difficulties, the patient was referred to he post-graduate clinic in pedodontics

The patient was teferred to the department of Maxillofacial Radiology for cone beam CT scan before endodontic treatment.

Clinical findings 01st of February and 13 th of March 2012
Soft tissue: When he came to the post-graduate clinic in pedodontics, he had still an abscess in region 22-63 and the incision wound was still bleeding.
At the 13th of March, at the post graduate endodontic clinic, there is a hard palpable tumor buccaly in the same region with some fluctuation, no sinus tract.
Dental: Tooth 22 has a flat and wide crown with a palatal temporary filling. Mixed dentition


Fig. 1 Palatal view 01.02. 2012


Fig 2: buccal view 01.02.2012


Fig. 3 Buccal view 14.03.2012

Radiographic findings 13 th of March 2012
Dental: Tooth 22 has a radiopaque occlusal filling. Dens invagination, a

variant of Type II- Oeahlers classification (see Fig. 1 in discussion). Axial and saggital CBCT scans reveal that thers is a separate invaginated apex that ends apically. The pulp prober surrounds the invagination, but the ct scans (Fig. 6, 7 and 8) reveal that there are some dential bridges, especially at the palatal and mesial aspect.

Apical: Diffuse apical radiolucency (PAI 5), 10 mm in diameter

Svarrapport:

OPG og intraorale tannbilder fra 01.02.12, CBCT 40x40 mm fra 06.03.12:

Sentralt i rotkanalen på tann 22 sees et dentin-struktur med emalje sentralt og pulparom i hele tannens lengde. I deler av rotkanalen er det dentinbroer mellom det sentrale og det perifere dentinet. Apex på tannen er åpen og virker noe resorbert. Det er en stor apikal oppklaring med diffus avgrensning.

R: Dens in dente tann 22 med apikal periodontitt og rotresorpsjon.

Bildene kan med fordel demonstreres her på avdelingen.

Linda Arvidsson
postdoc
spes. kjeve- og ansiktsradiologi

Radiological description (in Norwegian) from the Department of Maxillofacial Radiology

Fig 4. Periapical radiograph

Fig. 5 CT scan-Saggital view; invagination and apical radiolucency can be seen

Fig. 6 CT scan- sagittal view shows invagination and apical radioluvency

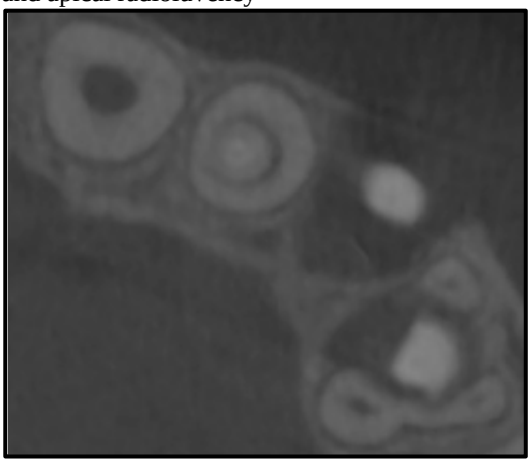


Fig 7: CT scan axial view shows an almost circular pulp proper in tooth 22 wih a dentinal bridge mesially.

Diagnosis 13 th of March 2012

Pulpal: Infected necrosis (K04.11)
Periapical: Chronic apical periodontitis (K04.50) .
Other diagnosis: Dens invaginatus (K08.8)

Treatment plan

Treatment of infected invagination. In addition, remove the whole invagination with bur and ultrasound since the pulp proper has been infected; most likely because of perforation when previous access opening.

Problem list

Cooperation problems; due to the time - consuming work to be done and he patients age.

Treatment 13 th of March 2012

Access opening revealed previous pulpal access at four places. Removal of tooth substance with LN and round burs. Instrumentation of invagination with handinstruments and BioRace #90/18 mm
*Chemical:*1% NaOCl, 16% EDTA
Intracanal medicament:$Ca(OH)_2$
Temporary filling: IRM

Fig 8. Working length radiograph

Treatment 22 nd of March 2012

No pain since last visit. There is still a hard, palpable tumor buccaly, but with no fluctuation.
Bur was used but the patient did not tolerate the use of ultrasound because of the sound and discomfort. It was therefore not possible to remove all the invaginated tooth substance, as seen in the radiograph (Fig. 9).
*Chemical:*1% NaOCl, 16% EDTA
Intracanal medicament:$Ca(OH)_2$
Temporary filling: IRMIt was decided to give the patient premedication next tme (Flunizipam)

Fig.9 The invaginated tooth substance is almost removed

Treatment 17 th of April 2012

The patient got Flunizipam 45 minutes before treatment. The sedative effect was satisfactory, but the patient still did not tolerate the use of ultrasound. The rest of the ”invaginated toothsubstance” and the pulp was anyway removed with ultrasound and long burs. Irrigation with NaOCl and EDTA.

It was an open apex (see CBCT scan radiograph (Fig 5), and the MTA was difficult to place properly. The apical beeding was controlled with CaOH2

The tooth was filled with MTA, IRM and composite.

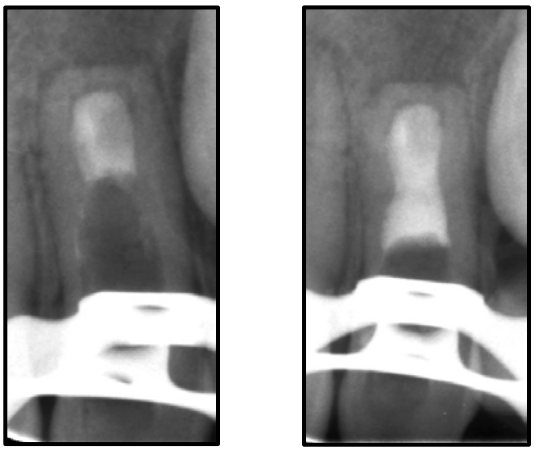


Fig. 10 MTA apically

Fig 11. Final radiograph

Prognosis

Endodontic: the prognosis seemed to be uncertain

Tooth: the prognosis seemed too be uncertain

Discussion

Dens invaginatus is a well-recognized phenomenon attributed to uncoordinated growth of part of the epithelium of a tooth germ in which the abnormal epithelium invaginates (1). Various names have been proposed to designate this phenomenon: dens in dente, dilated composite odontome, dents telescopes, gestant anomaly or dens invaginatus. The term most commonly used today is dens invaginatus, which was first suggested by Hallett et al.,(2).

The aetiology of this anomaly remains controversial (3,4), and although many hypotheses have been proposed, none have been proven or widely accepted (4). Thus, controversy exists in the dental literature regarding the terminology to be used for this anatomical defect (5). Studies have reported a high prevalence of variation for this anomaly. The wide discrepancy in its prevalence may be attributed to the different criteria applied by various investigators, (1,6) geographical differences (6) or different investigation methods (6,7).

Radiographically, this anomaly exhibits a radiopaque invagination that is equal in density to enamel and extends from the cingulum into the root canal (8). Dens invaginatus can be classified according to severity, with the most commonly accepted classification belonging to Oehlers (9), who described three types: (Fig)

-Type I, an enamel invagination in the crown only.

-Type II, an enamel-lined invagination that invades the root but remains confined within it as a blind sac and may communicate with the dental pulp;

-and type III, an invagination that extends from the crown to the apex and is penetrated by a second foramen laterally or apically on the root surface (c and d). In this type, any infection within the invagination can lead to an inflammatory response within the periodontal tissues, giving rise to a peri-invagination periodontitis (10). 'dens invaginatus' the clinical appearance of the crown may vary, ranging from a normal form to more unusual forms such as greater labio-lingual or mesio-buccal diameter, peg-shaped, barrel-shaped and conical. There may also be an associated talon cusp or grooving of the palatal enamel, coincident with the entrance of the invagination (3, 5, 10b).

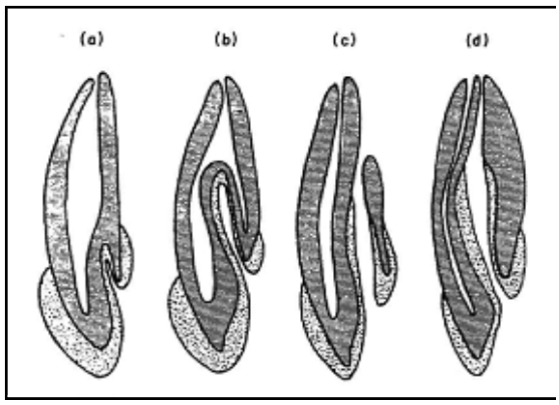
Because of its abnormal anatomical configuration, an invaginated tooth presents technical difficulties in its clinical management. Depending on the degree of malformation and the presence of clinical symptoms, there are different treatment modalities. Various techniques have been reported to treat dens invaginatus, including prophylactic treatment (11), conservative restorative treatment (12,), non- surgical root canal treatment (15), , endodontic surgery (13,14) and extraction.

Bacterial contamination of the invagination that occurred subsequent to the eruption of the tooth resulted in infection of the invagination, and this, in turn, led to the development of periapical inflammation (3). In this type of dens invaginatus, the invagination penetrates the entire root, usually without any communication with the pulp (15). The presence of communication between the invagination and the pulp may have important prognostic value; however, the tooth reported in this case did not appear to have any communication because the pulp responded to sensibility tests both before and after treatment. Clinical exploration during treatment of the invaginated canal corroborated this assumption.

If the invagination is necrotic or there is a 'peri-invagination periodontitis' and the pulp remains healthy, only the invaginated canal has to be treated (1, 3,15)

Dressing with Ca(OH)2 for extended periods has been reported to be associated with a higher risk of root fracture (16, 17).

Mineral trioxide aggregate (MTA) has been proposed as a promising alternative to $Ca(OH)_2$ in the treatment of non-vital permanent teeth with open apices (18,19).



Classification of invaginated teeth by Oehlers 1957

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