UNIVERSITY OF OSLO
DENTAL FACULTY

Department of Endodontics

Postgraduate program in **Endodontics**

Case book

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Contents

Case 1

Vital pulp-therapy

Symptomatic pulpitis of tooth 36 and 37

Patient

A 28 year old caucasian female (figure 1) was referred from her general dental practitioner (GDP) to the Postgraduate Endodontic Clinic June 2003 for recurring pain from her left lower quadrant.

Figure 1

Medical history

The patient has asthma and took the following medication:
Ventoline®. Ventoline is an inhalation-aerosol or powder. Salbutamol is the active ingredient in Ventoline; it is an andrenergic substance with effect on $ß_2$- receptors in bronchial musculature making breathing easier.
Bricanyl®. Bricanyl has the active ingredient terbutalin. Terbutalin is also an adrenercic substance with the same effect as Ventoline.
Pulmicort®. Pulmicort is a corticostereoid, the active ingredient budesonid is a glucocorticoid for inhalation with antiinflammatory effect in the allergic reaction.
The patient smokes approximately 5 cigarettes a day. Patient informs of a case of sexual harassment two years previously. She does not want to elaborate, but says she finds intra-oral examination and treatment very unpleasant.

Dental history and chief complaint

The patient sought her GDP June 2nd 2003 due to sharp and lasting pain provoked by cold from the left lower molar region. The patient was sure the pain came from tooth 36. Tooth 36 was diagnosed with symptomatic pulpitis and access cavity preparation was made. Eugenol-pellets and IRM were placed. The frequency of spontaneous pain had increased since the received dental treatment June 2nd. She was scheduled for emergency treatment at the Postgraduate Endodontic Clinic June 18th 2003. Her chief complaint was persisting and intolerable pain from cold stimulus in the left lower molar region.

Clinical examination

Figure 2

Figure 3

Figure 4

The photos (figures 2, 3 and 4) show the region from the lower left second premolar to the lower left second molar. Extraoral examination was done without registration of pathologic changes. Upon intraoral examination there was no tenderness to palpation or percussion from any teeth in the left upper or lower quadrant. Pocket probing depth was within normal limits. Tooth 35 had an MOD composite restoration. The first molar had an MO and an OD composite, and an occlusal temporary restoration. An arrested carious lesion with brown discoloration was present buccally. The second molar had an MO composite restoration. The patient told of some tenderness to biting from tooth 36. Teeth 34 and 35 were positive to electric pulp testing (EPT (~40/rate 5 scale 0-80)). Tooth 36 did not respond to EPT, and cold stimulus did not provoke any pain or sensory response. 37 gave response both later and higher on the EPT scale compared to 46, 47. (~70 vs. ~45 on a scale from 0-80) Provoked with Endo-Ice®, tooth 37 responded with intense pain lasting for 1-2 minutes after removal of the cold stimulus. Patient was informed about the implications of the findings. The referring dentist was consulted and the decision was made to initiate endodontic treatment of tooth 37.

Radiographic examination

Figure 5

The pre-treatment radiograph (figure 5) shows the region from the lower left second premolar to the lower left second molar. Tooth 35 had an MOD radioopaque restoration resembling a composite. The pulp space was clearly visible and the PDL-space could be followed all the way around the root. Tooth 36 had an MOD radioopaque restoration resembling a composite. The PDL-space can be followed around the two roots. Both roots were slightly curved towards the distal. Tooth 37 had an MO composite restoration that, in the plane of the X-ray, lay in close proximity to the pulp chamber. The pulp space was easily discernible in the two roots, and the lamina dura could be followed around the two roots.

Diagnosis

Acute pulpitis tooth 37
Chronic pulpitis tooth 36

Treatment plan

Pulpectomy of teeth 36 and 37

Treatment

Tooth 37

First visit
Two carpules Xylocaine with Adrenaline (X/A®) + 1 carpule Septocaine® were administered prior to treatment. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol. During treatment intra-ligamentary and intra-pulpal anaesthesia was also needed. An access cavity was prepared with steel fissure-burs in a high-speed handpiece and long shank round burs # 16 and12 in a slow-speed handpiece. 3 canal-orifices were localised. Initially the strong bleeding impeded proper vision of the canals. A working length X-ray (figure 6) was exposed with lengths indicated by the apexlocator: D K-20/21mm ref DLC, MB K-15/19.5mm ref MBC, ML H-15/19.5mm ref MLC. Corrections of lengths were made applying the apexlocator again when bleeding had subsided. The canals were instrumented to a length of 21mm with Protaper files S1, S2, F1 and F2 (figure 7). The D canal was instrumented as if it were two. During treatment the tooth was irrigated with Sodium hypochlorite (NaOCl) 1% and EDTA 15%. The canals were dried with sterile paperpoints. Due to lack of time, a slurry of calcium hydroxide in saline ($Ca(OH)_2$) was applied by counter-clockwise rotation of the last size K-file used, as an inter-appointment dressing. Cavit-G was placed in the canal-orifices and the tooth temporized with IRM.

Figure 6

Figure 7

Second visit August 27th 2003
The patient had been asymptomatic from 2 days following treatment of 37. Tooth 37 was objectively asymptomatic. Rubberdam was applied and disinfected as previously described. The $Ca(OH)_2$ was washed out with NaOCl and EDTA. Apical box-preparations were done with hand instrument K-files #35 through #45 in the mesial canals and #35 through 55 distally. The canals were dry. The mesial canals joined apically. This was confirmed by the two masterpoints not reaching working-length at the same time, but separately.

A masterpoint radiograph with 02-taper points was taken: 45/19 MB, 45/21 ML 55/21 D (figure 8). The rootfilling was placed using the lateral condensation technique with accessory points B and C and sealer AHPlus. Cavit-G and IRM was placed as topfilling. A post-treatment radiograph was exposed (figure 9).

Figure 8

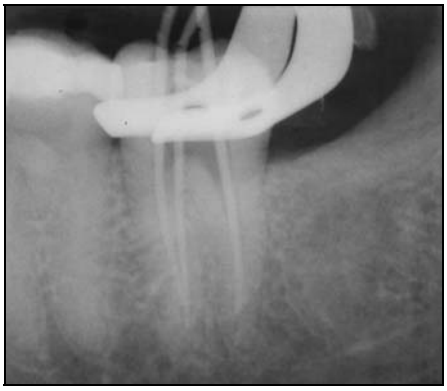

Figure 9

Tooth 36

First visit September 2nd 2003

Two carpules X/A® and 2 carpules of Septocaine® were administered to establish anaesthesia. A pre-treatment radiograph was taken (figure 10). The access-cavity preparation was modified and four canal-orifices localized. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol. Intrapulpal anaesthesia was needed. The treatment-session had to be terminated because of the patient's discomfort both physically and mentally. A eugenol-pellet was placed in the access cavity and covered with IRM as temporary filling. A prescription for Ibuprofen® no: 20 were given.

Figure 10

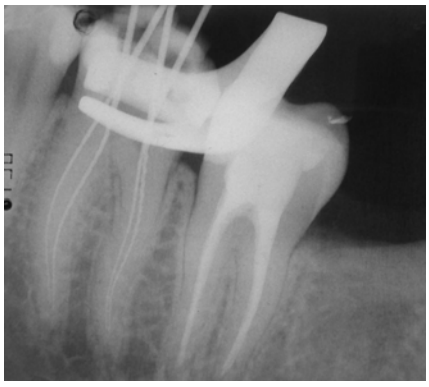

Figure 11

Second visit, September 10th 2003
(one week later)

Two carpules of Septocaine® were administered to establish anaesthesia. Rubberdam was applied and disinfected by proper means. The temporary restoration was removed. A stop in the DB canal was negotiated with rich amounts of EDTA and K-files #6, 8 and 10. Bright red bleeding was registered from all canals. A WL-radiograph (figure 11) was

taken after subtracting 1 mm from the length where the apexlocator indicated being in the PDL: K-15/24mm MB, H-15/24mm ML, K-15/23mm DB, H-15/23mm. The DL canal was instrumented with stainless steel (SS) K-files# 15 through 30 followed by NiTi K-files 35-50. The remaining canals were instrumented with Protaper files S1, S2, F1 and F2. Apical box preparations were done with NiTi K-files 35-45 mesially and 35-50 DL. Irrigation was done with copious amounts of NaOCl and EDTA. Canals were dried with sterile paper-points. A masterpoint radiograph (figure 12) was taken and the rootfilling placed: MB 45/24mm, ml 45/24mm, DB 50/23mm, DL 50/23mm. Accessory points B and C were placed with cold lateral condensation. Sealer was AH Plus. Cavit-G was placed over the canal orifices and IRM placed in the access cavity. A post-treatment radiograph was taken (figure 13).

In the epicrisis to the referring dentist it was suggested to leave Cavit-G over the canal-orifices under a composite restoration or metal-ceramic crown.

Figure 12

Figure 13

Prognosis

The prognosis was good for 36 and 37.

Follow-up

The patient returned one year later for a routine follow-up. The patient had no problems from the region. The X-ray (figure 14) showed no signs of periapical pathology.

Figure 14

Discussion

Endodontic treatment of the vital pulp is very successful provided the pulpectomy is skilfully performed and aseptic conditions are maintained. Pulpectomy with a subsequent root canal filling has a success rate of 90-96% (1).

The optimal placement of the wound surface in vital cases is guided by the attempt to make an atraumatic and aseptic pulp surgery with the smallest possible wound surface. The optimal wound surface seems to be 1-2mm from the radiographic apex. The pulp remnant will have ample blood-supply to keep vital and most of the apical delta found in many teeth is left untouched (2). If the length of the pulp remnant exceeds 2mm, the risk of necrosis from the pulp injury increases (3). In the case of tooth 36 both a straightening of the distal canals and some loss of working length (compare figure 11 and 12) can be observed. This did not seem to affect the treatment outcome.

Ideally, pulpectomy should be performed in one visit, The only true contraindication to this is apical tissue hemorrhage that cannot be fully controlled since sealers bond poorly to wet dentin. This was not the case here. The probability of it being two teeth as source of acute pain at one point in time is low (4). In an acute situation there is always the chance of mis-diagnosing, and here it may be that the dentist was led by the patient's statements more than the clinical evaluation.

References:

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3. Davis MS, Joseph SW, Bucher JF (1971) Periapical and intracanal healing following incomplete root canal fillings in dogs. Oral Surgery, Oral Medicine, Oral Pathology 31, 662-75.
4. Tronstad L. Clinical Endodontics 2nd Ed. P. 141-6. Thieme 2003.

Case 2

Treatment of a maxillary left first molar with atypical canal morphology

Patient

A 30 year old Asian female (figure 1) was referred from her General Dental Practitioner (GDP) to the Postgraduate Endodontic Clinic, Faculty of Dentistry, University of Oslo, in November 2002 for treatment of the maxillary left first molar (tooth 26).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

Tooth 26 had an OD gold-inlay done at the Student-clinic at the University of Oslo in 1999. The patient sought her GDP in September 2002 because of discomfort from tooth 26. Recurrent caries was found in relation to the inlay. The inlay and the caries were removed and an IRM placed as a temporary filling. The discomfort persisted and accentuated. The chief complaint was pain to cold stimuli. November 26th she had emergency treatment performed on the upper left first molar; an access-cavity was prepared and cotton-pellets with eugenol were covered with IRM.

Clinical examination

The pictures show the area from the upper left first premolar to the upper left second molar (figures 2, 3 and 4). Tooth 25 had an MOD tooth-coloured restoration. Tooth 26 had an occlusal temporary filling and a tooth-coloured restoration at the distal and palatal aspect of the tooth. Tooth 27 had an MO gold inlay. The gingiva was pale and firm. No pockets deeper than 3mm were found. Tooth 26 had persisting symptoms to cold, and pain on biting. Teeth 27 and 25 had normal reactions to cold, and like 26 elicited no tenderness to palpation or percussion.

Figure 2

Figure 3

Figure 4

Radiographic examination

Figure 5

The radiograph (figure 5) shows region 25-27.Tooth 25 had a radio-opaque material consistent with an MOD composite-filling. The pulp space was clearly visible. The lamina dura could be followed all the way around the root. Tooth 26 had an OD restoration in close relation to the pulp chamber. 3 separate roots were visible. The pulp spaces in the roots were hard to discern. The PDL-space could be followed all the way around the roots. Tooth 27 had a radio-opaque zone mesially and occlusally comparable to a metallic restoration. The three roots were gathered, and the PDL-space could be followed around them. The marginal bone level was within normal limits.

Diagnosis

Symptomatic pulpitis tooth 26

Treatment plan

Pulpectomy of the upper left first molar

Treatment

First visit November 27th 2002

One carpule of Xylocain-Adrenalin® was used to establish anaesthesia. Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol. The temporary filling was removed with a round carbide bur in a high-speed handpiece. The access-cavity was re-entered and a mesiobuccal, a distobuccal and two palatal canal-orifices were localized. The canals were negotiated with NiTi K-files #15 using a Root ZX® apexlocator. A working-length (WL) radiograph (figure 6) was taken: Mb K-file #15 18mm reference point MBC, db K-file #15 18mm ref MBC, p1(m) H-file #20 19.5mm ref MPC, p2(d) K-file #20 19mm ref MPC. Irrigation was done with copious amounts of sodium hypochlorite (NaOCl) and EDTA. Due to lack of time, root-canal-enlargement was terminated at an apical size #30. $Ca(OH)_2$-slurry was placed as inter-appointment dressing. The tooth was temporized with IRM.

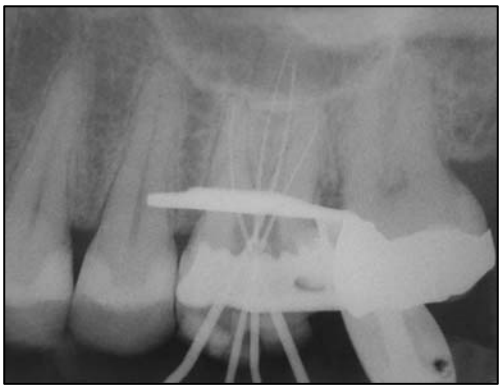

Figure 6

Second visit December 11th 2002, 15 days later

The tooth was asymptomatic. Rubberdam was applied and disinfected with chlorhexidine-ethanol solution. IRM was removed and the $Ca(OH)_2$ washed out, additional apical enlargement and flaring was done with K-files and Hedstrom hand instruments. Irrigation was done with NaOCl and EDTA. The presence of a possible mp canal was sought in the line between p1 and mb orifices without results. A masterpoint radiograph was exposed (figure 7). All canals were obturated with 02-taper guttapercha cones using the cold lateral-condensations-technique: Mb 40/18mm, Db 40/18mm, P1 50/18mm, P2 50/19mm. The sealer was AH Plus. The Access-cavity was filled with IRM. A post-treatment radiograph was taken (figure 8).

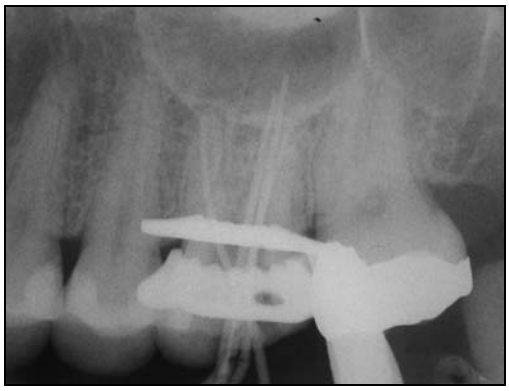

Figure 7

Figure 8

Prognosis

The prognosis was assumed to be good.

Follow-up

The patient was recalled after two years for evaluation of the endodontic treatment. The tooth was restored with a metal-ceramic prosthetic crown (figures 9 and 10). The tooth was asymptomatic. No periapical pathology was found on X-ray (figure 11).

Figure 9

Figure 10

Figure 11

Discussion

Two root canals are present in the palatal root of maxillary first molars less than 5% of the time. A suggested nomenclature is p1 mesially and p2 distally (1). There was loss of instrumentation length in the p1 palatal canal, or at least the canal was filled short. The mastercone has failed to reach the working-length in the p1 canal. I failed to anticipate the clinical implication of the root canal morphology. I should have checked that both my mastercones reached their intended position simultaneously. I overlooked the information my mastercone-radiograph gave me. I should have placed the p1 mastercone first to mimic my WL-radiograph. The canal-configuration (figure 12) in the palatal root might be a Vertucci type II or III (2).

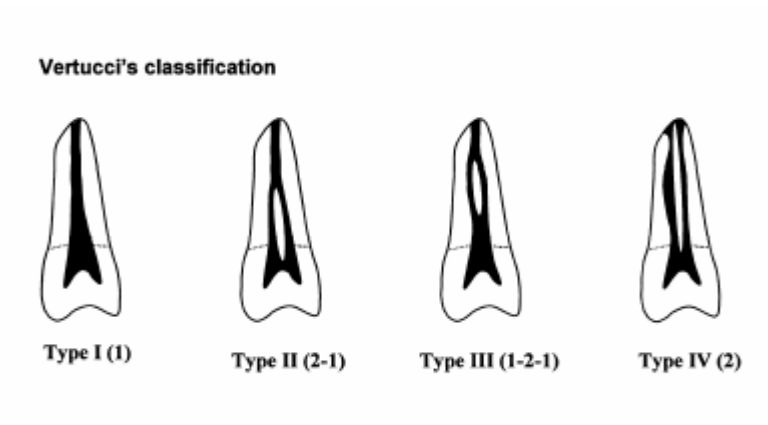

Figure 12

In vital pulp therapy partial pulpectomy seems to be the preferred technique (3). In the absence of infection it has been shown that soft and hard tissue repair takes place even if the canal was filled substantially short of working length (4). This supports my assumption that, given proper asepsis during treatment, the prognosis should be good in this case. Even so, it is sound to recall patients after vital pulp therapy also, as an apical periodontitis may have been induced during treatment. The peak incidence of emerging apical periodontitis seems to be at 1 year, with no added risk after that (5)

References:

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2. Vertucci FJ. Root canal anatomy of the human permanent teeth. Oral Surgery, Oral Medicine and Oral Pathology 1984: 58, 589-99.
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5. Ørstavik D. Time-course and risk analyses of the development and healing of chronic apical periodontitis in man. Int Endod J 1996:29:150-55.

Case 3

Treatment of a non-vital tooth

Chronic apical periodontitis of the lower right second molar

Patient

A 79 year old white female (figure 1) was referred to the Postgraduate Endodontic clinic, Faculty of Dentistry, University of Oslo from the student clinic in January 2003, for treatment of the lower right second molar (tooth 47).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

The patient had mild discomfort from region 47, and it had been like that for more than two years.

Clinical examination

Figure 2

The pictures show region 44-47 (figures 2, 3 and 4). There was some plaque-accumulation at the gingival margins. Pocket probing depth (PPD) was within normal limits (WNL). Tooth 44 had an OD amalgam restoration and a buccal composite with a surface defect and some discoloration at its superior margin. An infraction of the buccal enamel was visible. Tooth 45 had an MOD gold onlay with both cusps covered. Centrally in the occlusal surface an amalgam restoration was visible. On the buccal surface there were two composite restorations and some discoloration. Tooth 47 had a metal-ceramic full crown. The margins were probed without signs of poor fit. No pockets deeper than 3mm were detected, but the tooth had a furcation involvement of grade I buccally. There was a vague swelling buccally of 47. The swelling was firm and seemed fixed to the underlying bone, as if part of it. This area was tender when palpated.

Figure 3

Figure 4

Radiographic examination

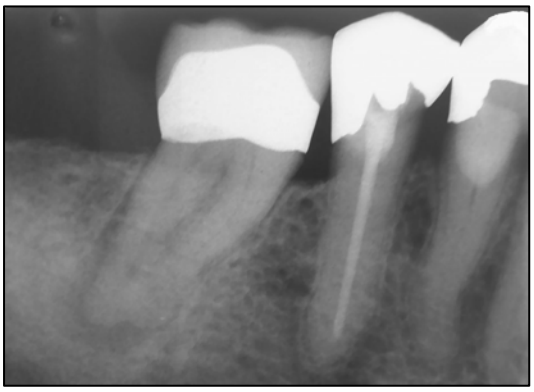

Figure 5

The radiograph shows region 44-47 (figure 5). There was a general marginal bone loss of between two and four millimetres. Tooth 44 had an OD radioopaque restoration resembling an amalgam and an oval, less radiodense zone in the cervical area correlating to the buccal composite. The PDL-space could be followed around a slightly curved root. The pulp space was clearly visible. Tooth 45 had an MOD radioopaque restoration of metallic origin. Centrally in the tooth a more radiodense line was seen, representing a root filling material. The PDL-space could be followed around the root. Tooth 47 had a coronal restoration resembling a metal-ceramic crown. The angle of the crown was a bit out of line with the long-axis of the tooth. The PDL-space could be followed down the mesial aspect of the root where it tapered into a radiolucency that extended from the root-tip of the mesial root around the apex of the distal root and 2/3rds up the distal root. Its size was estimated to 4 x 7 mm.

Diagnosis

Chronic apical periodontitis tooth 47

Treatment plan

Treatment of necrotic pulp tooth 47

Treatment

First visit, May 6th 2003

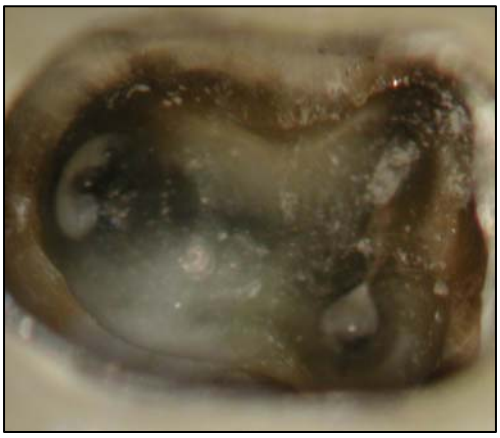

Figure 6

An access cavity was prepared with steel fissure-burs in high-speed handpiece and long shank round burs # 16, 12 and 10 in a slow-speed handpiece. MB, ML and D canal orifices were localised (figure 6). Pus evacuated from the ML canal, pus and blood from the distal. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The access-cavity and the distal canal-entrances were checked for signs of fracture-lines without positive findings. The canals were moderately obliterated. To obtain lenghts that gave response from the apexlocator, K-files #10 through #06 were used both to flare and with modified balanced-force-technique. In the MB canal appropriate working length could not be obtained. A WL radiograph (figure 7) was taken with K-file 15/20.5mm MB ref MLC, H-file 15/25.5mm ML ref MLC, K-file 15/21mm D ref DBC. Instrumentation was done with Protaper files S1, S2, F1 and F2 after a K-file #20 could pass easily to the working length. Flaring of the canal-entrances was done with the Sx-file before the use of the finishing files. Apical box preparations were completed with K-files to MB 45/20.5mm, ML 45/23.5mm, D 60/21mm. The canals were irrigated with copious amounts of 1% NaOCl and EDTA 15% and dried with paperpoints. $Ca(OH)_2$ was inserted with the last K-file for each canal using counter-clockwise rotation. The $Ca(OH)_2$ was then packed with paperpoints, and Cavit-G was placed as a temporary filling.

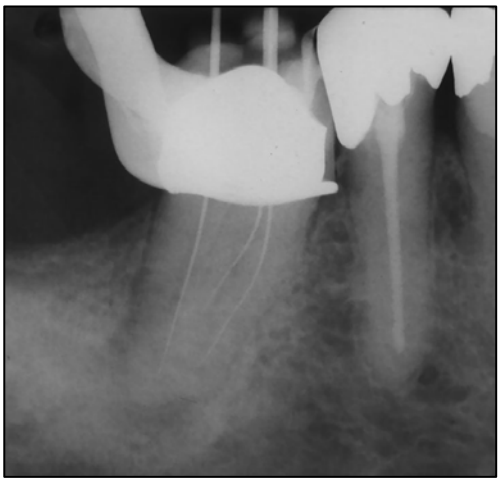

Figure 7

Second visit, May 28th 2003
(3 weeks later)

The tooth was asymptomatic. Rubberdam was applied and disinfected by proper means. The $Ca(OH)_2$ was washed out with the aid of NaOCl and EDTA. All canals were dry. A Masterpoint radiograph was taken (figure 8). The tooth was filled with 02-taper guttapercha 45/20.5 MB, 45/23.5 ML, 60/21 D using cold lateral condensation technique with accessory-points B and sealer AH Plus. The guttapercha was severed 1-2 mm into the canal orifices and excess of sealer removed with CHX (figure 9). Cavit-G® was placed over the canal orifices and IRM in the access cavity. A post-treatment radiograph was taken (figure 10).

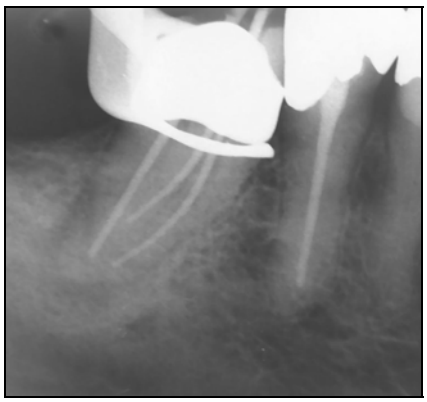

Figure 8

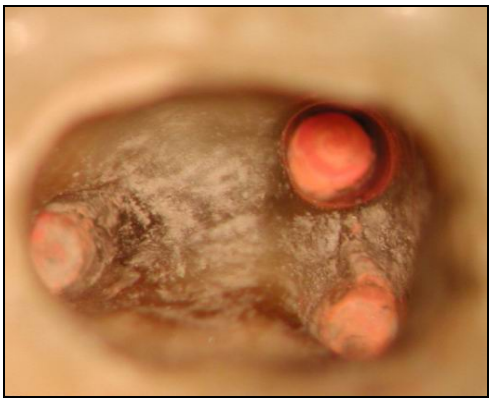

Figure 9

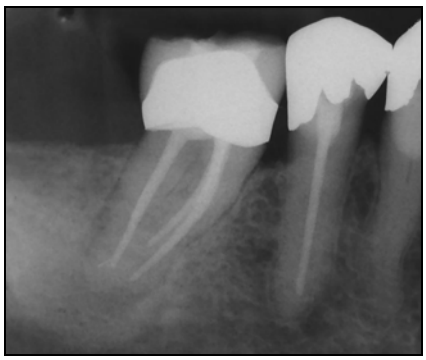

Figure 10

Third visit June 18th 2003, four weeks later

Rubberdam was applied and the bulk of IRM removed. Cavit-G® was left over the canal-orifices. The access-cavity was acid-etched, blow-dried and bonded with Syntac Sprint® and a Z-250® A3 composite restoration was light-cured in sections. The rubberdam was removed and the occlusion was corrected.

Prognosis

The prognosis was deemed uncertain due to the atypical shape of the radiolucency and the chance of having overlooked an infraction line in the distal root.

Follow-up

At 3 months, October 8th 2003, the patient was still asymptomatic and PPD was within normal limits. The radiograph showed clear signs of healing (figure 11).

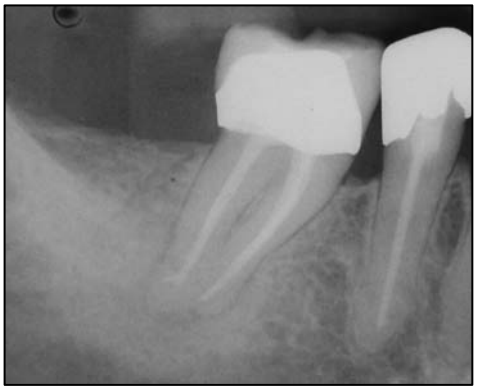

Figure 11

Two years after treatment the radiograph showed complete healing (figure 12).
At the clinical evaluation (figures 13 and 14) the tooth was asymptomatic and the PPD was still within normal limits. The oclusal composite showed signs of wear.

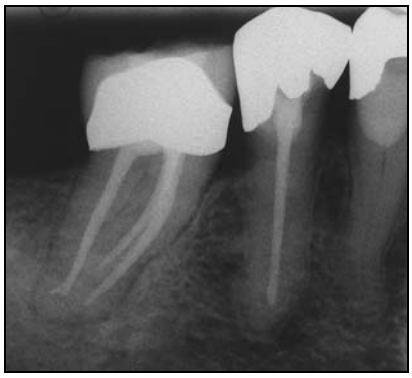

Figure 12

Figure 13

Figure 14

Discussion

Apical periodontitis is the host response following microbial infection of the pulp space (1, 2). There are several pathways by which the bacteria may invade the pulp. Deep carious lesions give direct entry. Bacterial entrance may also be a sequel to cavity preparation. Of vital teeth that have undergone fixed prosthodontic treatment more than 10% are expected to need endodontic treatment over a 10-year period (3). A recent report indicates that these numbers may be even higher (4). They found that 15% of crowns and 30% of abutments in fixed bridges needed root canal treatment (RCT) after ten years, and 20% and 35% after fifteen years.

Treatment of apical periodontitis is aimed at elimination of the microbial invasion of an otherwise sterile compartment. Success of initial treatment of apical periodontitis, based on radiographs, indicates a success-rate of 83-94% from well-controlled studies and 61-77% from epidemiologic studies (5). In this case the shape of the radiolucency prior to treatment could indicate a vertical root-fracture with a hopeless prognosis. Since no periodontal pockets > 4mm were found, RCT was initiated after informed consent. Proper WL was not achieved in the MB canal. In a tooth with apical periodontitis this impedes the prognosis of the treatment. In a long-term follow-up study of non-vital teeth with periapical lesions it was shown that instrumenting and filling close (0-2mm) to the apex had a success-rate of 94%, whereas when filled short (>2mm) of the root apex the success-rate was 68% (6). This is probably due to an inability to debride the apical segment of the canal.

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Case 4

Treatment of a non-vital maxillary left lateral incisor with dens invaginatus

Patient

A fortytwo year old Caucasian female (figure 1) was referred to the Postgraduate Endodontic Clinic, Faculty of Dentistry, University of Oslo, by her general dental practitioner (GDP) September 2002 for treatment of the upper left lateral incisor (tooth 22).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

Her main problem was pain on biting and intermittent swelling of the buccal mucosa from the maxillary left lateral incisor to the maxillary left canine over a period of 5 months. The GDP had placed a composite restoration in the gingival area distopalatally when first the discomfort started. It was later discovered on X-ray that there was a radiolucent zone periapically. Deviations in normal anatomy of the tooth restrained the GDP from accessing the pulp. Over the summer the patient was twice given antibiotics to reduce pain and swelling in the buccal mucosa.

Clinical examination

Figure 2

Figure 3

The photos (figures 2 and 3) show the region of the upper left central incisor to the upper left canine. The crowns of 21 and 23 were intact. Some discoloration was seen. A palato-gingival groove was found at the distal aspect of the palatal surface of tooth 22. In the region of the palato-gingival groove a faint bluish hue marked the composite placed by the GDP. The gingiva here seemed a little ruffled. The patient presented with tenderness to palpation buccally of 22. The pulp gave no response to Endo-Ice® and electric pulp testing (EPT). Pocket probing depth (PPD) was 5 mm distopalatally.

Probing the pocket it was clear that the gingival groove extended apically as a radicular groove. A guttapercha was placed in the pocket, but re-emerged (figure 4). No communication with the periapical lesion could be found with a fine probe.

Figure 4

Radiographic examination

On the periapical radiograph (figure 5) the maxillary left central and lateral incisor and canine can be seen. Teeth 21 and 23 had no restorations. The PDL-space could be traced all the way around the roots and the radiolucent area representing the pulp spaces were clearly visible. The crown of tooth 22 was intact, save the more radioopaque oblique line representing the composite placed in the palatogingival groove. The PDL could be traced down the mesial aspect of the root. The root itself was curved towards the distal in the plane of the X-ray. Halfway down the root the lamina dura tapered into a less radiolucent area approximately 7 mm in diameter.

Figure 5

Apically the root was more slender than would be expected from normal anatomical variations. The less radioopaque area centrally in the tooth was less pronounced than in the neighbouring teeth. The outline of the root could be followed up to the cervical area. Instead of leading directly up to the enamel-covered part of the crown distally, something that had the shape and radiodensity of dentine mimiced an accessory root. The PDL-space around this structure was difficult to extinguish. There was a marginal bone loss of 2-3 mm interdentally. A radiograph tracing the guttapercha was taken (figure 6). A CT-scan was performed, but the radiology department at the Faculty of Dentistry at the University of Oslo keeps the copyright to the radiographs and have not given permission topublish them.

Figure 6

Diagnosis

Chronic apical periodontitis of tooth 22
Dens Invaginatus type 3 (Oehlers)
Palato-radicular groove

Treatment plan

Treatment of the necrotic pulp in the main canal only
Intervention of the invagination was to be postponed and evaluated at a later stage. At the CT-scan of the left lateral maxillary incisor the presence of connective tissue inside the invagination could not be established from the scannings. Unfortunately the CT-scan is unavailable for presentation.

Treatment

First visit, September 4th 2002

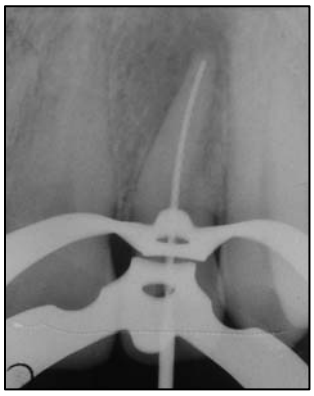

Figure 7

An access cavity was prepared with a diamond coated round bur in a high speed handpiece, followed by round burs # 14 and 12 in slow speed handpiece. The main canal was localized. No apparent communication with the invagination from the access cavity was detectable. Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol (CHX). The length of the canal was measured with a Root ZX® apexlocator. When the apexlocator indicated being in the PDL, one mm was subtracted from that length, and a working-length (WL) radiograph was taken (figure 7). The K-flexofile #15 at the WL-radiograph was 20mm from the incisal edge. The length was adjusted to 19.5mm. The canal was shaped with K-flexofiles #10-*20* and NiTi K-files *25*-50 with an apical box preparation and flared with Hedstrom-files. Irrigation was done with rich amounts of 1 % sodium hypochlorite (NaOCl) and 10ml 15 % EDTA. An inter-appointment dressing of $Ca(OH)_2$ mixed with sterile saline was introduced using the last K-file in counter-clockwise motion. The dressing was condensed with sterile paper-points. The tooth was temporized with Cavit-G covered with IRM. The patient was given instructions in personal plaque control.

Second visit, November 2nd 2002
(two months later)

The tooth was tender to percussion and palpation. The patient told of pain described as “electricity” radiating from the area of tooth 22. An X-ray was taken prior to re-entry to evaluate presence of $Ca(OH)_2$ in the canal, and possible consumption thereof since last appointment (figure 8). The temporary filling was intact. Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol. Irrigation was made with 1 % NaOCl and 15 % EDTA.

Figure 8



Fluid kept seeping into the root canal, and it could not be dried by means of sterile paper points. The WL was controlled with a new radiograph (figure 9). $Ca(OH)_2$ was packed as previously described, and a temporary filling of zinc oxide eugenol was placed.

Figure 9

Third visit, December 17th 2002
(fourteen weeks after initiation of treatment)

The X-ray gives an indication of new lamina dura forming at the mesial aspect of the root (figure 10). The patient still had discomfort from the area, and the tooth was somewhat tender to percussion. Rubberdam was applied and disinfected by proper means. Irrigation was made with 1 % sodium hypochlorite and 15 % EDTA. The canal was dry. It was decided to leave the invagination alone as the tooth seemed to respond to the treatment given. $Ca(OH)_2$ was packed as previously described, and a temporary filling of Cavit-G and IRM was placed.

Figure 10

Fourth visit, June 2nd 2003
(nine months after initiation of treatment)

The tooth was now asymptomatic. Rubberdam was applied and disinfected by proper means. The canal was irrigated with 1 % sodium hypochlorite and 10ml 15 % EDTA. The canal was dry. A masterpoint radiograph was taken (figure 11). The tooth was filled with guttapercha taper 02 ISO 50/19.5mm using the lateral condensation technique. Accessory points B, sealer AH Plus. The access cavity was washed with CHX. Pink Cavit was placed over the gp and moisted. When it felt solid, the walls of the access-cavity was rubbed with a diamond bur. Scotchbond-etch, All-bond-2 and a composite filling of Z-250 colour A3. A post-treatment radiograph was taken (figure 12).

Figure 11

Figure 12

Prognosis

The prognosis of the endodontic treatment was deemed uncertain.

One-year follow-up

September 21st 2004
(21 months after initiation of treatment)

Figure 13

The tooth was asymptomatic. A periapical X-ray was taken (figure 13). Radiographically the periapical lesion was healed.

Discussion

Dens invaginatus is a dental malformation, often leading to endodontic complications. The aetiology is unclear, but it is commonly believed that an invagination is the result of a deep folding of the foramen coecum during tooth development (1).The prevalence of dens invaginatus varies considerably depending on the type of classification. Ruprecht et al found dens invaginatus in 1, 7% of all examined patients (2). The anomaly arises most commonly in the maxillary lateral incisor and less frequently in the central incisors. It also occurs in posterior teeth, extending from the occlusal pit. Bilateral occurrence is a frequent finding; Grahnen et al reported 43% (1).

The most common classification was introduced by Oehlers in 1957 (figure 14) (3). Shultze and Brand introduced a more detailed classification also including invaginations starting at the incisal edge and dysmorphic root forms (figure 15) (4). The choice of treatment ranges from observation, fissure sealing, root canal treatment to extraction. Root canal treatment can imply endodontic treatment of the invagination alone, or the main canal, or both (5). Pulp necrosis is often found before root end closure (5).

Endodontic treatment in conjunction with endodontic surgery is also common when the anatomy hampers the ability to thoroughly clean and shape the canal(s) (5). The treatment outcome is linked to the difficulty of cleaning and shaping and the presence of a palato-radicular groove (PRG). The probability of periodontal breakdown is greater when a PRG is present. Mesial or distal PRGs are more often associated with pocket probing depth > 3mm (6). In this case, even though the periapical lesion is healed, a late failure is still possible if periodontal breakdown at the palatal side proceeds.

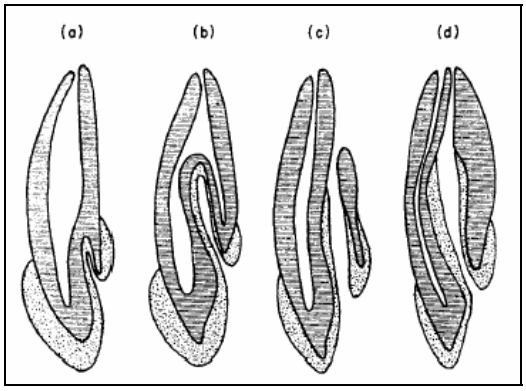


Figure 14 Classification by Oehlers 1957 (3)

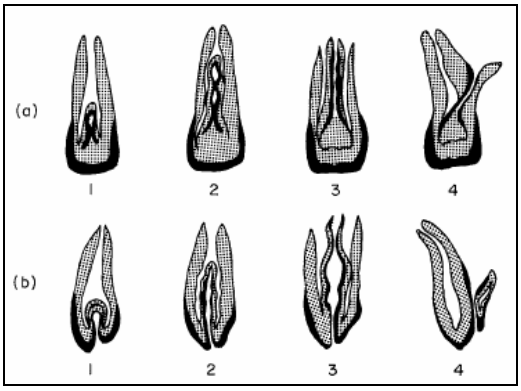


Figure 15 Schulze & Brand 1972 (4)

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Case 5

Teatment of a maxillary non-vital right lateral incisor with internal resorption

Patient

A thirtynine year old Caucasian male (figure 1) was referred from the Student Clinic to the Postgraduate Endodontic Clinic for treatment of the upper right lateral incisor (tooth 12).

Figure 1

Medical history

The medical history was non-contributory.

Dental history and chief complaint

The tentative diagnosis from the student was apical periodontitis and cervical root resorption. The tooth was to be part in a larger prosthodontic treatment. The patient was said to have grave dental anxiety. The patient had no pain or discomfort from the region of the upper right lateral incisor. He had an accumulated need for dental treatment. This was probably a result of anxiety for dental treatment, as mentioned in referral, combined with neglect, see full X-ray-status dated February 17th 2003 (Figure 2).

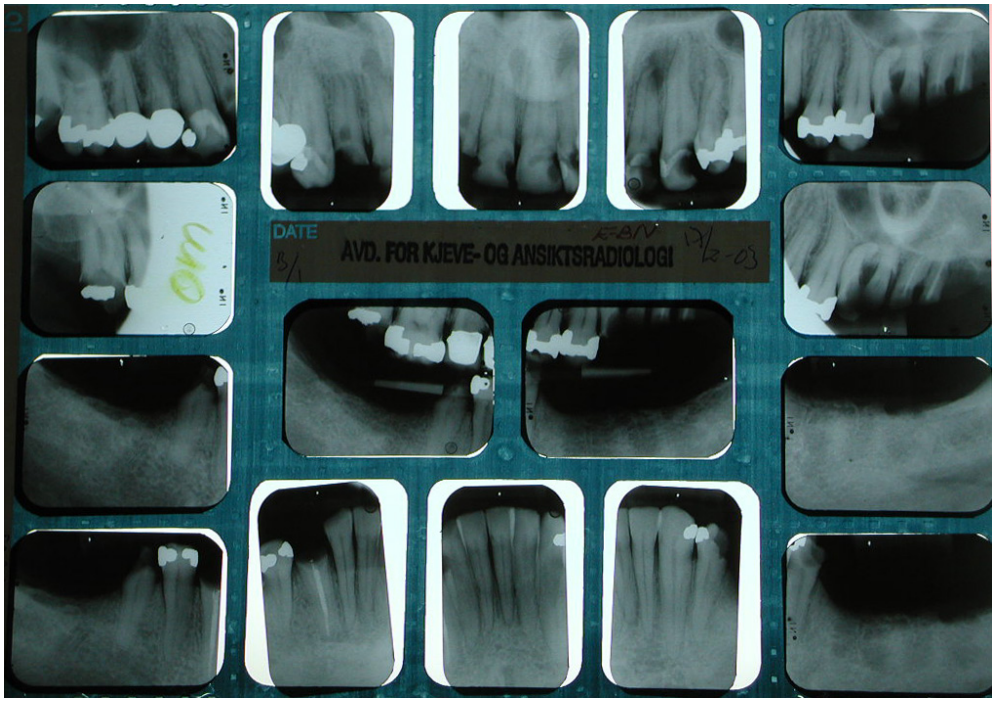

Figure 2

Clinical examination

There were no extra-oral or intra-oral pathological alterations of the soft tissues in region 12. The pictures (figures 3 and 4) show the region from the upper right canine to the upper right central incisor. Tooth 13 had a tooth coloured restoration with marginal defects and secondary caries at the gingival margin buccally. A temporary restoration covered most of the palatal and distal aspect of the tooth.

Figure 3

The bulk of the crown of tooth 12 was missing and a tooth-coloured restoration was placed at the buccal and incisal surfaces. The central incisor had buccal and mesial composite restorations. Distally the restoration was lost. There was recurrent caries cervical to the buccal filling. Tooth 12 reacted normally to palpation and percussion but gave no response to Endo-Ice® or electric pulp testing (EPT). Tooth 11 was positive to EPT.

Figure 4

Radiographic examination

Figure 5
Ortho-radial projection

Figure 6
Mesio-radial projection

Pretreatment radiographs (figures 5 and 6) were taken with different angulations. The radiographs show region 11 to 13. The central incisor had a radioopaque zone at the mesial aspect of the crown consistent with a composite filling. Tooth-structure was missing from the distal side of the crown. The pulp space was clearly visible.

The PDL-space could be followed around the tooth but widened into a radiolucent zone periapically (figure 5). However the lesion moved markedly in relation to the root comparing figure 5 and figure 6, and as the tooth responded positively to sensitivity-tests, it was assumed to be the canalis incisivus. The canine had a restoration at the distal/incisal aspect of the crown resembling a temporary filling and enamel-caries mesially. Centrally in the root a material with a radiolucency resembling guttapercha was visible. The PDL-space could be traced around the periapex. There was a general marginal bone loss of 1-2mm. The lateral incisor had a defect crown with an incisal/distal restoration with radiolucency comparable to a composite. The lamina dura could be followed to the apical third of the root where it widened into a radiolucent zone with a diameter of approximately 5 mm. The pulpal space could be seen with a somewhat irregular outline. The radiolucent areas superimposed on the pulpal space did not seem to move markedly from one angulation to the other. The outline of a normal anatomical pulpal space was not detectable within the lesions.

Diagnosis

Chronic apical periodontitis of tooth 12
Internal root resorption

Treatment plan

Treatment of necrotic pulp tooth 12

Treatment

First visit October 10th 2003

Figure 7

He consented to treatment without sedatives as long as he received local anaesthetic. 1 carpule of Xylocain-Adrenalin® (X/A) was administered. Caries and old fillings covering the entrance to the coronal pulp were removed, revealing that the pulp had not been previously exposed or accessed (figure 7). Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). An access cavity was prepared using round burs #14 and 12 in a slow-speed handpiece.

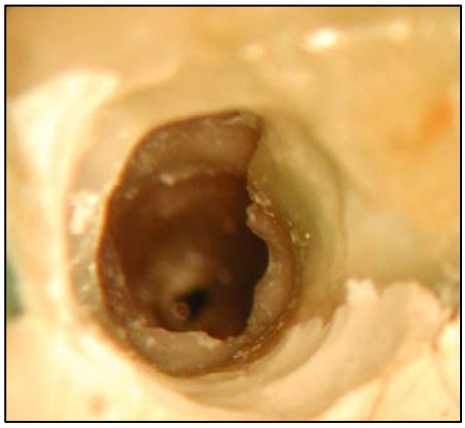

Figure 8 Access cavity

The pulp space was initially empty. All canal walls were smooth but highly irregular in shape and markedly tapered towards the coronal part (figure 8).

Exudate slowly filled the canal space (figure 9). When this subsided, the canal was dried with paperpoints and a slurry of Calcium-hydroxide-paste ($Ca(OH)_2$) was placed as dressing and covered with IRM.

Figure 9 Exudate

Second visit, October 24th 2003
(two weeks later)

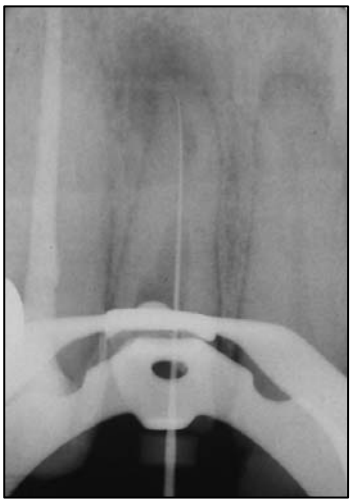

Figure 10

One carpule Citanest-Octapressin® was used to establish anaesthesia. Rubberdam was applied and disinfected with CHX. A K-file #15 was inserted in the canal. The patient experienced pain in the apical third of rootcanal and intra-pulpal injection of X/A was given. The #15 K-file was again inserted. When the Root ZX® apexlocator indicated being in periapical tissues, 1mm was subtracted from that length, and a working-length (WL) radiograph exposed (K 15/18.5mm figure 10). The same K-file was given an apical curve of approximately 70° (figure 11) and moved up and down the canal wall in the area representing the apical resorptive area. The apexlocator gave no audio response, and it was therefore assumed that the resorption had not perforated to the PDL. The canal was instrumented with SS and NiTi Hand-files to the size 70/18.5mm. Irrigation was done with copious amounts of 1% Sodiumhypochlorite (NaOCl) and 15% EDTA. The canal was dried with sterile paperpoints and $Ca(OH)_2$ was packed as inter-appointment dressing. The patient remarked on discomfort during this procedure. The access-cavity was rinsed with CHX and sealed with Cavit-G® and IRM®.

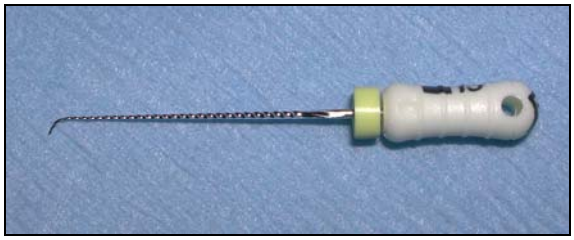

Figure 11

Third visit, November 11th 2003

The tooth was asymptomatic. One carpule of Septocaine® was administered to establish anaesthesia. Rubberdam was applied and disinfected as previously described. The canal was flushed with NaOCl and EDTA. The canal could not be kept dry. $Ca(OH)_2$ was inserted with a K-file #70 with counter-clockwise rotation and condensed with paperpoints. The canal was sealed with Cavit-G® and IRM®. An X-ray of the intracanal medicament was taken (figure 12).

Figure 12

Fourth visit, December 12th 2003
(eight weeks from initiation of treatment)

Figure 13

The tooth was asymptomatic. One carpule of Septocaine® was administered. Rubberdam was applied and disinfected by proper means. $Ca(OH)_2$ was washed out with NaOCl and EDTA. The canal was now dry, no moist was detected on a paperpoint inserted to working-length and left for a couple of minutes. Because of irregularities in the apical portion of the canal, the rootfilling was placed with the aid of System-B® (figure 13). The ISO 70-guttapercha was placed to working length (18.5mm) and a masterpoint radiograph taken (figure 14). Sealer AH Plus was transported to the apical region with paperpoints to leave a thin layer. A warm instrument was used to sever the gp at 15mm leaving an apical plug of 3.5mm. System-B was activated, inserted in the canal to 16.5mm, heat turned off while keeping a soft apical pressure for 3 seconds, reheated and removed, followed by axial condensation with a hand-instrument. An X-ray was exposed to check the apical fit (figure 15). A #80-cone was inserted and severed; leaving the apical 3mm. System-B was applied as previously described. The procedure was repeated twice, and X-ray exposed (figure 16). The remaining canal was filled with Cavit-G and the access cavity sealed with IRM. A post-treatment radiograph was exposed (figure 17). The referring student was advised against the use of a post. The resorption had weakened the cervical area of the tooth (2), and the irregularities would prompt removal of even more dentine to eliminate undercuts when preparing the post.

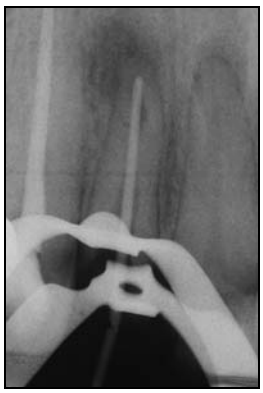

Figure 14

Figure 15

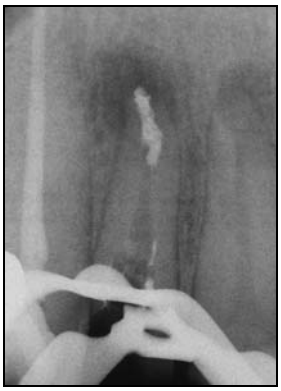

Figure 16

Figure 17

Prognosis

The endodontic prognosis was assumed to be good.

Follow-up

December 15th 2004. The patient came back for a clinical and radiographic examination one year after treatment. Unfortunately the patient had not permanently restored the tooth (figures 18 and 19). The X-ray showed signs of healing in progress (figures 20 and 21). He was put in contact with a prosthodontist.

Figure 18

Figure 19

Figure 20

Figure 21

Discussion

Internal root resorption is rare in permanent teeth, and is characteristized by an oval-shaped enlargement of the root canal space. External resorption, which is much more common, is often misdiagnosed as internal resorption. In internal root resorption there is resorption if the internal aspect of the root by multinucleated giant cells adjacent to granulation tissue in the pulp. For internal resorption to occur there has to be an infectious agent present together with vital pulp, and a breach in the odontoblast- and predentin layer. Reasons for the loss of predentin adjacent to the granulation tissue are not obvious. Trauma frequently has been suggested as an aethiological factor (1).

The usual radiographic presentation of internal root resorptions is a fairly uniform radiolucent enlargement of the pulp canal. Because the resorption is initiated in the root canal, the resorptive defect includes some part of the root canal space. Therefore, the original outline of the root canal is distorted. Only on rare occasions, when the internal resorptive defect penetrates the root and impacts the periodontal ligament, does the adjacent bone show radiographic changes. The treatment of the internal resorption is as for any necrotic tooth, with cleaning and shaping and the use of a dressing. The root should be filled with a warm technique because cold condensation poorly fills the internal defects. With modern dental techniques, this treatment alternative should be weighed against the advantage of implant dentistry (1).

Visually the rootfilling did not meet my requirements, being somewhat inhomogenous in appearance. Obtura® might have been a more appropriate tool for the task. Nevertheless there was no overfill, the root-filling was terminated close to the apex, and the irregularities of the apical resorption seem to be filled. The tooth was asymptomatic and the canal dry. Treatment was kept under close aseptic conditions. Therefore the endodontic prognosis was assumed to be good (2). In the apical part the $Ca(OH)_2$ should preferably have been packed to a radiodensity closer to dentine to confirm that all resorptive tissue was eliminated from the previous rounds of dressing.

A post was advised against as there was little dentine in the cervical area (3). The restorative prognosis of this tooth was probably more questionable than the endodontic prognosis. A firm grip for a prosthetic crown demands some sort of chamfer-preparation. A ferrule is supposed to give an “inner grip” that prevents vertical root-fracture, but it is usually combined with a post and therefore not suited in this case (4).

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Case 6

Treatment of a non-vital tooth

Apical periodontitis with sinus tract of the lower right second molar

Patient

A sixtytwo year old white male (figure 1) was referred from his general dental practitioner (GDP) to the Postgraduate Endodontic clinic, Faculty of Dentistry, University of Oslo, October 2005 for treatment of the lower left first molar (tooth 36).

Figure 1

Medical history

His medical history was non-contributory.

Dental history and chief complaint

The patient had periods with tenderness from the lower left molar region. The pain was low-grade and described as “pressure”.

Clinical examination

Figure 2

The pictures (figures 2, 3 and 4) show region 35 -37. Tooth 35 was missing. Tooth 36 had a buccal and an MODL amalgam restoration. Tooth 37 had an MOL composite restoration with discoloration at the margins. Buccally an amalgam was visible. Pocket probing depth (PPD) was within normal limits (WNL) for tooth 37. PPD in the furcation of 36 buccally was 10 mm.

Figure 3

Figure 4

Plaque-accumulation was visible at the lingual aspect of tooth 36, and approximately 1,5mm of the rootsurface was visible both at the buccal and lingual aspect.

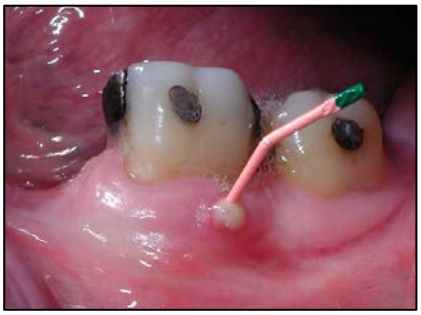

Figure 5

A sinustract was visible in the buccal mucosa of the first molar (figure 5). This was traced with a # 35 guttapercha-cone and an X-ray was taken (figure 7). An X-ray was also taken with a guttapercha in the buccal pocket (figure 8).

Radiographic examination

Figure 6

The radiograph (figure 6) shows region 35-37. Tooth 35 was missing. Tooth 36 had a coronal MOD restoration with the radiographic appearance of an amalgam. The two roots were straight with the pulps visible within the roots. The marginal bone loss of tooth 36 was 4 mm distally and 5 mm mesially. The lamina dura could be followed down the mesial root. In the apical third of the root it tapered into a radiolucent zone encompassing the periapical area of the mesial root and the whole furcation area. The apicomarginal extension of the radiolucency was 12mm. The lamina dura was visible down the distal aspect of the distal root, but missing down the mesial aspect of the distal root. A periapical radiolucency of 1mm was observed at the distal root.

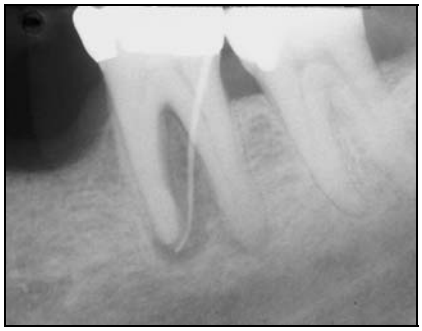

Figure 7

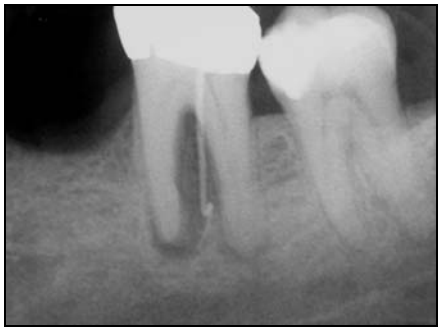

Figure 8

Diagnosis

Apical periodontitis with sinus tract tooth 36

Treatment plan

Treatment of necrotic pulp tooth 36

Treatment

First visit, October 27th 2005

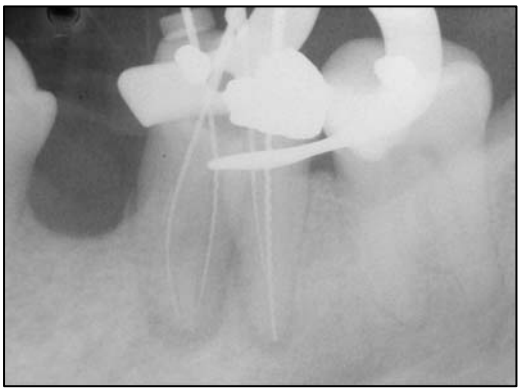

Figure 9

An access cavity was prepared with steel fissure-burs in high-speed handpiece and long shank round burs # 16 and 12 in a slow-speed handpiece. MB, ML and DB and DL canal orifices were localised. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). A WL radiograph (figure 9) was taken with K-file 15/21mm MB ref MBC, H-file 15/20mm ML ref MBC, K-file 15/21mm DB ref DBC and H-file 40/22mm DL ref DBC. The length in the distolingual canal was adjusted to 21mm. Instrumentation was done with Protaper files S1, S2, F1and F2 after a K-file #20 could pass easily to working length. Flaring of the canal-entrances was done with the Sx-file after the S2-file. Apical box preparation was completed with K-files to MB 45/21mm, ML 50/20mm, DB 50/21mm, DL 50/21mm. Canals were irrigated with 1% NaOCl and EDTA 15% and dried with paperpoints. A $Ca(OH)_2$–slurry was inserted with lentulo-spiral. $Ca(OH)_2$ in the access-cavity was removed with CHX and cotton pellets. Cavit-G was placed into the canal orifices and the access cavity filled with IRM.

Second visit, November 17th 2005
(3 weeks later)

Figure 10

The tooth was asymptomatic and the sinus tract had closed (figure 10). Rubberdam was applied and disinfected as previously described. The $Ca(OH)_2$ was washed out with the aid of NaOCl and EDTA. Ther was moist detected om the paperpoint placed in the ML canal. The RootZX® apexlocator indicated overinstrumentation. The length was corrected and the masterpoint radiograph taken (figure 11). The tooth was filled with 02-taper Resilon® mastercones 50/19.5 MB, 50/20 ML, 50/20 DB, 50/20 DL using cold lateral condensation technique with accessory-points MF and M. The sealer was Epiphany®. The cones were severed with a hot instrument two mm down the canal entrances and light-cured for 40 seconds. Sealer surplus in the pulp chamber floor was removed and an IRM placed in the canal orifices and access cavity. A post-treatment X-ray was taken (figure 12). Sealer surplus was registered at the mesial root.

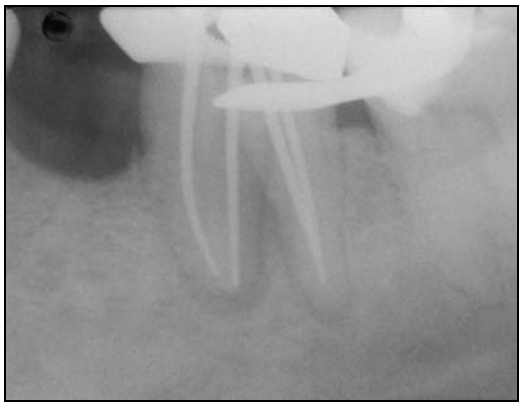

Figure 11

Figure 12

Prognosis

The prognosis was assumed to be good.

Follow-up

Figure 13

The patient was recalled after four months. The tooth had received a prosthetic crown the month following root canal treatment. Clinically the tooth was asymptomatic and no sinus tract was visible at the buccal aspect of the tooth (figure 13). PPD was now within normal limits also buccally. The two radiographs that were taken showed healing in progress (figures 14 and 15).

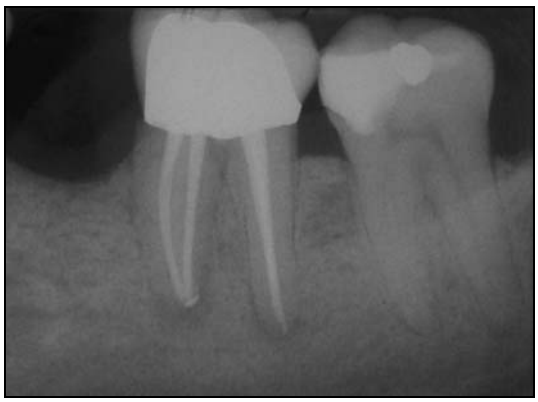

Figure 14

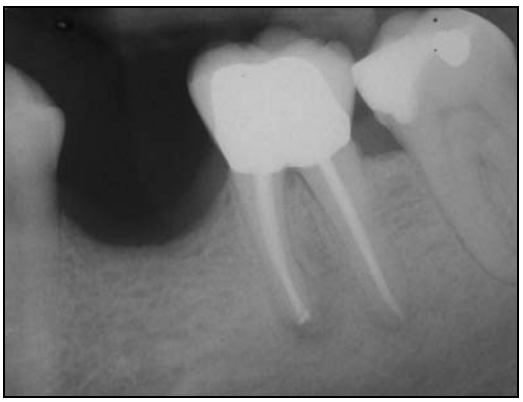

Figure 15

Discussion

All steps in treatment of apical periodontitis are aimed at eliminating microbial infection of the rootcanal-space and surrounding dentine. Whereas intra-operative factors such as the apical enlargement and extent of treatment does seem to have an impact on treatment outcome (1), the pre-operative factors age, gender and symptoms (here: sinus tract) do not seem to do the same (1).

A sinus tract is a state where dead and dying microbes and polymorphonuclear cells follow the path of least resistance to a surface. Cleaning and shaping of the root canal(s) eliminates the bulk of infection. Bacteria that have invaded the lateral canals and dentin canals, in particular, are largely beyond the reach of mechanical preparation. Microbes invade dentin to at least 250 µm in vivo (2). These bacteria can probably be targeted by irrigation by antibacterial solutions and, in particular, intracanal medicaments like calcium hydroxide, chlorhexidine or iodine compounds. The relative importance for the prognosis of treatment of deep dentinal infection and its elimination is, however, not known.

Calcium hydroxide ($Ca(OH)_2$) is a videly used intracanal dressing. Calcium hydroxide in a water vehicle has antimicrobial qualities that are believed to be due to the very high pH resulting from the dissociation of OH ions. The powder is poorly dissociated into calcium and hydroxide ions (3). An aqueous solution is saturated at 0.17%. Thus, most of the calcium hydroxide powder forms a slurry in water. Water must be present for the antimicrobial effect of calcium hydroxide. Calcium hydroxide is a slow acting agent and, in order to achieve sufficient antimicrobial effect, the dressing has to be left in the root canal for at least one full week (3). It appears that when well packed and allowed sufficient working time, the calcium hydroxide will completely disinfect the root canal with a high degree of predictability (3, 4). Disinfection is normally uncomplicated when treating teeth with infected necrotic pulps as the microflora is susceptible to calcium hydroxide (3, 4).

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Case 7

Treatment of a non-vital tooth

Chronic apical periodontitis of the upper left first molar with interappointment flare-up

Patient

A seventyfive year old white Norwegian female (figure 1) was referred from the Student Clinic to the Postgraduate Endodontic Clinic at the Faculty of Dentistry, University of Oslo, Oslo, Norway, February 2005 for treatment of the upper right first molar (tooth 16).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

Figure 2

Figure 3

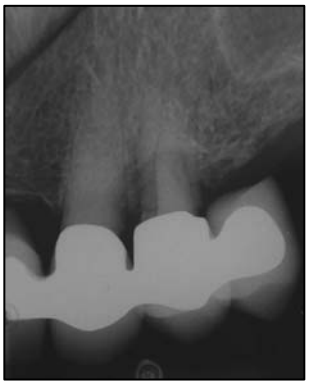


Figure 4

The tooth in question was a terminal abutment of a fixed full denture with a service-time of 6 years (figures 2, 3 and 4). Her primary dental problem was periodontal disease. She had received instructions in personal plaque control, and scaling, at the Student Clinic. The referral was made because of pain in the upper right molar region. The pain was throbbing and interfering with her sleep. It had started in the molar-region, but the tender area was now including the canine. She had previous experience with discomfort from the area, but not in this extent.

Clinical examination

Figure 5

Figure 6

The photos (figures 5 and 6) show the region from the upper right canine to the upper right first molar. Tooth 16 was the terminal abutment of a fixed full metal-ceramic denture (figures 5 and 6). 15 and 14 were pontics and 13, 12, 11, 21, 23 and 24 were abutments. The left first premolar had a distal cantilever. The pontics 14 and 15 rested extensively on the gingival mucosa. Tooth 16 was tender to percussion and palpation buccally. Tooth 13 and 11 tested positively to Endo-Ice®, but tooth 16 gave no response. The margins between the bridge and abutments were probed with no signs of loss of retention. Increased pocket probing depth (PPD) was registered. Tooth 16: 4mm M, 7mm P, 10mm D. Tooth 13: 4mm M+D. There was bleeding on probing from all pockets of tooth 16. Guttapercha was placed in the distal pocket, and a radiograph exposed (figure 8).

Radiographic examination

Figure 7

The radiograph (figure 7) shows tooth 16 as an abutment tooth of a fixed bridge of metal-ceramic origin where the premolars were pontics. Radiographically the restoration seemed to fit the abutment tooth acceptably. The lower border of the maxillary sinus was visible as a thin radioopaque line over the roots of tooth 16. Three roots were visible, the mesiobuccal superimposed over the palatal. The pulp spaces seemed obliterated. The lamina dura widened into a radiolucent zone of 5mm encircling the palatal root.

Figure 8

A radiolucency of 2 mm was also seen in the apical area of the distobuccal root. There was marginal bone-loss of 3 mm mesially and 7mm distally from the cervical margin of the distal abutment.

The problem regarding tooth 16 was explained to the patient and it was stressed that the outcome of endodontic treatment was uncertain. She still wanted to do whatever possible to keep the terminal abutment of the fixed bridge.

Diagnosis

Chronic marginal periodontitis of tooth 16
Chronic apical periodontitis
Probable established endodontic-periodontic problem

Difficulty assessment: Access through prosthetic bridge, obliteration of canals

Treatment plan

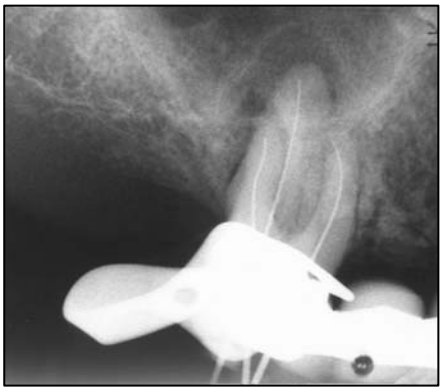
Treatment of necrotic pulp tooth 16

Treatment

First visit, March 3rd 2005

An access-cavity was prepared using round diamond-coated burs for the ceramics and hard-metal fissure burs in a high speed handpiece until the pulp-chamber was localized. A long-shank round bur # 14 was used to clean the pulpchamber and the MB and P canals were localized. A #10 round bur was then used to search for the DB canal. Due to lack of time a eugenol-pellet was placed in the access-cavity and sealed with IRM.

Second visit, March 15th 2005



Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The DB canal was now localized with the aid of the microscope, drilling deeper into the DB root. Canals were negotiated with stainless steel (SS) K-files 06, 08, 10 and 15. A working-length (WL) radiograph was exposed (figure 9) with the following measures:
MB K-file15/21mm ref MBC,
DB K-file 20/20mm ref DBC,
P H-file 20/21mm ref PC.

Figure 9

The canals were further instrumented with Protaper files S_1, S_2, F_1 and F_2 and K-file #35. The canals were irrigated with 1% sodium hypochlorite (NaOCl) and EDTA 15% and dried with paperpoints. Exudate was registered from all canals. $Ca(OH)_2$ was inserted with #35 K-file using counter-clockwise rotation. The $Ca(OH)_2$ was then packed with paperpoints and Cavit-G was placed as a temporary filling. The patient was given a prescription of Apocillin 660 mg x 20 since she was spending the Easter in the mountains. She was instructed to take the medication only if there was swelling or general malaise.

Third visit: Flare-up. March 16th 2005.

The patient returned the day after treatment with a swollen right cheek (figure 10). There was no fluctuation or restricted area where pus had seemed to gather. A 2 cm vertical incision was made at the buccal aspect of tooth 16. There was bleeding, but no pus, from the incision. Instructions were given to the patient to start with the prescribed tablets from the day before. A prescription of Ibumetin® 400mg x 20 was added. She was instructed to call if the situation did not improve, or worsened, within 48 hours.

Figure 10

March 18th 2005

I called the patient to check up on her. She told me that the swelling had receded and that she felt a little better.

Fourth visit: April 13th 2005

The patient came to the given appointment. The phoenix abscess related to tooth 16 was gone. She was unable to go through with the treatment session due to pain from head and neck. Her physician had requested a CT of the head and neck, but she did not want the radiographic examination. I referred her to a physiotherapist.

Fifth visit: April 20th 2005

She had seen the physiotherapist the day before. In addition to physiotherapy she was to perform a few exercises every day to help her relax. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The temporary restoration was removed and the canals checked for presence of $Ca(OH)_2$ with a K-file 35. Search for the MB2 with round bur #10 and microscope was negative. The file placed in the MB canal at the disto-radial WL-radiograph also seemed centred in the root (figure 11). Canals were fully instrumented with apical box-preparation to 45/21mm MB reference point MBC, 45/20mm DB ref DBC and 60/21mm P ref PC. Irrigation was done with NaOCl and EDTA. $Ca(OH)_2$ was packed with Lentulo spiral filler and sterile paperpoints as inter-appointment dressing. The access cavity was washed with CHX and dried with cotton-pellets and paperpoints. Canal entrances were covered with Cavit-G and the tooth temporized with IRM. The patient was given instructions in oral hygiene of the abutment teeth in the prosthetic bridge.

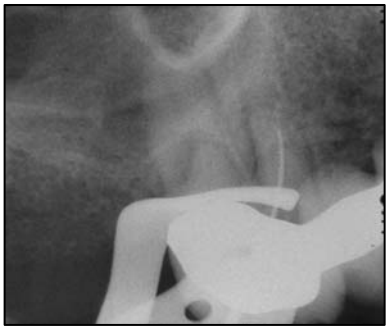


Figure 11

Sixth visit. June 15th 2005
(three months after initiation of treatment)

The tooth was asymptomatic. PPD distally was approximately 9mm. All pockets were bleeding on probing. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). Irrigation was NaOCl and EDTA. The canals were dry. A masterpoint radiograph was taken (figure 12). The canals were filled with 02-taper mastercones: 45/21mm MB, 45/20mm DB and 60/21mm P, and accessory points B using the cold lateral condensation technique. Sealer was AH Plus. The guttapercha was severed with a hot instrument 2mm apical to the pulp chamber floor for each canal. The access-cavity was rinsed with CHX and dried with cotton pellets and paperpoints. IRM was placed over the canal-entrances and moistened with water to induce setting. After 5 minutes the walls of the pulp chamber were rubbed with a diamond-bur and acid-etched for 15 seconds. Syntac-sprint® bonding and Z-250® A3 composite placed. Rubberdam was removed, occlusion checked and a post-treatment radiograph taken (figure 13).

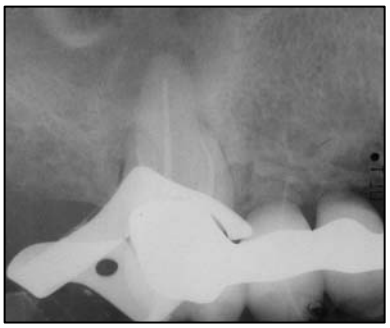


Figure 12

Figure 13

Prognosis

The endodontic prognosis was assumed to be good. The periodontal prognosis was deemed uncertain since the endodontic treatment did not seem to decrease the PPD. The periodontal disease was active despite periodontal treatment at the Student Clinic.

Follow-up

5 months post-treatment, November 9th 2005

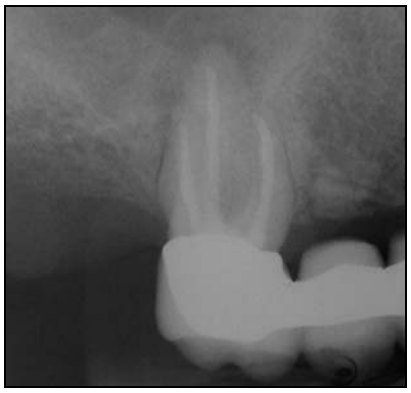


Figure 14

Figure 15

The patient returned with pain from the area of the upper right canine to the upper right first molar. There was some vague swelling and tenderness to touch extraorally. Tooth 13 was still positive to Endo-ice®. A probe was inserted 10mm into the palatal and distal periodontal pockets and pus was noted. One ortho- and one mesioradial radiograph was taken (figure 14 and 15). A periodontist was consulted and the endodontic/periodontic treatment was considered a failure. An extraction was proposed to the patient. When she realised that it would imply loss of the second, and maybe the first, premolar by splitting the bridge, she did not want to go through with the suggested treatment.

The patient has been back for treatment of other teeth since. But as the situation again has calmed down she insists on keeping the posterior abutment for her bridge.

Discussion

Extruded sealer (figure 15) in the curvature of MB root may be a strip perforation, or extrusion into the not found MB2. This angulation clearly shows that the periodontal-endodontal rarefaction also involves the mesiobuccal root. This may have been the case throughout the treatment or a change to the worse afterwards. Not localizing and debriding the mb2 canal is sure to have an impact on the prognosis. Surgery, either periodontic- or endodontic, was not considered an option here. Opinions differ on the subject. Comparing apical surgery on teeth with periapical lesions of endodontic origin (1) with apical surgery on those with a periodontal involvement measured as loss of (buccal) cortical plate (2), the gap in treatment outcome is massive. Zuolo et al (1) reported success exceeding 90% after 1-4 years on teeth with no periodontal bone loss. Skoglund and Persson reported a success rate of 37% after ½ -7 years when the tooth had advanced marginal bone loss.

References:

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Case 8

One-step treatment of a non-vital tooth

Chronic apical periodontitis of the lower left second premolar

Patient

A seventyfive year old white Norwegian female (figure 1) was referred from the Student Clinic to the Postgraduate Endodontic Clinic at the Faculty of Dentistry, University of Oslo August 2005 for treatment of the lower left second premolar (tooth 35).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

The lower left second premolar was monitored with sensitivity-testing and X-ray after a radiographic widening of the PDL-space was found in June 2005 when evaluating a periodontal defect of tooth 36. There were no subjective symptoms related to tooth 35. The tooth was mobile grade I. Occlusion was adjusted. The lower left first molar (tooth 36) was tender to percussion and palpation, 35 was not. Endo-Ice® gave late response both from 35 and 36 when compared to contralateral teeth.

Clinical examination

September 2005. Tooth 35 had a prosthetic crown. There was no contact-point between teeth 35 and 36. Tooth 36 has an MOD composite restoration. Pocket probing depth (PPD) of the second premolar was within normal limits (WNL), but for the first molar PPD was 10mm mesially. 35 did not respond to thermal testing with Endo-Ice®. Tooth 36 was still tender to palpation and percussion after scaling and rootplaning at the Student Clinic two months earlier. Tooth 35 was asymptomatic but mobile grade I.

Radiographic examination

Figure 2

The radiograph (figure 2) shows the region from the lower left second premolar to the lower left first molar. Tooth 35 had a coronal restoration in line with a metal-ceramic crown. The pulp space was vaguely visible in the root. The PDL was traceable down the mesial and distal aspect of the root. In the apical area the the PDL was widened and the lamina dura was not seen. Mesially the marginal bone loss is estimated to 3 mm; distally 4 mm. Tooth 36 had an MOD radioopaque restoration resembling a composite. At the mesial aspect of the mesial root a wide radiolusent zone was seen from the cervical area to the apex indicating total loss of alveolar bone save a triangular area mid-root. This area had radiodensity comparable to bone or calculus. The PDL-space was visible from the periapical area of the mesial root to the furcation area. In the furcation area a radiolucency of 2 x 4 mm was seen. The PDL-space is discernible around the distal root. The pulp-chamber is not visible and in the roots the pulp spaces were vaguely visible. At the distal aspect of the tooth the marginal bone loss was around 5mm.

Diagnosis

Tooth 35
Chronic apical periodontitis

Tooth 36
Chronic marginal periodontitis
Chronic apical periodontitis

Difficulty assessment:
36: Obliteration of root-canals. Probable established endodontic-periodontic problem
35: Access through a prosthetic crown

Treatment plan

Treatment of necrotic pulp tooth 35
Treatment of necrotic pulp tooth 36, explorative surgery with scaling and rootplaning
Apical surgery if adequate

The patient was informed about the limitations of the endodontic treatment of tooth 36 regarding re-establishment of marginal bone. When explained of the uncertainty and difficulty related to treatment of tooth 36 she decided to abstain from treatment of that tooth at the present time.

Treatment

First visit: March 3rd 2005

An access-cavity was prepared without local anaesthetic, using round diamond-coated burs for the ceramics and hard-metal fissure burs in a high speed handpiece, until the pulp-chamber was localized. A long-shank round bur # 14 was used to clean the pulpchamber and the single, centrally located canal was found. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). A K-file # 10 was inserted into the canal, and the patient gave a pain-response at approximately 12mm. 0, 5 carpule of Xylocain-Adrenalin® was given as an intrapulpal injection.

Figure 3

A working-length (WL) radiograph was exposed (figure 3) with K-file15/18,5mm reference point BC. The canal was instrumented with handfiles to K 55/19mm and irrigated with 1% sodium hypochlorite (NaOCl) and EDTA 15% and dried with paperpoints. The canal was dry. A five minutes inlay of 2 % chlorhexinine digluconate (CHX-2-G) was placed and the canal again dried with paperpoints. A masterpoint radiograph was taken (figure 4).

Figure 4

The rootfilling was placed with #55 02-taper mastercone and accessory points B in a cold compaction technique.
Sealer was AH Plus. The guttapercha was severed with a hot instrument 2mm apical to the pulp chamber. The access-cavity was rinsed with CHX and dried with paperpoints. IRM was placed over the canal-entrance and moistened with water to induce setting. After 5 minutes the walls of the pulp chamber were rubbed with a diamond-bur and acid-etched for 15 seconds. Syntac-sprint® bonding was applied and a Z-250® A3 composite placed. Rubberdam was removed, occlusion checked and a post-treatment radiograph was taken (figure 5).

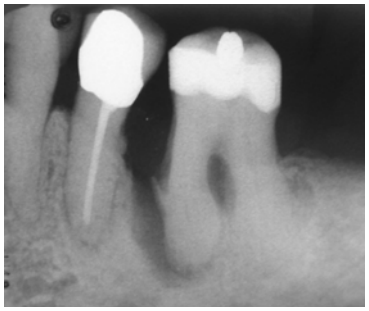

Figure 5

Prognosis

The prognosis was assumed to be good.

Discussion

The aetiology of the problem regarding tooth 36 was unclear. It seemed possible that it had evolved from a poor contact-point between the second premolar and the first molar, probably with many years of food-impaction. The nature of the oblique sclerotic ridge related to the mesial aspect of the mesial root of 36 was uncertain too. Calculus, or a bony ridge, was the obvious alternatives.

During treatment of tooth 35 there was sensitivity in the canal under treatment. This may indicate, as some authors relate, that noxious products from bacteria in the root-canal may induce periapical bone destruction well in advance of total pulp necrosis, with vital pulp still in the apical root canal (1) Or in this case, it may be that the periapical rarefaction was, in fact, a result of occlusal trauma or mobility induced by marginal periodontitis (2). Prognosis of one-step treatment of apical periodontitis has been found to be equal, or superior to, conventional two-step treatment with antibacterial dressing between appointments (3).

What is indisputably important for the outcome of treatment is the presence or absence of bacteria at the time of filling. It was found that the prognosis for treatment of apical periodontitis is 94% when teeth were culture-negative and 64% when teeth were culture-positive at the time of filling (4). From this I draw the conclusion that the road to a culture-negative tooth is irrelevant as long as the goal is achieved.

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Case 9

Treatment of a non-vital tooth

Chronic apical periodontitis of the upper left first premolar

Patient

A fiftytwo year old white female (figure 1) was referred from the Postgraduate Prosthodontic Clinic to the Postgraduate Endodontic clinic, Faculty of Dentistry, University of Oslo, October 2003 for evaluation and treatment of the upper left first premolar (tooth 24).

Figure 1

Medical history

The patient smoked from 5 to 10 cigarettes a day.

Dental history and chief complaint

The patient was under treatment for chronic marginal periodontitis at the Postgraduate Periodontic Clinic, Faculty of Dentistry at the University of Oslo. In the second quadrant the second premolar, the first, second and third molars had been lost. She had been through several dental surgeries, both for marginal periodontitis and for placement of titanium-implants. The patient had cancelled several appointments at the Endodontic Clinic. She presently experienced pain and swelling from the region of tooth 24 and wanted treatment. Her main concern was the mobility of the upper left first premolar. She had consulted the Department of Oral Surgery a few days earlier; they recommended extraction for that tooth, alternatively endodontic treatment as a final resort. She was intent on keeping the tooth, so she made an appointment at the Postgraduate Endodontic Clinic.

Clinical examination

The pictures (figures 2 and 3) show the region from the upper left lateral incisor to the upper left second premolar. The lateral incisor had a metal-ceramic prosthetic crown. Tooth 23 had a composite restoration at the distal and the buccal surface. Tooth 24 had an MOD composite restoration. Tooth 25 had a prosthetic crown. There was some gingival retraction noticeable, and loss of interdental papillae in relation to the prosthetic crown on tooth 22. Tooth 24 was tender to palpation in the vestibulum.

Figure 2

The tooth was also tender to percussion in horizontal and vertical direction. Pocket probing depth of 9mm was found mesiopalatally. Pus was detected from the pocket upon probing. There was no sign of scar tissue or an open sinus tract in the buccal or palatal mucosa. 24 gave no response to electric pulp testing or Endo-Ice®. The tooth was mobile grade II.

Figure 3

Radiographic examination.

The X-ray (figure 4) depicts the region of tooth 23 to 25. Tooth 23 had radiolucent areas corresponding to buccal and distal composite restorations. The lamina dura could be followed around the apical half of the root. The marginal bone loss here was more than ⅓ of the root length. Tooth 24 had an MOD radiolucent zone in line with a composite filling. The marginal bone loss reached midroot at the distal side of the root. At the mesial aspect of the tooth there was a bony pocket of 2-3 mm also encircling the periapical area. No PDL-space could be seen at the mesial side of the root. At the distal side the PDL-space was visible for a few millimetres between the distinct radiolucency of the bony pocket and the periapical radiolucency. Tooth 25 had been replaced with an implant.

Figure 4

Diagnosis

Upper left first premolar:
Chronic marginal periodontitis
Apical periodontitis with sinus tract
Endo-perio lesion

The patient was informed about the limitations of the endodontic treatment in that it was not expected to cure the apicomarginal defect. The patient still wanted to go through with the root canal treatment.

Treatment plan

Treatment of necrotic pulp tooth 24

Treatment

First appointment, November 11th 2002

One carpule of Xylocaine-Adrenaline® was used to establish anaesthesia. An access-cavity was prepared using a round diamond-coated bur for the ceramics and a hard-metal fissure bur in a high speed handpiece until the pulp-chamber was localized. A long-shank round bur # 12 in a slow speed handpiece was used to clean the pulp chamber and buccal and palatal necrotic canals were localized. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). A K-file # 10 was inserted in the canals and negotiated until the apexlocator Root ZX® indicated being in the PDL. One millimetre was subtracted from that length and a working-length (WL) radiograph was exposed (figure 5) with the following measures: B K-file15/20mm ref BC, P H-file 20/20mm ref PC. The canals were further instrumented with Protaper S_1, S_2, F_1 and F_2 and K-file #35. Canals were irrigated with 1% sodium hypochlorite (NaOCl) and EDTA 15% and dried with paperpoints. Exudate was registered from both canals. $Ca(OH)_2$ was inserted with #35 K-file using counter-clockwise rotation. The $Ca(OH)_2$ was then packed with paperpoints and Cavit-G was placed over the canal entrances and IRM in the access-cavity as a temporary filling.

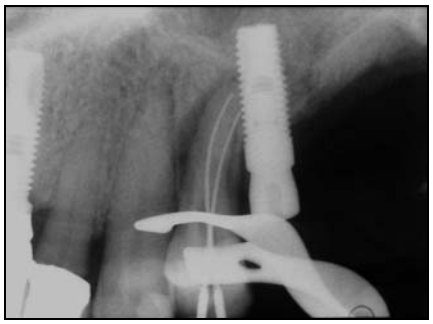
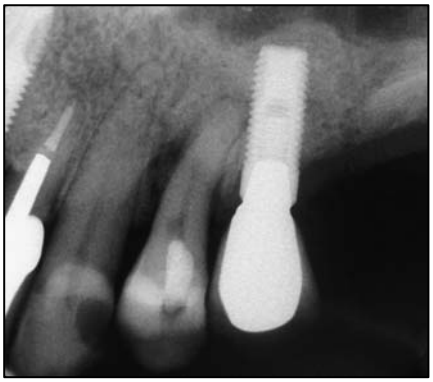


Figure 5

The patient did not return for the scheduled appointment. I was unable to get in touch with her by phone and she did not reply to mail.

Second visit, May 3rd 2005
(29 months after initiation of treatment)



The patient had no symptoms from 24, but it was still mobile grade II. An X-ray was taken (figure 6). Rubberdam was applied and disinfected with CHX. The temporary filling was removed. The canals were instrumented to 40/20mm B, 40/21mm P. Irrigation was with 1% sodium hypochlorite (NaOCl) and EDTA 15%. The canals were dried with paperpoints. $Ca(OH)_2$-slurry was placed with a lentulo-spiral filler and Cavit-G was placed as a temporary filling.

Figure 6

Third visit, May 24th 2005
(three weeks later)

The tooth was asymptomatic. Rubberdam was applied and disinfected with CHX. The canals were irrigated with NaOCl and EDTA and dried with paperpoints. The canals were dry. A mastercone radiograph was taken (figure 7). The canals were filled with Resilon® 02-taper mastercones: 40/20mm B, 40/21mm P and accessory points MF and M using the cold lateral condensation technique. Sealer was Epiphany®. The rootfilling was severed with a hot instrument 2mm apical to the pulp chamber floor in each canal. Light-curing: 40 seconds. The walls of the pulp chamber were rubbed with a diamond-bur and acid-etched for 15 seconds. Syntac-sprint® bonding and Z-250® A3 composite was placed. The rubberdam was removed, occlusion checked and a post-treatment radiograph taken (figure 8).

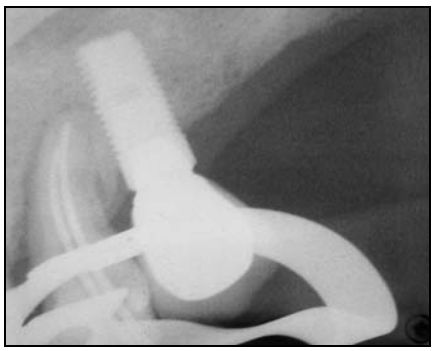


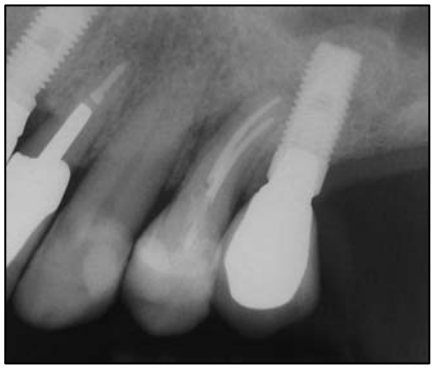
Figure 7

Figure 8

Prognosis

The prognosis for the endodontic treatment was assumed to be good. The periodontal prognosis was uncertain.

Follow-up



January 10th 2006. The patient was back 10 months after endodontic treatment was completed. An X-ray was taken (figure 9). The patient was asymptomatic. No sinus tract was observed in the mucosa. The mesiopalatal pocket was still 8mm with bleeding on probing, but no pus.

Figure 9

Discussion

Figure 10 Pretreatment 2002

Figure 11

The endodontic treatment seems to have been successful as the periapical lesion appears healed (figure 9). The pretreatment rasiograph is shown for comparison (figure 10). In the projection where both mastercones are visible (figure 9), the implant disturbs the evaluation of the periapical area. The overall prognosis of the tooth is still uncertain as the marginal periodontitis does not seem to respond to treatment.

The close proximity of the implant to the periapical area of tooth 24 gives rise to speculation of the aetiology of the periapical disease. The placement of the implant may have hampered the bloodsupply of the neighbouring premolar and subsequently induced an apical periodontitis. Survival time for single tooth implants has been investigated (1, 2), but fewer studies dedicate to the structures adjacent to the implants (3, 4). They found that teeth adjacent to single-tooth implants show an extremely low complication rate. In a 10-year follow-up of 196 adjacent teeth only 2 needed endodontic treatment (4). But one implant was considered a failure as an adjacent root was contacted. It should be noted that in these studies the adjacent teeth were intact or almost intact prior to treatment.In another 3-year survey (3) none of the teeth adjacent to 78 implants needed endodontic treatment. In this study one of the inclusion criteria was that the direction of which the implants were placed did not cause any harm to the roots of neighbouring teeth.

A radiograph of the contra-lateral side (figure 11) shows that the endodontic complication of tooth 24 may, indeed, be completely independent of the implant (figure 9). Dietrich et al (5) sub-classifies apicomarginal defects into class I perio-endo-lesion, class II periapical lesion, class III dehiscence and class IV miscellaneous. Class I perio-

endo-lesion is further subdivided into class I/1 periodontal, class I/2 combined and I/3 endodontal. He also states that the discrete subdivision of the Class I lesions is a difficult task not always possible. Whether or not the patient has marginal periodontitis is a key factor in this respect. Periodontal breakdown from periodontal disease usually starts interdentally, and intra-bony periodontal defects are mainly located proximally. Therefore, a Class I apicomarginal defect confined to the buccal aspect of the root strongly suggests an endodontic origin. In all other cases with periodontitis, periodontal disease is likely to have contributed to the pathogenesis of the defect.

The severity of periodontal disease as assessed by the attachment loss and the pattern of bone loss in the remaining dentition can suggest to what extent it has contributed. In this case it seems plausible that the marginal periodontitis bears the main blame for the defect. Successful endodontic treatment is expected to revert only the boneloss caused by the periapical infection. Treamtent of the periodontal disease will destroy the attachment apparatus. Successful periodontal treatment and disappearance of the bony pocket will at best create a situation with long junctional epithelium but no real attachment regeneration. Dietrich et al also stated that further studies are necessary to show how these variables affect treatment outcome.

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Case 10

Re-treatment of a lower right first molar with a broken instrument

Patient

A nineteen year old white Norwegian female (figure 1) was referred from her general dental practitioner (GDP) to the Postgraduate Endodontic Clinic, Faculty of Dentistry at the University of Oslo September 2005 for treatment of the mandibular right first molar (tooth 46).

Figure 1

Medical history

The medical history was non-contributory.

Dental history and chief complaint

The patient had contacted her GDP for pain in the lower right molar-area the first week of September 2005. The tooth was previously rootfilled. Re-treatment was commenced. A NiTi-file had fractured in the mesio-buccal canal. The patient was informed and a referral made. She still had spontaneous pain from tooth 46 and was worried about the fractured file.

Clinical examination

Figure 2

Figure 3

Figure 4

The photos (figures 2, 3 and 4) show the region from the lower right second premolar (tooth 45) to the lower right second molar (tooth 47). There was some plaque-accumulation in the gingival area of all teeth. Tooth 45 was intact. Tooth 46 had mesial and distal composite restorations and an occlusal temporary restoration. Tooth 47 had a class I composite filling. Tooth 45 and 47 tested positive to Endo-ice®. Tooth 46 gave no response to Endo-ice® and was tender to percussion and palpation in the vestibulum. Pocket probing depth (PPD) was within normal limits for all teeth.

Radiographic examination

Figure 5

The radiograph (figure 5) shows the region from 45 to 47. Tooth 45 was intact, and the lamina dura could be traced all the way around the root. Tooth 47 had an occlusal radioopaque area representing a composite. In the projection of this X-ray the restoration seemed to be in close proximity to the pulp. The lamina dura could be followed all the way around the two gathered roots. Tooth 46 had an MOD restoration including the area of the pulp chamber with the radiographic characteristics of composite and temporary filling-material. Centrally in the roots a material with radiodensity in line with guttapercha was observed. In the mesial root the radiodensity was more pronounced in the coronal two thirds. The lamina dura tapered into a radiolucent zone approximately 4mm in diameter encircling the mesial root tip. At the distal root a marked widening of the PDL-space was visible. The marginal bone level was within normal limits.

Diagnosis

Chronic apical periodontitis of the lower right first molar

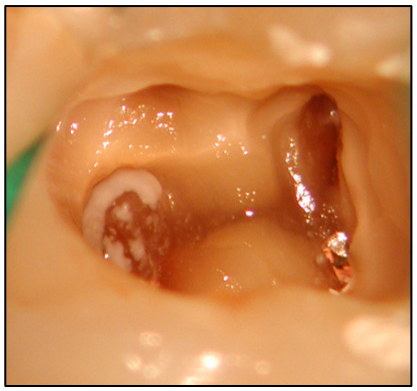
Difficulty assessment: Broken instrument in mesio-buccal root canal

Treatment plan

Retreatment of tooth 46

Treatment

First visit, October 5th 2005



Access cavity was prepared with steel fissure burs in a high-speed handpiece and long shank round burs # 16, 12 and 10 in a slow-speed handpiece. MB, ML and D canal orifices were localised. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The broken instrument was visible in the mesio-buccal canal-entrance (figure 6).The gutta-percha was removed from the mesio-lingual and distal canals with minute amounts of chloroform, pre-RaCe-instruments and Hedstrom-files # 50, 30 and 20.

Figure 6

Figure 7

Figure 8

A working-length (WL) radiograph was exposed with K-file 30/21mm D and K-file 15/22mm ML (figure 7). The fractured instrument came loose with use of a piezoelectric ultrasound with a K-file-25 tip and a few drops of chloroform to dissolve the guttapercha in which the instrument was lodged. An additional WL-radiograph was exposed with a K-file # 15/22mm MB (figure 8). The fractured file was a nine-millimetre Hedstrom-file (figure 9). The distal canal was oval and was flared in buccolingual direction. The canals were instrumented with apical box-preparation to sizes 45/22mm MB, 45/22mm ML and 55/21mm D. Irrigation was done with copious amounts of 1% NaOCl and EDTA 15%. The canals were dried with sterile paperpoints. $Ca(OH)_2$-slurry was inserted with a lentulo spiral filler, and the tooth temporized with Cavit-G and IRM.

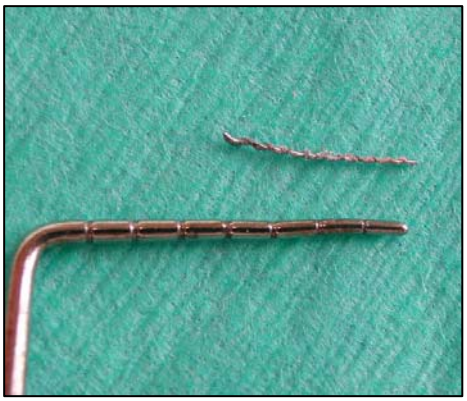


Figure 9

Second visit, October 26th 2005
(3 weeks later)

The tooth was asymptomatic. Rubberdam was applied and disinfected as described earlier. The $Ca(OH)_2$ was washed out with the aid of NaOCl and EDTA. The canals were dry. A masterpoint radiograph was taken (figure 10). The tooth was filled with 02-taper guttapercha 45/22mm MB, 45/22mm ML and 55/21mm D using cold lateral condensation technique with accessory-points B and sealer AH Plus. The gp was severed with a warm instrument 2mm into the canal orifices. The access cavity was rinsed with CHX and thoroughly dried with cotton pellets and paper-points. IRM was placed into the canal-entrances and in the access cavity. A post-treatment radiograph was exposed (figure11).

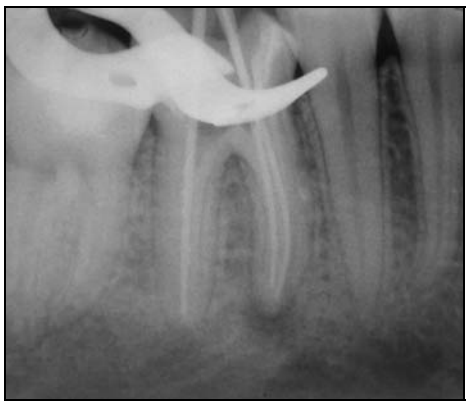


Figure 10

Figure 11

Prognosis

The prognosis was assumed to be good.

Follow-up

When this went in print the patient had not been able to return for a recall.

Discussion

The widening of the PDL registered in the pre-treatment radiograph became apparent in the post-treatment radiograph. Having followed strict aseptic techniques it was assumed that this was because of different angulations of the radiographs. The quality of the post-treatment radiograph was not optimal, and the root-filling seemed somewhat inhomogenous in the mesial canals. No voids could be detected and it was decided to leave the rootfilling as it was.

When an endodontic instrument fractures during use in a root canal, the best option is to remove it, if possible. Only after removal can the root canal be negotiated, cleaned and shaped properly. If the root canal cannot be cleaned and shaped successfully, remnants of pulp tissue and bacteria may remain and compromise the outcome of root canal treatment (1). However, attempts to remove fractured instruments may lead to ledge formation, over-enlargement and transportation of the prepared root canal, or root perforation. As a clinician I had to evaluate the options of attempting to remove the

instrument, bypassing it or leaving the fractured portion in the root canal. This decision was made with the consideration of the pulp status, canal infection, the canal anatomy, the position of the instrument and the type of fractured instrument.
There are several methods for removing fractured instruments. Ultrasound is often used, as in this case. In short, the steps are:
A. Creation of straight line access to the coronal end of the instrument under microscope (Gates-Glidden burs or other alternatives). B. With aid of a microscope create a groove around the coronal end of the instrument using ultrasound (size 25 K-file tip or other alternatives). C. Ultrasonic file is kept in contact with, or best bypass, the fractured instrument until movement registered. D. Loosened instrument removed by flushing with irrigation or Hedstrom-file, pliers or Masseran-kit.
This does not always work. Suter et al have evaluated the possibilities of success (2). There were no significant differences in the success rate according to the type of root the instrument was removed from (incisor, premolar, molar). The position of the fractured instrument did not seem to make a difference unless lodged beyond the apical foramen:

Instrument in coronal third of rootcanal 17 of 19 removed (89, 5 %).
Instrument in middle third of rootcanal 27 of 31 removed (87, 1 %).
Instrument in apical third of rootcanal 35 of 40 removed (87, 5 %).
Instrument beyond the apical foramen 0 of 2 removed (0 %).

There was a significant correlation between the amount of time needed to remove the instrument and the corresponding reduction in the success rate. The criteria for success were complete removal of the instrument without root perforation. What was not evaluated was the curve of the root where the files came out compared to those where the instrument did not. In clinical practise the apically placed fractured instruments in curved canals are those where establishment of straight access to, and a groove around, the coronal part of the instrument may proove impossible without perforating. It was found that NiTi instruments, lentulo-needles and SS-files were prone to fracture when using ultrasound. This happened in 30 % of the cases, and more often with NiTi files. The over-all success rate of removing fractured instrument was 78 %. This was an operator-derived success-rate and does not necessarily apply to others.

In certain clinical situations it may also be better to leave a fractured instrument in the root canal, for example when the instrument fractures in a canal with a vital pulp, or if it fractures when removing a calcium hydroxide dressing. This is based on the assumption that it is the infection and not the instrument in itself that creates the problem. Studies on treatment outcome indicate that the fractured instrument has an impact on prognosis only in cases with apical periodontitis (1, 3).

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Case 11

Treatment of a non-vital tooth

Chronic interradicular periodontitis of the upper left first molar

Patient

A fiftytwo year old Caucasian female (figure 1) was referred from the Student Clinic to the Postgaduate Endodontic Clinic, Faculty of Dentistry, University of Oslo December 2002 for endodontic treatment of the upper left first molar (tooth 26).

Figure 1

Medical history

Her medical history was non-contributory.

Dental history and chief complaint

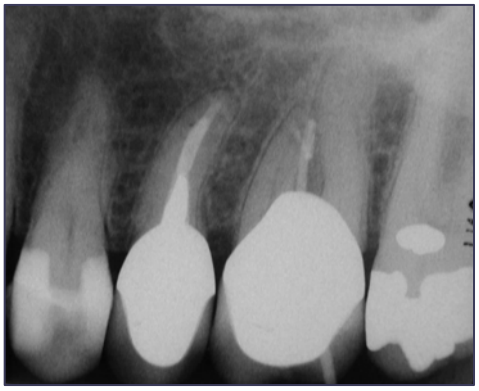

Figure 2

The patient sought help at the Clinic of Diagnostics, Faculty of Dentistry at the University of Oslo November 19th 2002 due to pain from the upper left molar-area. Clinical findings were persistent pain to cold stimulus on tooth 26. No periapical radiolucency was observed on X-ray. A prescription of Apocillin 660mg No: 30 was made. Treatment was started the next day with access cavity-preparation. Eugenol-pellets and IRM were placed. The patient was scheduled for endodontic treatment at the Student Clinic. December 2nd 2002; Clinical investigation of tooth 26 revealed a periodontal pocket of 12mm buccally. A radiograph was exposed with guttapercha in the bony defect (figure 2). One carpule of Xylocain-Adrenalin® was used to establish anaesthesia. The temporary filling was removed and canals localised. A perforation in the furcation area was observed. IRM was placed over the perforation and in the access cavity and the patient was referred to the Postgraduate Endodontic Clinic.

Clinical examination

The pictures (figures 3, 4 and 5) show region 24-27.
Tooth 24 had an MOD tooth-coloured restoration.
Tooth 25 had a prosthetic crown.
Tooth 26 had a prosthetic crown, occlusally interrupted by a temporary filling material.
Tooth 27 had MO and P amalgam restorations.
Tooth 26 was tender to percussion.
Pocket probing dept was within normal limits and the deep 12mm buccal pocket diagnosed at the Student clinic could not be found.
Tooth 24 and 27 were positive to EPT.
There was no response to cold from tooth 26.

Figure 3

Figure 4

Figure 5

Radiographic examination

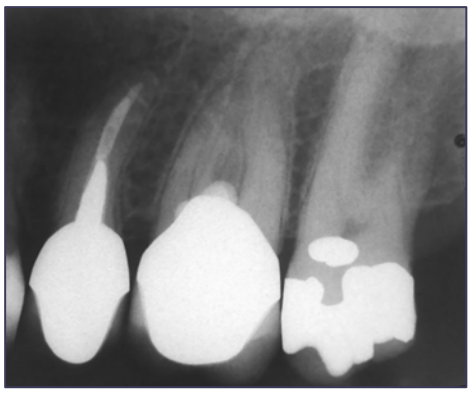

Figure 6

The radiograph (figure 6) shows region 25-27.
Tooth 25 had a densely radioopaque restoration coronally representing a metal-ceramic crown and metallic post. Further apically a less radio-opaque material representing guttapercha was seen. The PDL-space could be traced all the way around the root. Tooth 26 had a radiodense restoration coronally being a prosthetic crown. In the furcation area a less radiodense material was seen that may represent temporary filling-material in the perforation. The PDL-space could be followed around the apices of the three radiographically gathered roots. Tooth 27 had an MOD and an oval of radiodense filling material projected over the pulpchamber, in line with amalgam. Interpretation of the PDL-space was difficult. The marginal bone-level was within normal limits.

Diagnosis

Necrotic pulp of tooth 26
Perforation in the trifurcation

Treatment plan

Closure of the perforation
Treatment of necrotic pulp tooth 26

Treatment

First visit, March 12th 2003

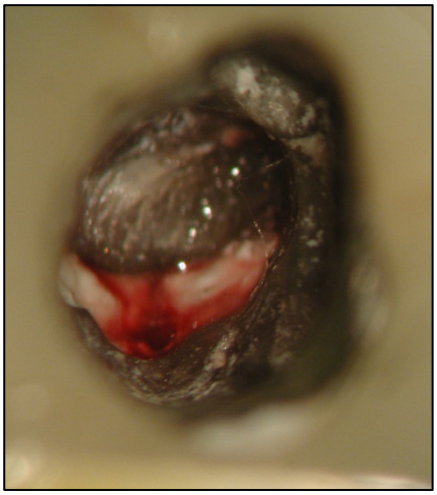

Figure 7 Perforation 1,5 x 4mm

The temporary filling was removed. A perforation was visible in the floor of the pulp chamber. It extended from the buccal axial wall towards the palatal canal. The size of the perforation was estimated to 1.5 x 4 mm (figure 7). Four canals were localized, the distobuccal, mesiobuccal and mesiopalatal ones with some difficulty. After having smoothed the access cavity they were found not in the floor, but on the walls of the pulp chamber, indicating a too aggressive search as the perforation proved (figures 8 and 9). Rubberdam was applied and disinfected with 5 mg/ml chlorhexidin in 70 % ethanol. There was bleeding from the mb, mp and p canals and probing them was painful to the patient.

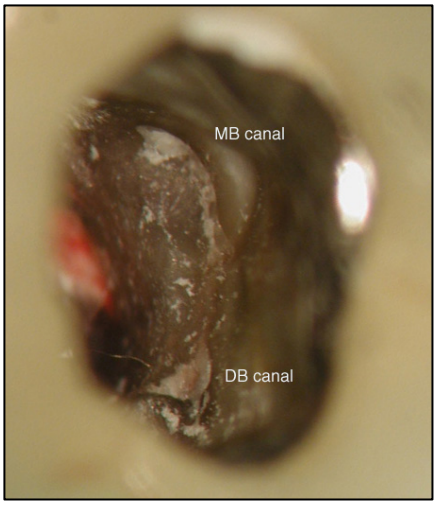

Figure 8 Buccal canals

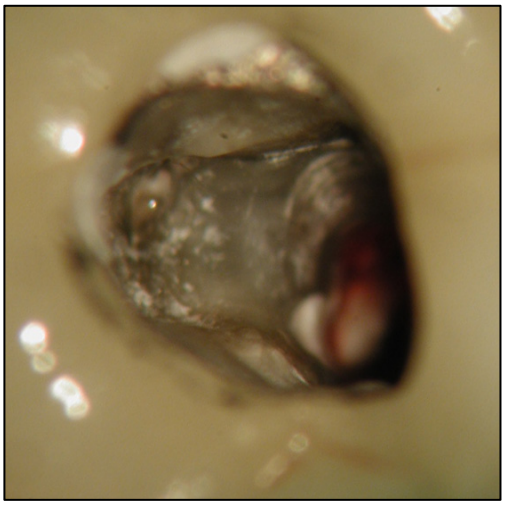

Figure 9 Palatal canal

One carpule of Xylocain-Adrenalin® was placed to establish anaesthesia and the disinfection repeated. The part of the IRM® that extended into the interradicular area was removed with the aid of an endodontic probe. $Ca(OH)_2$ was placed in the perforation to control bleeding. The mesiopalatal canal was protected with an accessory gutta-percha-point B. The mb, db and p canals were covered with Cavit-G®.

MTA® was placed and covered with a moist cotton-pellet. X-rays were exposed to view the extent of the MTA (figure 10, 11 and 12). The tooth was temporized with Cavit-G.

Figure 10 Caudal projection

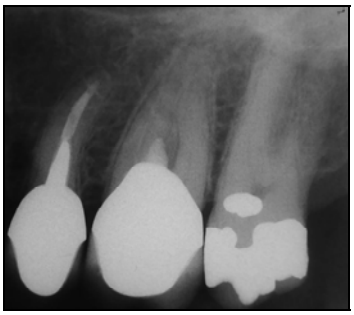

Figure 11 Cranial projection

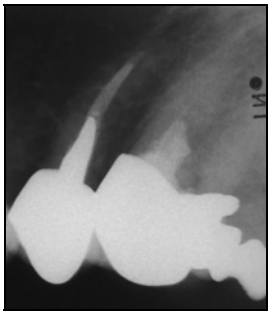

Figure 12 Disto-radial-

Second visit, March 18th 2003

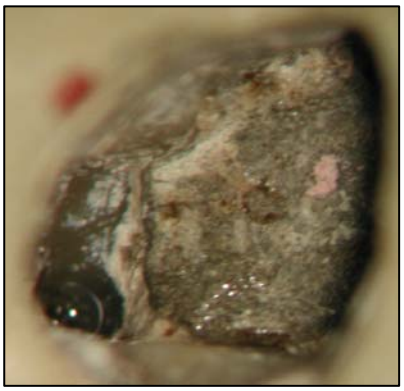

Figure 13

One carpule X/A was administered. Rubberdam was applied and disinfected with chlorhexidin-ethanol solution. The temporary filling and cotton pellet were removed and the MTA found to have set (figure 13). With the aid of a Root ZX® apexlocator a working lenght radiograph was taken: Mb K-15/19mm reference point mesiobuccal cusp, mp H-15/18mm ref MBC, db K-15/19mm ref DBC, p H-25/18.5mm ref PC (figure 14). The apexlocator was applied again to a K-file in the palatal canal until it indicated being in the PDL. One millimetre was subtracted from that length and a new radiograph was exposed at 19.5mm (figure 15). Apical box-preparations were done with hand-files: 45/19mb, 45/18mp, 45/19db, 60/19.5p. Irrigation was by means of NaOCl and EDTA. $Ca(OH)_2$ was placed as inter-appointment dressing with a lentulo-spiral and covered with IRM as temporary filling.

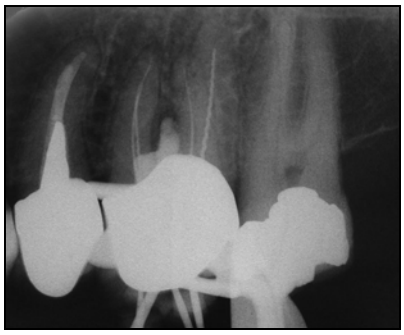

Figure 14

Figure 15

Third visit, April 22nd 2003

The tooth was asymptomatic. One carpule X/A® was administered to accommodate the patient's wishes. Rubberdam was applied and disinfected as previously described. The dressing was removed with NaOCl and EDTA. The canals were dry. A masterpoint radiograph was taken (figure 16). MB and MP canals fused in the apical 2-3 mm; the master-cones could not both pass to working length at the same time. The canals were filled using 02-taper guttapercha cones 45/19 mm MB, 45/18 MP 45/19 DB, 60/19.5 P using cold lateral condensation. Accessory points B and C were placed. Sealer was AH Plus®. IRM was placed as topfilling. A post-treatment radiograph was exposed (figure 17).

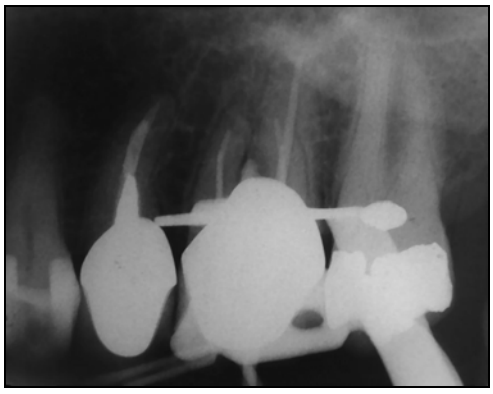

Figure 16

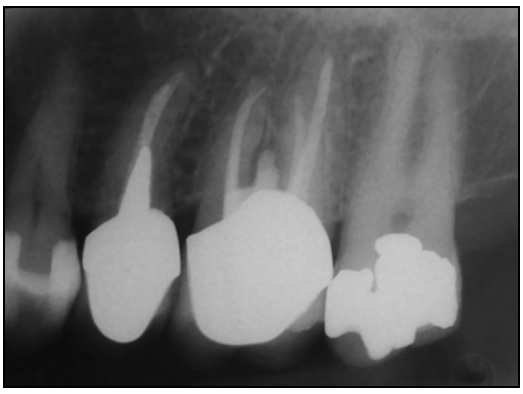

Figure 17

Fourth visit, May 13th 2003

Rubberdam was applied and disinfected as previously described. The IRM was removed save over the canal orifices. A composite Filtec Z-250® was placed in the access-cavity after acid-etching and Syntac Sprint® one-step bonding.

Prognosis

The prognosis was assumed to be good.

Follow-up

Figure 18 One-year follow-up May 2004

At the one year follow-up the patient was asymptomatic. The interradicular lesion show signs of healing on X-ray (figure 18).

At the two-year follow-up the situation for the patient was the same, no symptoms or problems from the area of tooth 26 (figures 19 and 20). The radiograph taken (figure 22) seemed to confirm the impression that the interradicular radiolucency observed at the post-treatment radiograph (figure 21) was healed.

Figure 19 Two years follow-up May 2005

Figure 20

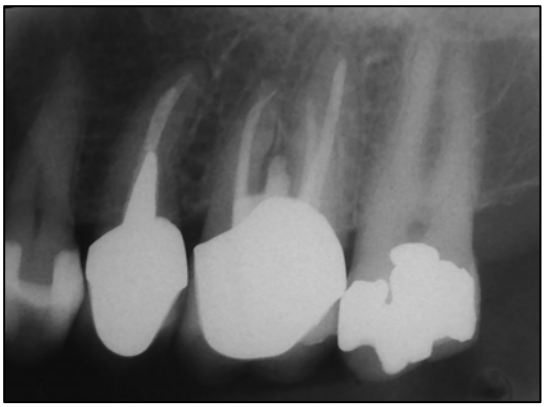

Figure 21 Post treatment April 2003

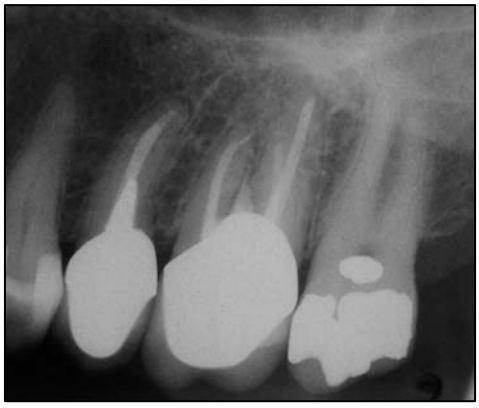

Figure 22 Two years follow up May 2005

Discussion

In this case it seems that the prescription of antibiotics was erroneous. With clinical findings indicative of symptomatic pulpitis there is nothing to gain from antibiotics. Currently, endodontic debridement (pulpectomy or pulpotomy) is the most predictable method to relieve the pain of irreversible pulpitis (1) The biological reason why pulpotomies and pulpectomies reduce pain is based on reducing tissue levels of inflammatory mediators and the elevated interstitial tissue pressure that stimulate peripheral terminals of nociceptors.

However, antibiotics are often prescribed indiscriminately to treat endodontic emergencies (2). A prospective, randomized, double-blind, placebo-controlled study has been conducted to determine the effect of penicillin on pain in untreated teeth, diagnosed with moderately to severely painful irreversible pulpitis (3). The outcome variables were the differences in spontaneous pain and percussion pain between the

penicillin and the placebo groups after 7 days, as well as the amount of analgesic medications used by both groups during this period. Importantly, this study revealed no statistically significant results between the two groups on any of the parameters evaluated, indicating, quite convincingly, that antibiotic use does not relieve pain due to irreversible pulpitis. In other words, antibiotic usage produced the same responses as that seen in patients given an inert placebo tablet.

Knowledge of what eases pain in pulpitis-cases should have made the dentist perform access-cavity-preparation with eugenol supplemented with analgesics. The first objective is to maximize the dose of the non-narcotic (e.g. NSAID or acetaminophen) before prescribing an analgesic containing a narcotic. The rationale is that a maximally effective dose of a non-narcotic generally provides greater analgesia and fewer side-effects than a combination drug containing both a non-narcotic analgesic with an opioid (4).

For restoration of the perforation a choice of MTA was done due to its biocompatibility. MTA powder consists of fine hydrophilic particles. The principle compounds are tricalcium silicate, tricalcium aluminate, tricalcium oxide, and silicate oxide. Minute amounts of other mineral oxides are also present. Bismuth oxide is present for its radioopaque properties. Tests show that calcium and phosphorous are the main ions present in the material. Presence of oxides helps explain the high pH of MTA. Hydration of MTA leads to a colloidal gel that solidifies to a hard structure within 3 hours (5).

Regarding the deep buccal periodontal pocket found at the Student Clinic that was not present at the clinical examination at the Postgaduate Endodontic Clinic: It is assumed that the furcal perforation was done at the patient's very first sitting, and that the treatment given at the Student Clinic was successful in healing the iatrogenic periodontal defect. Still it is probable that apical migration of the epithelial attachment to the perforation site was already established and not reversible. The furcal perforation was large. The three variables: Old, large perforation in the crestal area combined leads to a poor prognosis for the treatment (5). Loss of tooth structure also leads to weakening of the tooth (6). Although MTA offers a high degree of compatibility to the periodontal ligament (7), future development of a furcation-involvement may cause problems as the MTA will have constant contact with the oral flora and the MTA will be continuously contaminated. The development of subgingival plaque could be promoted due to the rough surface of MTA (8). As MTA is not a hard material (5), it can be partially scraped off during mechanical cleaning of the root surface and stable periodontal healing might not be expected (9).

Overfilling of MTA, as here; may be prevented by the modified matric concept (10) where a resorbable collagen.matrix is placed in the area of granulation tissue extra-radicularly. The MTA is subsequently placed against the matrix. The most important factor to determine the likelihood of success of treatment of perforations is the time elapsed since occurrence of the perforations, or, more exactly, the question whether the wound site is already infected or not (5).

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Case 12

Treatment of a non-vital tooth with multiple perforations

Chronic apical periodontitis of the upper left first molar

Patient

A sixtyeight year old white female (figure 1) was referred from the Student Clinic to the Graduate Endodontic Clinic, Faculty of Dentistry at the University of Oslo, October 2003, for evaluation of the upper left first molar (26).

Figure 1

Medical history

Her cholesterol was high, for that she took one tablet of Lipitor® a day. Calcigran Forte® and Didronate® 400mg/day was taken to slow down osteoporosis. Active ingredients in Calcigran Forte® are Calcium-carbonate and vitamine D_3. Didronate contains di-sodium etidron which is a bis-phosponate and an analogue to the body's pyro-phosphate. Arthrotec® is a drug with anti-inflammatory effect; two tablets a day were taken for rheumatic pain. The patient was a type II-diabetic and her condition was treated with regulations in dietary intake only. She had an $HbA_1c < 8$ mmol/L indicating a well-controlled diabetic situation. It should therefore not be necessary to take extra precautions before, during or after treatment regarding infection-control and prevention.

Dental history and chief complaint

Tooth 26 was root filled at the student clinic in 2001. The tooth had, since then, been painful from time to time. She had sought counsel with a general dental practitioner (GDP). The tooth was diagnosed with a symptomatic apical periodontitis and the tooth had been retreated February 2003, without rubberdam. When she signed up for dental treatment at the student clinic, she was referred to the Postgraduate Endodontic Clinic due to periodic headache and pain from the upper left quadrant.

Clinical examination

The pictures (figure 2, 3 and 4) show the region from the maxillary left first premolar to the first molar. The canine and the second premolar were abutments in a fixed partial denture. Tooth 24 was missing. Tooth 26 had a metal-ceramic crown with composite restorations occlusally and mesially where the porcelain had fractured. The patient complained about severe pain by light touch of the skin over the nose and zygomatic bone on the left side. There were no signs of extra- or intra-oral swelling of this region. Reaction to palpation of masticatory muscles and temporomandibular joint (TMD) were within normal limits. No supra-occlusion was detected. Tooth 23 and 25 gave positive response to Endo-Ice®. Pocket probing depth (PPD) was within normal limits, but tooth 26 had a furcation involvement grade I buccally. The tooth was tender to percussion but not palpation. She was intent on keeping the tooth. She was informed that an attempt would be made but that I couldn't guarantee success nor that all the problems she was experiencing would disappear if the treatment should be successful.

Figure 2

Figure 3

Figure 4

Radiographic examination

Figure 5

The radiograph (figure 5) shows the region from the left maxillary canine to the first molar. The crowns of all teeth had radioopaque restorations, in line with metallic-ceramic restorations; a single crown for 26 and a fixed bridge from 23 to 25. The PDL-space could be followed all the way around the canine and second premolar. An area with the radiodensity and structure of dentine was seen in the cancellous bone under the pontic. This was assumed to be a part of the root of the previously extracted 24. A material with the radioopacity of guttapercha was seen in the roots of the first molar. The PDL-space could be followed halfway down the mesial root where it widened into a well circumscribed radiolucency with the diameter of 7mm. The radiolucency encircled all three roots. The guttapercha cones in the two buccal roots were crossing each other indicating marked curvature of the MB and DB roots.

Diagnosis

Chronic apical periodontitis tooth 16

Treatment plan

Treatment of necrotic pulp tooth 16
Surgical intervention if needed

Treatment

First visit, October 7th 2003

Figure 6

One carpule xylocaine with adrenaline was administered. An access cavity was prepared with round steel burs in high and slow-speed handpieces. Four canal orifices were localised. An operation microscope supported most steps of the treatment. Sealer and amalgam was found covering a perforation in furcation area approximately 1 x 2mm in diameter situated between DB and MB canal orifices (figure 6). Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol (CHX). Some guttapercha (gp) was removed with Gates Glidden burs. The perforation was irrigated with sodium hypochlorite (NaOCl) 1 % and calciumhydroxide ($Ca(OH)_2$) dressing left in place for 10 minutes to control bleeding (figure 7) The four canal orifices were covered with Cavit G®. Two applications of $Ca(OH)_2$ were needed to stop the bleeding. The $Ca(OH)_2$ was removed by means of sterile saline and dried with sterile paperpoints. Mineral Trioxide Aggregation (MTA) was placed and condensed with sterile paperpoints. Moist cotton pellets were placed over the MTA and the access cavity was sealed with IRM.

Figure 7

Second visit, October 15th 2003
(one week later)

The patient reported less episodes of headache since the last appointment. The tooth was still tender to percussion. One carpule xylocaine with adrenaline was administered. Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol (CHX). The MTA (figure 8) was found to have set. An operation microscope supported most steps of the treatment.

Figure 8

Gp was removed with H-files 50, 40 and 30 and piezoelectric ultrasound with a K-file. Minute amounts of chloroform on sterile paperpoints were used to remove gp in concavities of the palatal and mesiobuccal canal walls. A radiograph was exposed to confirm that all guttapercha had been removed (figure 9).

Figure 9 Removal of gp controlled

During the session the following deviations were found:
1. Strip perforation on the mesial aspect of the palatal canal approximately 2-3 mm from working length (apexlocator indicated being in the periodontal ligament (PDL) and there was bleeding).
2. Apical perforation of the DB canal.
3. Perforation in a via falsa of the DB canal.
4. Perforation to the PDL of the MP canal with transparent exudation.

Lack of time made it necessary to end the session. Irrigation was sodium hypochlorite 1 % and EDTA 17 %. 2 % chlorhexidine in 70 % ethanol was left in the canals for 5 minutes. The canals were dried with sterile paperpoints and $Ca(OH)_2$–slurry was placed with a lentulo-spiral. IRM was placed in the access cavity.

Third visit, October 22nd 2003
(two weeks after initiation of treatment)

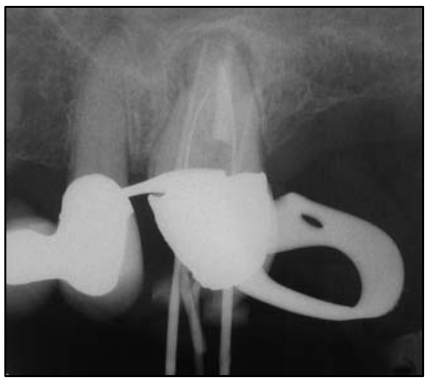


Figure 10

Rubberdam was applied and disinfected by proper means. Initially only the palatal canal was reopened and irrigated with NaOCl and EDTA. The canal was dry after leaving the last paperpoint in place for 60 seconds. MTA was placed and covered with a moistened cotton-pellet and Cavit-G®. The other canals were then reopened and the $Ca(OH)_2$ was washed out with NaOCl and EDTA using a 27G needle. A working length (WL) radiograph was taken (figure 10): MB (perforation) guttapercha #35/21mm with reference-point PC. MB 30/18mm ref. MBC. DB canal K-30/17mm ref. DBC, DB (perforation) gp #35/16mm ref. DBC. A gp was then placed in the MB canal and the perforation in the MP canal filled with MTA. An application of $Ca(OH)_2$–slurry was done in the DB canal and perforation. IRM was placed as temporary filling.

Fourth visit, November 12th 2003
(five weeks after initiation of treatment)

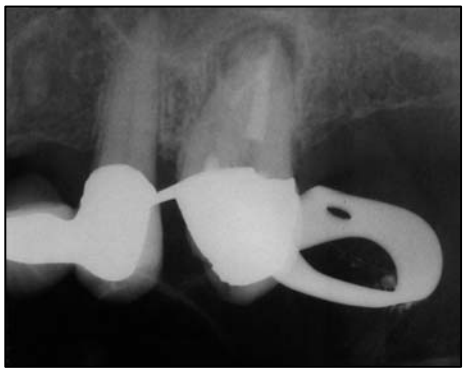


Figure 11

The tooth was tender to palpation. Rubberdam was applied and disinfected as previously described. The temporary IRM was removed. The gp placed to protect the mb canal separated when pliers was used to remove it. The X-ray taken clearly shows the gutta-percha remnant but less of the MTA placed in the MP canal. The MTA in the furcal perforation was also visible (figure 11). I tried to make the gp come out with chloroform and pre-contoured K-files without any luck. Bleeding confirmed a perforation. The mastercone X-ray illustrated that 2mm of gp persisted (figure 12). It was decided to obdurate the canals anyway and perform surgery if the tooth remained symptomatic. $Ca(OH)_2$ was applied for 10 minutes to control bleeding in the MB canal. Irrigation was sodium hypochlorite 1 % and EDTA 15 %. The root-canals were filled with guttapercha 02-taper-cones and cold lateral compaction; DB 30/17mm, DB perforation 30/16mm, MB 40/16mm. Accessory points B. AH Plus was used as sealer. Guttapercha was removed 2 mm into the canals and the access cavity washed with CHX. Cavit-G® was placed into the canal orifices, covered with saline and left to set for 10 minutes. A composite Filtec Z-250® was placed in the access-cavity after acid-etch and Syntac Sprint® one-step bonding. A post-treatment radiograph was exposed (figure 13).

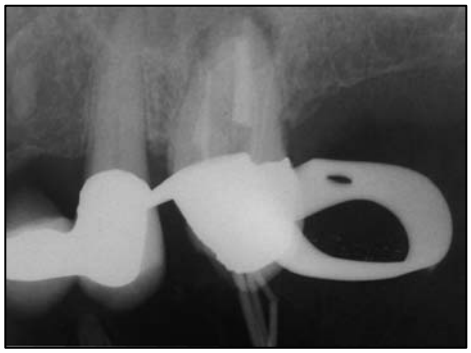


Figure 12 Mastercone radiograph

Figure 13 Post-treatment radiograph

Fifth visit, December 8th 2003
(two months after initiation of treatment)

The patient was seen for a check-up 10 days after completion of root canal treatment. Tooth 16 was still tender to percussion and palpation. The patient was informed that the more diffuse pain of head and neck might persist after surgery, so also the headache. The patient consented to further treatment and was scheduled for surgery.

Sixth visit, December 9th 2003

Apicectomy of MB and DB roots of tooth 26

Surgical procedure:
Four carpules of X/A was administered to establish anaesthesia. The patient was asked to rinse with 10ml Corsodyl® 5mg/ml solution for one minute. An aseptic field was created. A marginal incision was done with a scalpel-blade #12 from tooth 13 distally to 26 distally with a releasing incision anteriorly. A full-thickness flap was elevated (figure 14) and a perforation of the cortical bone 3 x 3 mm was found 10mm apically of the marginal bone-level. The granulation tissue was firmly attached to the roots and circumferential bone. It was enucleated in the best manner possible and sent for histology. The buccal roots were curved to a degree where they overlapped (figure 15). The mesiobuccal and distobuccal apices were resected with a steel fissure-bur in a high-speed hand-piece with copious amounts of sterile saline as coolant (figure 16). The retropreparation was done with diamond-coated retro-tips in an ultrasonic unit (figure 17). MTA was placed using a Zeiss Opmi-pico® microscope (figure 18).


Figure 14

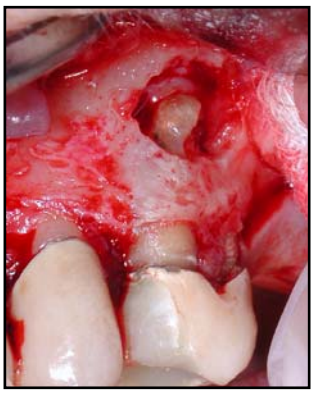


Figure 15


Figure 16


Figure 17

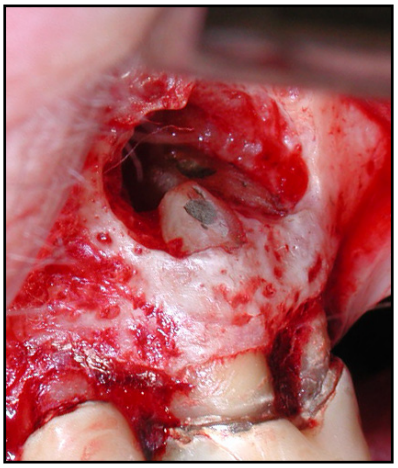


Figure 18

An X-ray of the procedure was taken (figure 19). Five sutures were placed after keeping the flap in place with light finger-pressure for five minutes (figure 20). The patient was given instructions for the first 10 days after surgery. Prescription of Apocillin® 660 mg x 30, Ibuprofen® 600mg x 30, Corsodyl® solution. She was given an icepack to hold to her cheek in intervals the first hours.

Figure 19

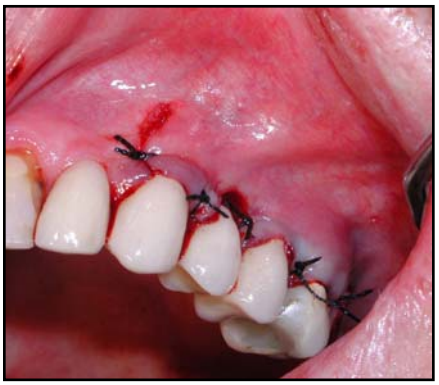


Figure 20

Sixth visit, 17.12.03
(eight days after surgery)

The patient told of moderate pain the first four days. The headache had been unchanged since the procedure. The tooth was tender to percussion, and the area representing the flap was tender to palpation. Healing was proceeding by secondary intention. Five sutures were removed (figures 21 and 22).

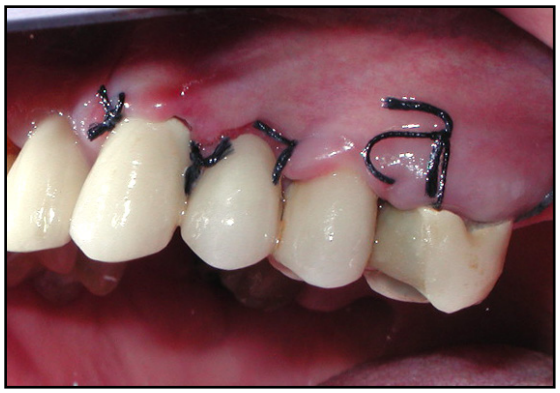


Figure 21

Figure 22

Histology:

Sections of wall from cyst, partially covered with squamous cells. Moderate chronic and intense subacute inflammation was seen in the connective tissue of the cystic wall. Foreign matter with microscopic appearance in line with endodontic filling-material AH Plus (figure 23, 24), $Ca(OH)_2$ and paper-points (figure 25, 26).

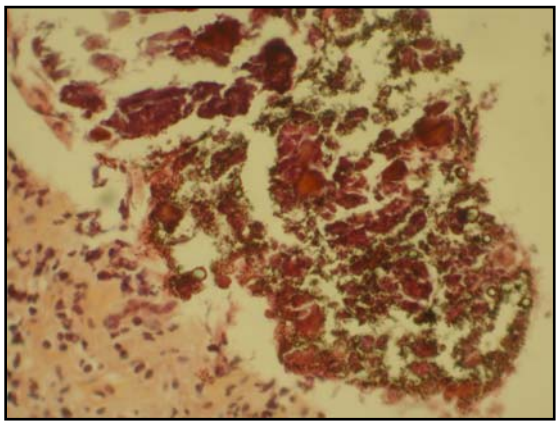


Figure 23 HE-stain (original 25x magnification)

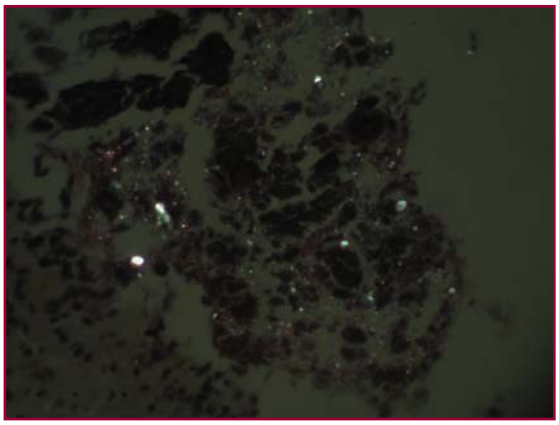


Figure 24 Particles with birefringent properties

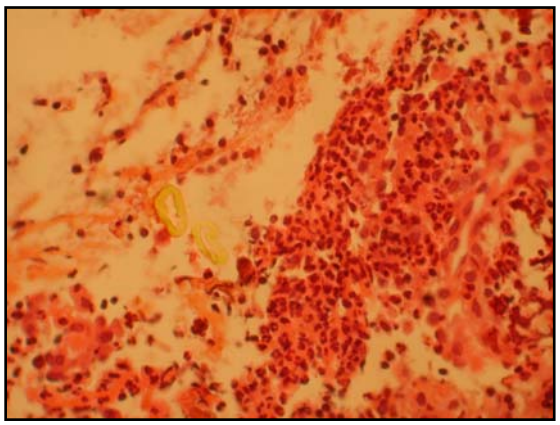


Figure 25 HE-stain (original 25x magnification)

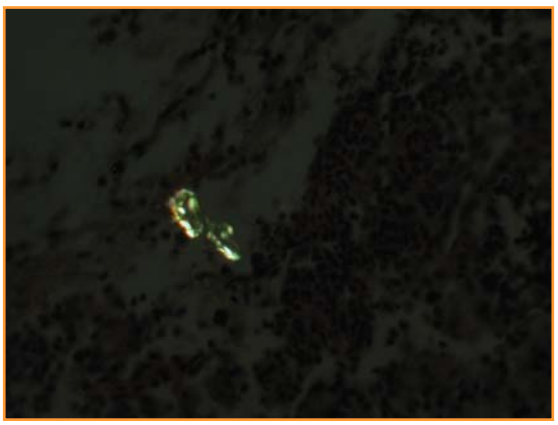


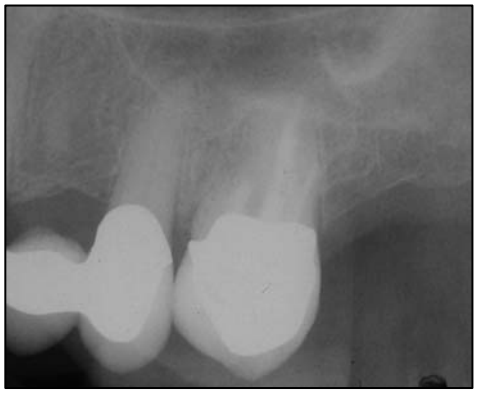
Figure 26 Particles with birefringent properties

Figure 23 and 25 are 25 x magnification-slides from histology stained with Hematoxylin-Eosin. Figure 24 and 26 are the same slides as figure 23 and 25 seen through a filter that brings out birefringent properties in substances foreign to the body.

Prognosis

Considering the multiple defects and the chance that some part of the treatment perhaps wasn't optimally executed, the prognosis was deemed uncertain.

Follow-up



18 months after surgery the patient was back for control of tooth 16. There were no symptoms from the area. The patient was no longer bothered with headache. Pain with no apparent point of origin was still present with intervals from weeks to months. The radiograph indicated that periapical healing had taken place (figure 27).

Figure 27
18 months follow-up June 2005

Discussion

In retrospect it is obvious that in a private practise a cost-benefit analysis would be appropriate. Disregarding the fact that a more experienced operator could have completed the treatment in fewer sittings and with fewer errors, it would still be a time-consuming procedure with an uncertain outcome. Having seen the result it is always satisfactory to have accomplished the task. With few exceptions, apical periodontitis is a result of a bacterial infection (1). Failure in removing the ethiological factor(s) often results in persistence of pathology. This may not have been a true refractory apical periodontitis (2). The histology defined the periapical lesion in this case to be a cyst. A block section would be needed to distinguish a radicular pocket cyst, which is expected to heal after proper treatment, from a radicular true cyst that is self-sustaining and tends to persist after proper conventional root canal treatment (3). It is very possible that although the best effort was put down in the retreatment, the intraradicular source of infection was not eliminated.

One known factor in perpetuation of inflammation is foreign bodies (4-6). Different kinds of foreign particles were found in the periapical lesion. Although opinions vary as to the importance of the foreign particles in the apical periodontitis, it is obvious that cellulose (paper-points) in the periradicular tissue seems unnecessary. In that respect I, as an Endodontist, have to agree with Koppang in that the use of paper-points should be discontinued (4). But in lack of a better tool it is likely that cellulose fibres will be found in future histologies dispatched by me.

The treatment outcome after endodontic surgery is considered to be better in conjunction with root canal treatment (conventional retreatment retreatment) than alone. Figures based on weighed averages show 80 % versus 59 % (4). A more recent review of the literature has been done by Friedman. The seven studies evalualted differed considerably in treatment outcome; 37-91% healed several years after surgery. The variation reflects differences in inclusion criteria. Several pre-operative factors may influence outcome of treatment; the outcome may be better in teeth with small lesions and excessively short or long root canal fillings, and it may be poorer in teeth treated surgically for the second time. The choice of root-end filling and the quality of the root-end filling may influence the outcome, while the retrograde procedure clearly offers a better outcome than the standard root-end filling (7).

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Case 13

Retreatment of a mandibular right first molar with anatomically related sclerotic area of mandibular bone

Patient

A fifty-seven year old white Norwegian female was referred from her general dental practitioner (GDP) to the Postgraduate Endodontic Clinic January 2005 for re-treatment of the previously root-filled mandibular right first molar (tooth 46) that was scheduled for a new prosthetic crown.

Medical history

The patient used Lipitor® to control her cholesterol levels. She experiences palpitations from local anaesthetics containing Adrenaline. She is otherwise healthy with no known allergies.

Dental history and chief complaint

Tooth 46 was root canal treated (RCT) in 1980 (figures 1, 2 and 3) because of pain. The RTC had been done with guttapercha and Procosol®. Prior to this the patient had been to the Department of Oral Surgery at Ullevål Hospital, Oslo, Norway for evaluation of the sclerotic zone in relation to tooth 36. This was diagnosed as ossifying fibroma. The region had been monitored since then, and the fibrous area had remained unchanged (figures 4 and 5). The patient's problem was periodic swelling in the buccal mucosa overlying tooth 36, and tenderness to biting.

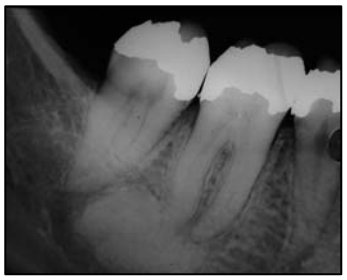

Figure 1 November 1979

Figure 2 February 1980

Figure 3 November 1980

Figure 4 September 2004

Figure 5 December 2004

Clinical examination

The photos (figure 6-8) show the region from the lower right first premolar (tooth 44) to the lower right second molar (tooth 47). There was some gingival inflammation in relation to bacterial plaque in the gingival area of all teeth. Teeth 44 and 45 had MOD amalgam restorations and composite restorations near the gingival margin buccally. Tooth 46 had a gold-acrylic crown with discoloration and signs of wear. Tooth 47 had an MO amalgam filling, and the distal aspect of the occlusal surface showed extensive wear or loss of filling-material. Tooth 44, 45 and 47 tested positive to Endo-Ice®. Pocket probing depth (PPD) was within normal limits for all teeth. Tooth 46 was tender to percussion.

Figure 6

Figure 7

Figure 8

Radiographic examination

The radiographs (figures 9 and 10) show the region from 45 to 47. Teeth 44 and 45 had coronal restorations with radio-opacities in line with amalgam. The less radioopaque areas in the cervical area of 44 and 45 resemble composites. The lamina dura could be followed all the way around the roots of these teeth.

Figure 9 mesio-radial projection

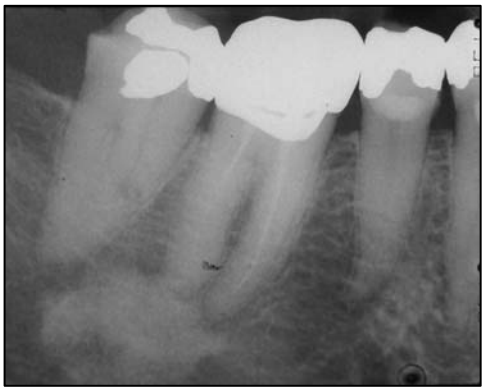

Figure 10 ortho-radial projection

Tooth 46 had a coronal radiodense restoration with the characteristics of a prosthetic crown. Centrally in the roots a material more radioopaque than dentine was seen, representing an incomplete root-filling of guttapercha. The lamina dura could be followed around the two roots. Tooth 47 had a radioopaque MO-restoration resembling an amalgam. The PDL-space could be followed all the way around the two closely gathered roots. There was a general marginal bone loss of approximately 2 mm. Apical and distal of the apices of 46 a more radiodense area of the bone was seen. It was approximately 1 cm in diameter and of varying density. Its borders were undefined and irregular with no clear demarcation from the surrounding bone. The lesion did not move much in relation to the roots from one projection to the next (figures 9 and 10), implying that the lesion lay in close proximity to the apices.

Diagnosis

Tooth 36 sclerotizing chronic apical periodontitis

Treatment plan

Retreatment of tooth 36

Treatment

First visit, February 23rd 2005

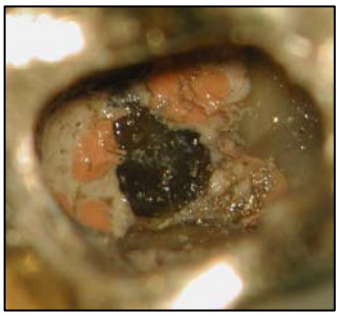

Figure 11

Access cavity was prepared (figure 11) with steel fissure burs in a high-speed handpiece and long shank round burs # 16, 12 and 10 in a slow-speed handpiece. MB, ML and D canal orifices with guttapercha were localised. Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The guttapercha was removed with minute amounts of chloroform, pre-RaCe-instruments and Hedstrom-files # 50, 30 and 20. A working length radiograph was exposed with K-file 50/19mm D, H-file 40/19mm ML and K-35/16mm MB. Attempts were made to negotiate further in all canals with irrigation of chloroform first, then EDTA and K-files 20,15,10, 08 and 06. The canals were instrumented to sizes 50/16mm MB, 45/21mm ML and 60/19mm DB, 60/19mm DL with copious amounts of 1% NaOCl and EDTA 15% and dried with paperpoints. $Ca(OH)_2$ was inserted with the last K-file for each canal, using counter-clockwise rotation. The $Ca(OH)_2$ was then packed with paperpoints and the tooth temporized with Cavit-G and IRM.

Second visit, May 1st 2005
(8 weeks later)

The tooth was asymptomatic. Rubberdam was applied and disinfected by proper means. The $Ca(OH)_2$ was washed out with the aid of NaOCl and EDTA. Canals were dry. A masterpoint radiograph exposed (figure 12). The tooth was filled with guttapercha 50/16mm MB, 45/21mm ML, 60/19 DB, 60/19mm DL using cold lateral condensation technique with accessory-points B and sealer AH+. IRM was placed in the access cavity. A post-treatment radiograph was taken (figure 13).

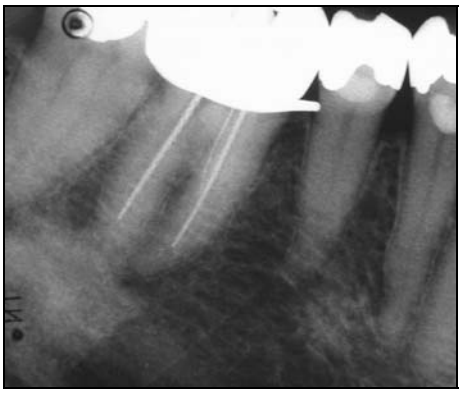

Figure 12

Figure 13

Prognosis

The prognosis was assumed to be good.

Follow-up

The patient has not been willing to return for a follow-up examination. Her GDP sent this radiograph taken January 24th 2006 (figure 14), eight months after root canal treatment. He confirmed that the tooth was asymptomatic.

Discussion

Teeth that exhibit inadequate endodontic treatment but fulfil the definition of success may be left as they are. This is no longer the case when a new restoration is needed. Since one of the more important factors influencing the outcome of endodontic therapy is the preoperative periapical status of the tooth (1), the prognosis here needs to be related to the pre-treatment diagnosis given to the tooth. Sjögren et al showed in their follow-up study that teeth without periapical lesions were treated successfully in more than 96 % of the cases. If this is a condensing osteitis, the short filling of the mesiobuccal canal may negatively influence the prognosis.

Sclerotizing (condensing) apical periodontitis sometimes arises from chronic pulpal inflammation or a low grade pulpal infection (2, 3, 4). The inflammatory stimulus then causes a reactive bone tissue formation at the apex of the involved tooth (2, 3). Untreated, this condition will usually develop into chronic apical periodontitis (2). The sclerosis is accomplished by deposition of new bone along existing trabeculae, which increases in size and constricts the marrow spaces. Condensing osteitis occurs most frequently in the mandible, around teeth with caries and/or restorations, a periapical granuloma or radicular cyst, or root canal treatment (5).

Some lesions may present difficulties in the differential diagnosis of sclerotizing apical periodontitis.
-Periapical cemental dysplasia.
-Cementoblastoma.
-Cementifying and ossifying fibroma.
-Fibrous dysplasia.
Most sclerotizing lesions have three stages, the last of which gives a radioopaque appearance (6). In this case, success of the endodontic treatment should be possible to observe on follow-up radiographs as resolution of the condensing area. The sclerotic area persisted eight months after RCT. The treatment must either be considered a failure, or a differential diagnosis considered.

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Case 14

Retreatment of a maxillary central incisor with cervical resorption

Patient

A fiftytwo year old white Norwegian female (figure 1) was referred from the Student Clinic to the Postgraduate Endodontic Clinic December 2004 for treatment of the maxillary left central incisor (tooth 21).

Figure 1

Medical history

The medical history was non-contributory.

Dental history and chief complaint

The maxillary left central incisor had been traumatized some thirty years ago when she had hit her face in a skiing-accident. The patient had noticed a gradual colour-change after that. The tooth had later been root canal treated and bleached internally. 5 years ago her general dental practitioner had placed veneer on the tooth. The tooth had again been bleached with the walking bleach technique. She presently had no discomfort from the tooth but had been informed about the radiographic findings.

Clinical examination

Figure 2

Figure 3

The photos (figures 2 and 3) show the region from the upper right lateral incisor to the upper left lateral incisor. There was general gingival inflammation. There were calculus and gingival retraction buccally of the lower incisors. Both lateral incisors had amalgam restorations placed in the foramen coecum. Tooth 11 and 21 had buccal veneers extending over the incisal edge. The left lateral incisor also had a mesial class III composite. Tooth 21 had a class V glassionomer restoration at the palatal surface. Bluish discoloration was visible at the mesiopalatal aspect of the crown of 21. The tooth was tender to biting. Tenderness to percussion was noted. Mesiopalatally the pocket probing depth was 5 mm, and there was bleeding on probing. 12, 11 and 22 tested positive to Endo-Ice®.

Radiographic examination

The radiographs (figures 4, 5 and 6) show the region from the upper right central incisor to the upper left lateral incisor. Tooth 11 had lamina dura visible all the way around the root. Tooth 21 had a radioopaque material centrally in the root representing gutta-percha. The PDL-space could be traced all the way around the root. The root was shorter than that of the contra lateral tooth and the outline of the apical area was altered. The guttapercha stopped flush with the root. There was a marginal bone loss of 2 mm. In the cervical region at the mesial aspect a wedge-shaped radiolucent area was apparent. In the alveolar bone related to this area the PDL-space was not discernible. Applying the buccal object rule with the guttapercha as the reference point, it was found that the lesion was positioned somewhat palatally.

Figure 4 mesio-radial

Figure 5 ortho-radial

Figure 6 disto-radial

Diagnosis

Cervical root resorption Class II tooth 21

Treatment plan

Removal of restoration in access-cavity
Re-treatment if communication between resorption and root-filling
Surgical removal of the resorbing tissue and placement of a filling

The patient was informed about the extent of treatment and prognosis regarding cervical resorption. She was also informed that gingival retraction could be expected after surgery.

Treatment

First visit, January 1st 2005

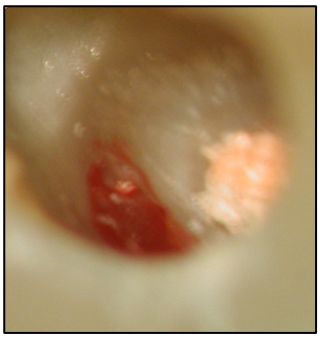

Figure 7

Rubberdam was applied and disinfected with a solution of 5 % chlorhexidine in 70 % ethanol (CHX). The access cavity was re-entered with a diamond-coated round bur in a high-speed handpiece and long shank round burs # 14 and12 in a slow-speed handpiece. The guttapercha was localised, and granulation tissue observed at the mesial aspect (figure 7).

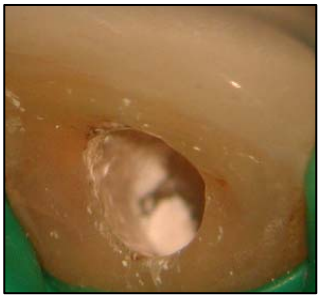

Figure 8

Ca(OH)2 was applied to the granulation tissue for 10 minutes to control bleeding. The access-cavity was then washed with EDTA and CHX and dried with sterile paperpoints. An IRM was placed over the resorption-defect (figure 8).

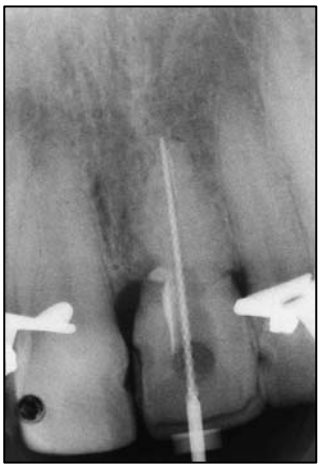

Figure 9

The guttapercha was removed with Pre RaCe-files and Hedstrom-files # 50, 30 and 20. A working length radiograph was taken with K-file 60/19, 5 mm (figure 9). The Root ZX-apexlocator applied to the file at 19,5 mm gave an audio-sign indicative of being in the PDL. The canal was instrumented with SS-files to 90/18,5 mm. Irrigation was made with 1% NaOCl and EDTA 15% and the canal dried with paperpoints. $Ca(OH)_2$ was inserted with the last K-file for each canal, using counter-clockwise rotation. The access-cavity was washed with CHX and the tooth was temporized with Cavit-G and IRM.

Second visit, February 10th 2005
(3 weeks later)

The tooth was asymptomatic. Rubberdam was applied and disinfected by proper means. The $Ca(OH)_2$ was washed out with the aid of NaOCl and EDTA. The canal was dry. A masterpoint radiograph was taken (figure 10). The tooth was filled with guttapercha 90/18 mm using cold lateral condensation technique with accessory-points B and sealer AH+. IRM was placed over the guttapercha, moisted with water and left to set for 5 minutes. The walls of the access-cavity were rubbed with a diamond bur. The access-cavity was etched for 15 seconds with Scotscbond Etch ®, rinsed with water and blow-dried. One-step Syntac-Sprint® bonding was applied and a Z-250 shade A2 composite was placed (figure 11). A post-treatment radiograph was taken (figure 12).

Figure 10

Figure 11

Figure 12

Third visit, March 8th 2005

Surgical procedure:

Figure 13

3 carpules of Xylocain-Adrenalin® was administered to establish anaesthesia. The patient was asked to rinse with 10ml Corsodyl® 5mg/ml solution for one minute. An aseptic field was created. A marginal incision was made with a scalpel-blade #12 from tooth 12 distally to 23 distally. A full-thickness flap was elevated both buccally and palatally (figures 13 and 14). The palatal flap was temporarily sutured to the upper right first molar to ease the access as the procedure was done from the palatal aspect (figure 15). The granulomatous tissue was removed by curettage. The resorption defect was smoothed with a round bur in a slow-speed hand-piece with copious sterile saline as coolant.

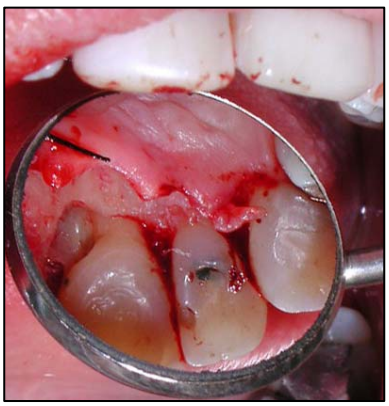

Figure 14

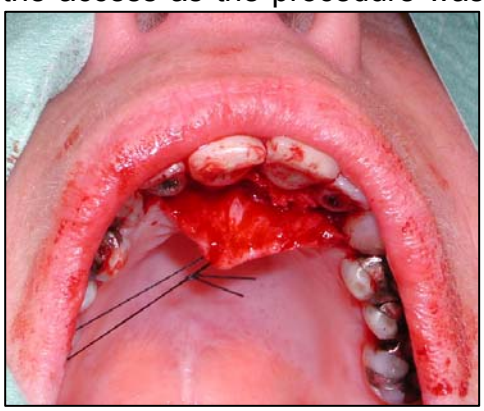

Figure 15

A bony pocket of approximately 4 mm was found apical to the defect. The cervical 1-2 mm was widended with a diamond-coated ultrasonic tip using a Zeiss Opmi-pico® microscope. A Gingipak® thread with Adrenaline was placed in the pocket. The area was washed before application of phosphorous acid. The area was again washed and blow-dried. A composite filling of Tetric-flow® and Tetric Ceram® shade A1 was placed. The surface was corrected and polished and an X-ray taken (figure 18). Five sutures were placed after keeping the flap in place with light finger-pressure for 5 minutes (figures 16 and 17). The patient was given instructions for the first 10 days after surgery. Prescription of Ibuprofen® 600mg x 30, Corsodyl® solution. She was given an icepack to hold to her lip in intervals the first hours.

Figure 16

Figure 17

Figure 18

Fourth visit, April 15th 2005
(one week after surgery)

The patient had experienced some discomfort, but taken Ibuprofen® every sixth hour the first two days, as recommended. The suture distally of 21 had fallen out the day before. The soft tissue representing the area where the flap had been elevated was tender to palpation. Healing was progressing acceptably with some fibrin visible (figure 19). The papilla distal to the left central incisor did not look good. The sutures were removed (figure 20). The patient was informed to initiate tooth-brushing in the area, and to stop the rinsing with Corsodyl®.

Figure 19

Figure 20

Prognosis

The prognosis was uncertain.

Follow-up

May 31st 2005
(3 weeks after surgery)

The tooth was asymptomatic. There was little gingival retraction, but loss of interdental papilla mesial and distal to the left central incisor. A radiographic was taken (figure 21). Caries was visible 11m. 1 carpule of Xylocain-Adrenalin® was administered to establish anaesthesia. The old filling and caries was removed and the area isolated with rubberdam. The cavity was etched for 15 seconds with Scotscbond Etch ®, rinsed with water and blow-dried. One-step Syntac-Sprint® bonding was applied and a Z-250 shade A2 composite was placed and polished.

Figure 21

April 24th 2006
(12 months after root canal treatment, 10 months after surgery)

The tooth was asymptomatic. The situation with the papillae mesial and distal of the left central incisor was improved (figures 22 and 23). There was a pocked mesiopalatally of 6 mm with bleeding on probing (figure 24). A radiograph was taken (figure 25). Apical to the composite placed during surgery a somewhat radiolucent zone is seen. The patient was informed that it was possible that resorptive tissue was left during surgery, and that the tooth needed re-evaluation also the following year.

Figure 22

Figure 23

Figure 24

Figure 25

Discussion

There was a void in the material approximately one millimetre from the apical termination of the root filling. It is apparent that sealer is lacking in the void. The difference in radioopacity apical and cervical to the void gives rise to the assumption that calciumhydroxide has been left apically. If it is, over time it will be resorbed or chemically altered. Ideally the rootfilling should be removed and a technically more superior rootfilling placed.

Cervical root resorption is a progressive external root resorption, which is of inflammatory origin. It occurs immediately below the epithelial attachment of the tooth, usually but not exclusively in the cervical area of the tooth. Its exact pathogenesis is not fully understood (1). Since its histological appearance and progressive nature is identical to other forms of progressive inflammatory root resorption, it appears logical that the pathogenesis would be the same. Due to the inhibitory effects of organic pre-cementum and predentin, even in the presence of inflammation, an intact root is resistant to resorption. However, if an injury removes or alters the (protective) pre-dentin or pre-cementum, inflammation of the pulp or periodontium will induce root resorption with multinucleated clastic cells similar to those seen in bone resorption (2).

Causes of the root damage immediately below the epithelial attachment of the root include orthodontic tooth movement, trauma, non-vital bleaching and other less definable cause (3). The pulp plays no role in cervical root resorption and is mostly normal in these cases (1). Because the source of stimulation (infection) is not the pulp, it has been postulated that it is the bacteria in the sulcus of the tooth that stimulate and sustain an inflammatory response in the periodontium at the attachment level of the root (1). This theory is strengthened by the fact that bone resorption similar to marginal periodontitis will always accompany this type of root resorption and it is universally accepted that marginal periodontitis has a bacterial cause. The destructive phase of root resorption is diagnosed primarily by the radiolucent appearance of the root and adjacent bone on X-rays. Destructive inflammation and root resorption will continue as long as a stimulus for the inflammation is present. Pressure, pulpal infection and sulcular infection are common, long-lasting stimuli for progressive root resorption.

Heithersay has classified this type of resorption into four classes in order of severity (4) (figure 26). Resorptive defects in the root coronal or just below the attachment level are relatively easy to treat but, on the other hand, if the defect extends below the bone level, it is very complicated and sometimes impossible to treat. This tooth had been subjected to trauma and non-vital bleaching, both being etiologic factors for cervical root resorption. It seems likely that the resorptive process has continued. If so, the persisting factor for maintaining resorption is likely to be sulcular infection. The tooth then falls into a category 3 or 4 resorption, leaving the tooth a hopeless case.

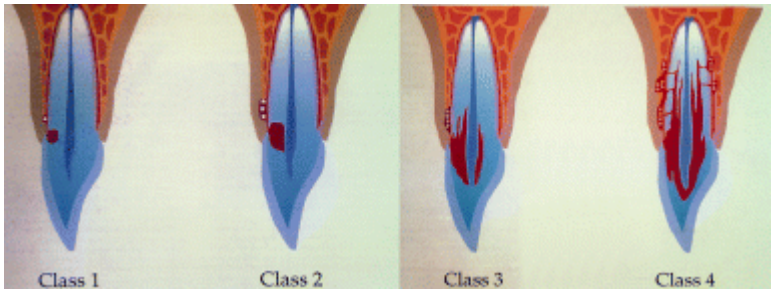

Fig. 26 Heithersay's classification of invasive cervical root resorption

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Case 15

Radisectomy of a periodontally involved upper left first premolar

Patient

A fortytwo year old white Norwegian female (figure 1) was referred from the Postgraduate Periodontic Clinic to the Postgraduate Endodontic clinic, Faculty of Dentistry, University of Oslo, September 2005 for evaluation and treatment of the upper left first premolar (tooth 24).

Figure 1

Medical history

The patient was of good health with no known allergies. She smoked 12 cigarettes a day. She had been smoking since the age of sixteen.

Dental history and chief complaint

The patient had a periodontal problem for which she was under treatment at the Periodontic Clinic. Tooth 24 had been root canal treated one year previous (figure 3), the diagnosis pre-treatment had been chronic apical periodontitis (figure 2). She had spontaneous pain from maxillary left premolar-area.

Figure 3 pretreatment radiograph 28.09.04

Figure 2 one-year follow-up 10.09.05

Clinical examination

Figure 4

Figure 5

The pictures (figures 4 and 5) show the region from the upper left canine to the upper left second premolar. There was buccal gingival retraction on all three teeth with defects assumed to be abrasive. The papillae did not fill the approximal space mesial and distal to the first premolar. Tooth 24 had an OD amalgam restoration. Tooth 25 had MO and OD amalgam restorations. Tooth 24 was mobile grade I. Pocket probing depth was 10 mm mesially and distally, 12 mm palatally and 2mm buccally. There was bleeding on probing from all periodontal pockets, and pus was detected from the palatal pocket. Pocket probing depths from year 2004; see below (figure 6).

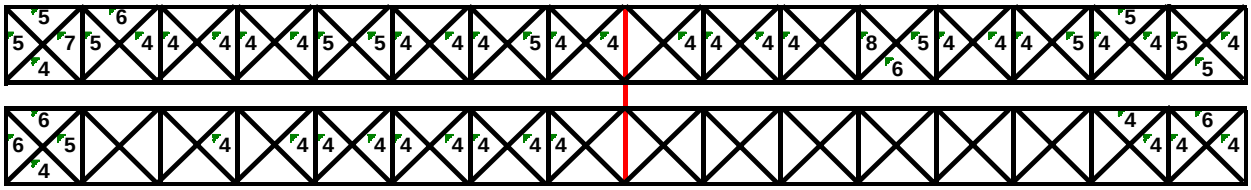

Figure 6

Radiographic examination

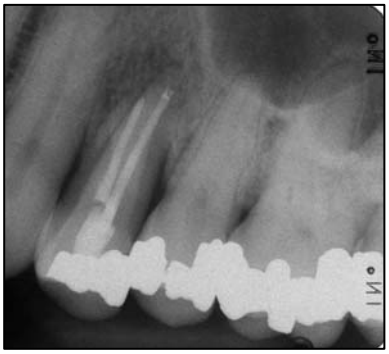

Figure 8

Figure 7

The radiograph (figure 7) shows the region from the upper left first premolar to the upper left second premolar. Tooth 24 had an OD radioopaque restoration in line with an amalgam filling. A less radioopaque material was visible in the area representing the pulp chamber and in the two root canals, resembling a temporary filling material and guttapercha. The PDL-space was visible around the buccal root. A radiolucent zone of approximately 5 mm is seen in the periapical area in conjunction with loss of the lamina dura around the palatal root. Tooth 25 had an intact lamina dura traceable around the root. Number 35 guttapercha cones were inserted, one in the mesial, one in the distal and two in the palatal pockets, and a radiograph was taken (figure 8).

Diagnosis

Apical periodontitis with sinus tract on the palatal root of tooth 24
Chronic marginal periodontitis
Endo-perio lesion

Treatment plan

Scaling and root planning of all teeth, except the upper left first premolar. This treatment was given by a resident at the Postgraduate Periodontic Clinic. Scaling and root-planing of tooth 24 was to be postponed until 2 months after endodontic-periodontic treatment (see below). Exploratory surgery tooth 24. Evaluation of the situation during surgery with different treatment-alternatives:

- Apicoectomy of the palatal root
- Radisectomy of the palatal root
- Extraction

Treatment

Endodontic-Periodontic Surgery October 26th 2005.

Figure 9

4 carpules of Xylocain-Adrenalin® was administered to establish anaesthesia. The patient was asked to rinse with 10ml Corsodyl® 5mg/ml solution for 1 minute. An aseptic field was created. A marginal incision was done with a scalpel-blade #12 from tooth 22 distally to 26 mesially. A full-thickness flap was elevated at the palatal aspect and complete loss of alveolar bone around the palatal root was noted (figure 9). There seemed to be bone covering the palatal side of the buccal root. The granulation tissue was removed and the palatal root resected in the furcation area with steel fissure-burs in a high-speed hand-piece with copious amounts of sterile saline as coolant. Due to the alveolar bone level this level of resection would be supra-gingival after surgery. The retropreparation was done with diamond-coated retro-tips in a piezoelectric ultrasonic unit and IRM was placed using a Zeiss Opmi-pico® microscope. When the IRM had set, the resected surface was smoothed with a steel fissure bur. The resected area was then acid-etched for 15 seconds, rinsed with saline and blow-dried. Syntac-sprint® one step-bonding was applied and a thin layer of Tetric-flow® composite was placed and light-cured. An X-ray of the procedure was taken (figure 10). The surgical site was debrided with copious amounts of saline. 3 sutures were placed after keeping the flap in place with light finger-pressure for 5 minutes. Using a diamond bur, the tooth was brought out of occlusion. The patient was given instructions for the first 10 days after surgery. Prescription of Ibuprofen® 600mg x 30 and Corsodyl® solution was given. She was given an icepack to hold to her cheek at intervals the first hours.

Figure 10

Prognosis

The prognosis for this tooth was uncertain.

Follow-up

November 5th 2005

Figure 11 November 5th 2005

Figure 12 Everstick®

The patient was back for removal of sutures after 10 days, as recommended for periodontal surgery. Healing was progressing uneventful (figure 11). The patient was asymptomatic. The tooth was still mobile grade I, and it was decided to stabilize the tooth with Everstick ® (figure 12). Patient was informed of the importance of oral hygiene at the site and given instructions in flossing. Three weeks after surgery the patient was back for a review of her technique in oral hygiene (figure 13). It was evident that her technique with Superfloss® was satisfactory. Two radiographs were taken (figures 14 and 15).

Figure 13

Figure 14

Figure 15

Discussion

During surgery different treatment strategies were discussed. Since the buccal root seemed embedded in alveolar bone, extraction was ruled out as choice of treatment. Apicoectomy of the palatal root was futile, since all the alveolar bone was lost (1). A radisectomy was performed. Parameters for future hygienic procedures weighed heaviest when choosing where to cut. An oblique angle from the bifurcation upwards was therefore made to the resection. The Periodontist giving advice during surgery said the plan was to make a prosthetic crown shaped as a canine if the treatment outcome was successful. Still it was decided to keep the palatal cusp. I was in disagreement of this choice. The palatal cusp, although reduced in height will have an effect as a cantilever and negatively infuence the possibilities of healing at the site (2, 3).

A void is clearly visible (figure 15) in the area between the retrograde IRM and the occlusal filling. This was an error in the placement of the IRM. But as the whole area would rest supra gingivally, it was decided to not prolong the procedure and close the surgical field without redoing the IRM.

References:

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Case 16

Retreatment of a maxillary left first premolar
Retreatment of a maxillary left second premolar with apical periodontitis with sinus tract and a radicular cyst
Treatment of a maxillary left first premolar as a sequel to surgery

Patient

A sixtysix year old white male (figure 1) was referred from the Student Clinic to the Postgraduate Endodontic Clinic, Faculty of Dentistry at the University of Oslo, Oslo, Norway, April 2005 for treatment of the upper left first and second premolar.

Figure 1

Medical history

The medical history was non-contributory.

Dental history and chief complaint

The patient experienced intermittent spontaneous pain from the maxillary left premolar area. The teeth felt tender and there was a history of foul taste and swelling of the buccal mucosa in the region. Teeth 24 and 25 were previously root canal treated. The patient could not tell the year of endodontic treatment, but could confirme that it was more than four years earlier. Prosthodontic treatment was planned for the maxillary left first and second premolars.

Clinical examination

Figure 2

Figure 3

Figure 4

The pictures (figures 2, 3 and 4) show the region from the left maxillary canine to the left maxillary second premolar. Some discoloration was seen. The canine had an OD composite restoration. The first premolar has an MODP IRM-filling. The second premolar had a metal-ceramic crown. Probing the edges, the fit was found satisfactory. There were no signs of extra- or intra-oral swelling of the area. There were no symptoms from tooth 24. Tooth 25 was tender to palpation. There was a sinus tract buccally in the transition between the attached and the free gingiva. Tooth 26 was positive to EPT and Endo-Ice®.

Radiographic examination

Figure 5

Figure 6

The X-ray (figure 5) shows the region from the left maxillary first premolar to the second premolar. The bulk of the crown of tooth 24 was restored with a material with radioopacity resembling IRM. A radioopaque mass in the center of the root represented a root-filling material. The PDL-space could be followed around the buccal root. The palatal root was superimposed over the radiolucent zone surrounding the root of tooth 25. Even so, the PDL-space seemed to go undisturbed until it crossed the outline of tooth 25. Tooth 25 had a densely radioopaque structure coronally that was in line with the radiographic appearance of a metallic crown and post. A less radiodense mass represented the rootfilling-material. The PDL-space could be followed down midroot mesially and distally before widening into a somewhat diffuse radiolucent zone whose outline exceeded the borders of this X-ray. Its largest outline was measured at 17 mm.The PDL-space indicated that two roots were present. The guttapercha cone # 35 inserted into the sinus tract is shown in the second X-ray (figure 6).

Diagnosis

Tooth 24
Inadequately root filled tooth
Tooth 25
Chronic apical periodontitis with sinus tract

Treatment plan

Tooth 24
Re-treatment prothetica causa
Tooth 25
Re-treatment, apical surgery if needed

Treatment

Tooth 24

First appointment, April 15th 2005

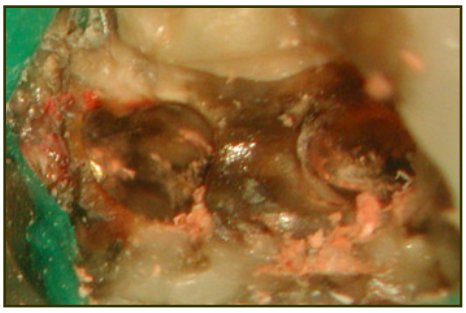
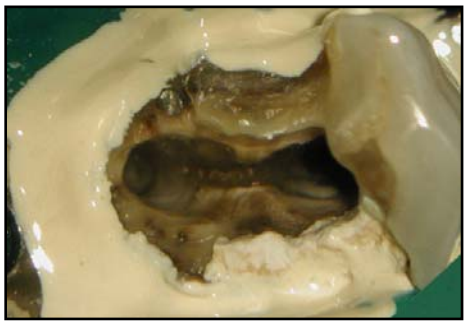


Figure 7

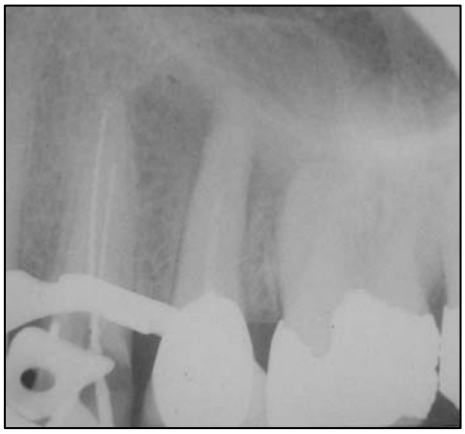
Clamp and rubberdam was applied and disinfected with 5 mg/ml chlorhexidine-ethanol solution (CHX). The IRM was removed and the two canal orifices with guttapercha localized. Guttapercha was removed with Gates Glidden burs and Protaper F3 and F2 files (figure 7). A working-length radiograph was exposed with K-file 40/20mm in the buccal canal and a Hedstrom-file 40/21mm in palatal canal. Apical box-preparation was ended with K-file 50 in both canals. Irrigation was done with sodium hypochlorite (NAOCl) 1 % and EDTA 17 %. The canals were dried with sterile paper points. Calcium-hydroxide dressing was introduced with lentulo-needle #35 and condensed with sterile paper points. The tooth was temporized with IRM.

Second appointment, June 1st 2005



The tooth was asymptomatic. Rubberdam was applied and disinfected as previously described. The $Ca(OH)_2$ was washed out with 10ml of NaOCl and EDTA each (figure 8). The canals were dry.

Figure 8



A mastercone-radiograph was taken (figure 9). The tooth was rootfilled with 02-taper Resilon® 50/20mm buccally, 50/21mm palatally and accessory points MF and M. Epiphany® was uses as sealer. A warm plugger was used to sever the Resilon® below the canal-entrances. Excess sealer was removed from the access cavity followed by 40 seconds of light-curing. IRM was then placed as a top filling.

Figure 9

A post-treatment radiograph was taken (figure 10).

Figure 10

Tooth 25

First appointment, April 26th 2005

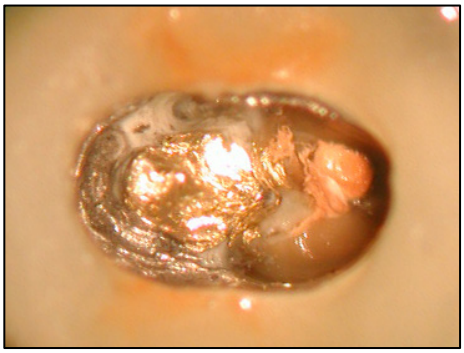


Figure 11

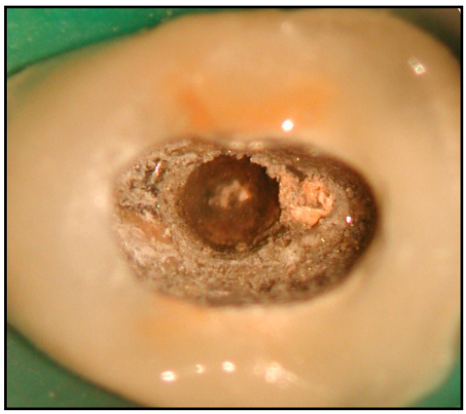


Figure 12

An access cavity was prepared with a high-speed hand-piece, using a round diamond bur for the ceramics, and hard-metal fissure burs for the metallic part of the crown. The post was protruding from the buccal canal in an oblique direction towards the palatal aspect (figure 11). Rubberdam was applied and disinfected with CHX. The post was removed by means of a K-file ultrasonic tip in a P_5® piezoelectric ultrasonic unit (figure 12). The guttapercha in the buccal canal was removed with a torpan bur size 50 and minute amounts of chloroform. In the microscope an area of pale yellow colour could be seen when all the guttapercha had been removed. Due to the slight curve of the canal this could not be removed without removing considerable amounts of dentine further coronally, and was therefore not done. An LN bur was used to search for the palatal canal orifice (figure 13). Eventually moist appeared on the dentine-surface (figure 14), and when further probed, pus seeped through (figure 15). An 08-file was inserted into the canal and worked in a balanced-force technique until the Root ZX® apexfinder indicated being in the PDL at 20 mm

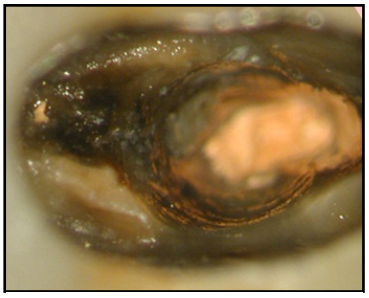


Figure 13

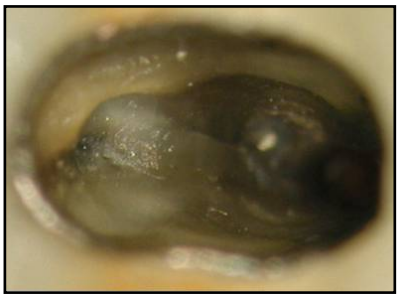


Figure 14

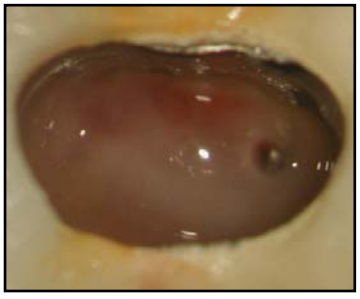


Figure 15

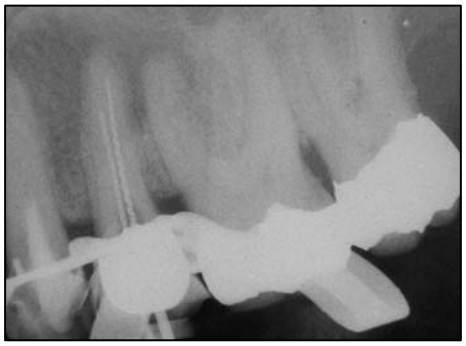


Figure 16

After instrumentation of the palatal canal with an apical box, a WL-radiograph was exposed with K-40/16.5b, H-40/19mm p (figure 16). Irrigation was done with NaOCl and EDTA with a 27G needle. The canals were dried with sterile paper-points before a $Ca(OH)_2$ slurry was placed and the tooth temporized with Cavit-G and IRM

Second visit, May 10th 2005
(three weeks later)

The sinus tract was still present. The patient told of an episode about a week after the previous treatment where red fluid discoloured a part of his pillowcase the size of a fist. This was assumed to come from the sinus tract. Rubberdam was applied and disinfected with 5 mg/ml chlorhexidine in 70 % ethanol (CHX). The temporary filling was removed and the inter-appointment dressing washed out with copious amount of NaOCl and EDTA. The canals were instrumented with 02-taper K-files to sizes 50/17b and 50/19p with reference point palatal cusp of prosthetic crown. Chlorhexidine-di-gluconate was left in the canals for 5 minutes before applying a dressing of $Ca(OH)_2$ that was condensed with sterile paperpoints. Cavit-G was placed into the canal orifices and the access-cavity. An X-ray of the packing of $Ca(OH)_2$ was taken (figure 17).

Figure 17

Third visit, June 1st 2006
(six weeks after initiation of treatment)

The sinus-tract persisted (figure 18). It was decided to fill the tooth if it was possible to keep the canals dry, and then perform surgery. Rubberdam was applied and disinfected with CHX. The temporary filling was removed and the inter-appointment dressing washed out with copious amount of NaOCl and EDTA.

Figure 18

The canals were dried with sterile paper-points and remained dry after leaving paper-points in dried canals for one minute. MTA was placed. The last radiograph of a series taken to check the density and apical extent of the MTA is shown (figure 19). Wet cotton-pellets were placed in contact with the MTA and covered with IRM.

Figure 19

Apical surgery, September 6th 2005
(two months after RCT with MTA)

Apicoectomy of tooth 25

Figure 20

A photo was taken of the persistent sinus tract (figure 20). 3 carpules of Xylocaine-Adrenaline® was administered to establish anaesthesia. The patient was asked to rinse with 0, 5 % chlorhexidine-solution (Corsodyl®) for 60 seconds. A surgical field was created. A marginal incision was made with a # 12 scalpel blade from 27 distally to 23 mesially with a releasing incision anteriorly. A full-thickness flap was elevated, revealing a massive fenestration of cortical bone buccally of tooth 25 (figure 21). Granulation tissue was enucleated (figure 22), exposing the mesial root of tooth 26 (figure 23) and a minute communication with the maxillary sinus.

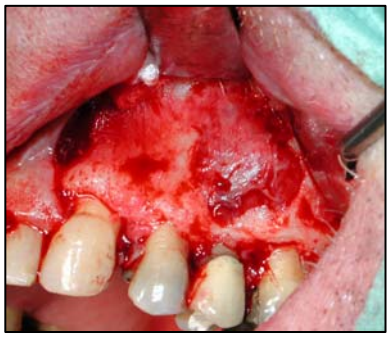


Figure 21

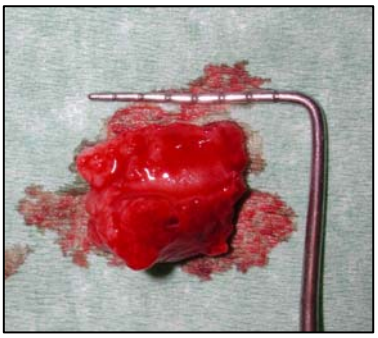


Figure 22

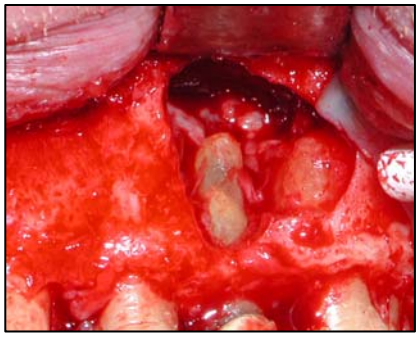


Figure 23

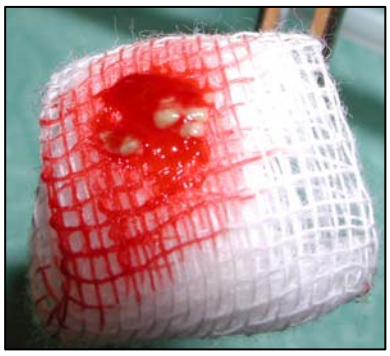


Figure 24

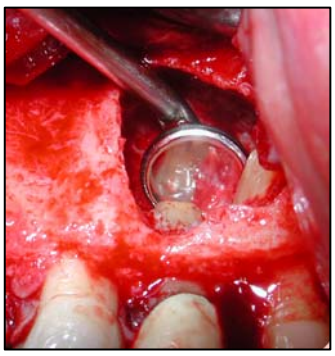


Figure 25

The granulation tissue was sent for histologic examination. The root of 25 was hourglass-shaped. Yellowish granulae were found in the bony cavity after enucleation (figure 24). Resection of apices was done with a fissure bur in a high speed handpiece (figure 25). No isthmi were seen under the Zeiss Opmi Pico® microscope. No retrograde filling was placed.

A radiograph was taken to check the resection (figure 26). The site was debrided with copious amounts of saline and the flap kept in place for 5 minutes with light finger-pressure. Six interrupted sutures were placed (figure 27). The patient was given oral post-operative instructions and asked to use Corsodyl® mouthwash morning and evening until the next appointment. A prescription of 20 Ibuprofen® 600 mg was given. The patient received an ice-pack to keep to the cheek at intervals for the first hours.

Figure 26

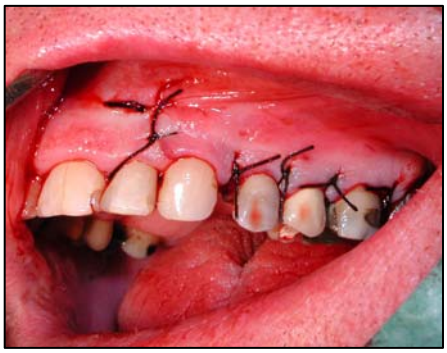


Figure 27

September 12th 2005

The patient returned six days after surgery (figure 28). He reported having had less pain than anticipated. He had taken analgetic medication every sixth hour the day of surgery ant the day after. Since then he had managed without analgetics. The area representing the flap was tender to palpation. There was some tenderness to percussion from tooth 25. The sutures were removed (figure 29). The re-establishment of interdental papillae 24M, D and 25D was unsatisfactory. Tooth 26 responded normally to Endo-Ice and EPT. It was assumed that the surgical procedure had severely damaged the nerve- and blood-supply to the mesiobuccal root. The patient was scheduled for endodontic treatment of tooth 26.

Diagnosis

Asymptomatic pulpitis tooth 26

Treatment plan

Pulpectomy of tooth 26

Figure 28

Figure 29

Histology:
Granulation tissue with moderate and intense chronic and sub acute inflammation
Fibrous connective tissue with moderate chronic inflammation consistent with parts of cyst wall where the epithelium has been destroyed, or has not been visible in the sectioning

Tooth 26

September 28th 2005

The patient gave informed consent to endodontic treatment of tooth 26. Two carpules of Septocaine® were administered to establish anaesthesia. Rubberdam was applied and disinfected with CHX. Access-cavity preparation was performed with a steel fissure bur in a high-speed hand-piece and round burs # 14 and 10 in a slow-speed hand-piece. Three canal-orifices were localized and bright red bleeding was registered from all canals. A WL-radiograph (figure 31) was exposed after subtracting 1 mm from the length where the apexlocator indicated being in the PDL: MB K-15/19.5 mm ref MBC, DB K-15/19.5 mm DBC, P H-15/19.5 mm ref PC. The mesiopalatal calan was later found and an additional WL-radiograph exposed: MP H-15/19, 5 mm ref MBC (figure 32). The canals were instrumented by hand to K-20 followed by Protaper S1, S2, Sx, F1 and F2. Apical box preparations were done with NiTi K-files #35 to #45 MB, MP and DB, and #35-60 P. Irrigation was done with NaOCl and EDTA. The canals were dried with sterile paper-points. 02-taper guttapercha cones were placed and a masterpoint radiograph was taken (figure 33). The root filling was placed using cold lateral compaction: MB 45/19.5 mm, ML 45/19.5 mm, DB 45/19.5 mm, P 50/19.5 mm. Accessory points B and C. Sealer was AH Plus. A warm instrument was used to sever the gp 1-2 mm apical of the pulp chamber floor. Cavit-G was placed over the canal-orifices and IRM used to fill the access-cavity. A post-treatment radiograph was taken (figure 34).

Figure 30

Figure 31

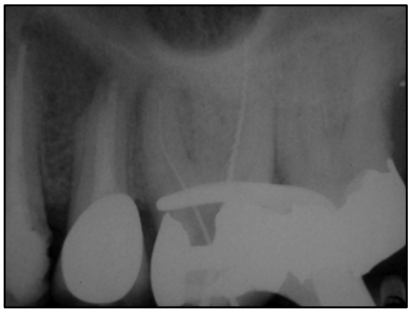


Figure 32

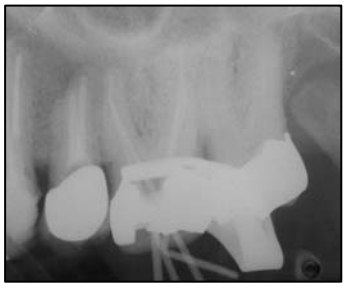


Figure 33

Figure 34

Prognosis

The prognosis of tooth 24 was good.
The prognosis of tooth 25 was assumed to be good.
The prognosis of tooth 26 was good.

Follow-up

The patient was seen six months after surgery. The upper left first premolar and molar had received prosthetic crowns in December 2005 (figure 35). The patient has no subjective symptoms, and no tenderness to percussion or palpation was found for tooth 24, 25 and 26. Two radiographs were taken (figure 36, 37). The periapical lesion of tooth 25 had healed.

Figure 35

Figure 36

Figure 37

Discussion

Several studies show that prognosis for vital pulp therapy and treatment of teeth with no radiographic signs of apical periodontitis is good (1, 2).The challenge of endodontic treatment lies with the cases of established apical periodontitis. The technical quality of the primary endodontic treatment in this case was poor. The technical result of the re-treatment was not optimal. The orthograde placement of MTA might have been easier with the aid of ultrasound. It still seems in place to mention the established factors contributing to failure of high standard endodontic treatment: 1. Intraradicular infection (3). 2. Extraradicular infection (4). 3. Foreign body reaction (5). 4. True radicular cysts (6). Of these factors, only the intraradicular infection can be affected by re-treatment of the root canal(s).

Tooth 25 did not respond favourably to re-treatment. The histology taken during surgery indicated a radicular cyst. There are two distinct categories of radicular cysts, namely, those containing cavities completely enclosed by epithelial lining (true cyst), and those containing epithelial-lined cavities that are open to the root canals (bay cyst, periapical pocket cyst). An apical true cyst is considered to be a direct sequel to apical granuloma, although a granuloma does not always develop into a cyst. For inexplicable reasons, only a small fraction, less than 10%, of periapical lesions advance into true radicular

cysts (6). Lesions of apical periodontitis cannot be differentially diagnosed into cystic and non-cystic lesions from radiographs alone (7, 8). The aim of conventional root canal treatment is elimination of infectious agents from the root canal and prevention of reinfection by obturation. A periapical pocket cyst is likely to heal after conventional endodontic treatment if the infection has been successfully removed (6). In contrast, the dynamics of a true cyst are self-sustaining by virtue of its independence of the presence or abcense of irritants in the root canal. Therefore, true cysts, particularly the large ones containing cholesterol crystals, are less likely to be resolved by conventional root canal treatment (9).

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Case 17

Surgical intervention of root filled tooth with prosthetic post and crown

Chronic apical periodontitis of the upper left first molar

Patient

A 58 year old white female (figure 1) was referred from the Student Clinic in to the Postgraduate Endodontic Clinic, Faculty of Dentistry, University of Oslo, January 2003, for treatment of the upper left first molar (tooth 26).

Figure 1

Medical history

Her cholesterol was high. She was taking 40 mg Zocor® a day. She had just diagnosed with Diabetes Mellitus type II, needing no medication for that. Her $HbA1_c$ was 6, 6 with dietary regulation only.

Dental history and chief complaint

The upper left first molar had been root canal treated at the Post Graduate Endodontic clinic in 1996. At that time the diagnosis was asymptomatic pulpitis. The buccal canals had not been located due to obliteration, and only the palatal canal had been root canal treated. The tooth had soon after endodontic treatment received a prosthetic crown. In 1999 the crown had been removed due to loss of retention, and a post and a new crown had been placed. The tooth was referred for endodontic treatment because the patient had spontaneous pain in the upper left molar region.

Clinical examination

Figure 2

Figure 3

The pictures (figures 2 and 3) show the region from the upper left first premolar to the upper left first molar. All three teeth had metal-ceramic crowns. The gingival margins showed signs of inflammation bordering the prosthetic restorations. Tooth 26 had a furcation involvement grade I buccally. Pocket probing depth (PPD) was 5mm buccally and mesially of the first molar. The patient had spontaneous pain in the area of the maxillary left first molar, and the tooth was tender to biting. The tooth was also found to be tender to percussion, and the mesiobuccal root was tender to palpation.

Radiographic examination

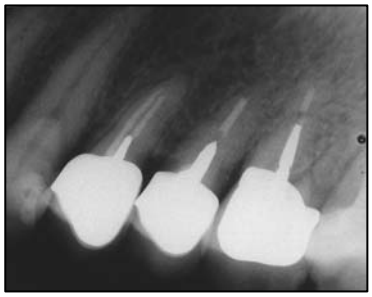


Figure 4 disto-radial

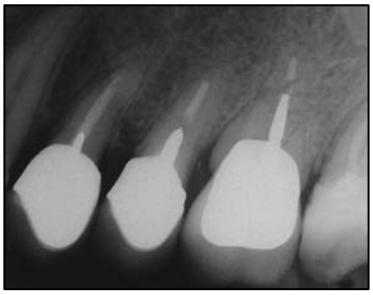


Figure 5 ortho-radial

Figure 6 disto-radial

The radiographs (figures 4, 5 and 6) show the region from the upper left first premolar to the upper left first molar. There was a general marginal bone loss of approximately two millimetres. Tooth 24 had a radioopaque coronal restoration compatible with a prosthetic crown and post. In the root a less radioopaque material in line with two guttapercha-cones was visible. The lamina dura could be followed all the way around the root. Tooth 25 had a radioopaque coronal restoration compatible with a prosthetic crown and post. In the root a less radioopaque material in line with guttapercha was visible. Tooth 26 had a radioopaque coronal restoration compatible with a prosthetic crown and post. Apical to the post in the palatal canal was a radioopaque material respresenting the apical seal of guttapercha. The PDL-space could be followed around the palatal and distobuccal roots. The crown was poorly fitted to the mesiobuccal root. Mesial and distal to the mesiobuccal root was a radiolusent sone in the cervical area. From there the PDL-space could be followed to the periapical area where it tapered into a radiolusent zone of approximately two millimetres. No pulp space was discernible neither in the mesiobuccal nor the distobuccal roots.

Diagnosis

Chronic apical periodontitis related to the mesiobuccal root of tooth 26.

Treatment plan

Radisectomy of the mesiobuccal root of tooth 26

Treatment

First visit, June 11th 2003

Figure 7

Three carpules of Xylocain-Adrenalin® were administered to establish anaesthesia. The patient was asked to rinse with 10ml Corsodyl® 5mg/ml solution for 1 minute. An aseptic field was created. A marginal incision was done with a scalpel-blade #12 from tooth 23 distally to 26 distally with a releasing incision mesially. A full-thickness flap was elevated (figure 7). The granulation tissue was removed. The amount of bone was assessed. There was communication from the cervical area to the periapical area of the mesiobuccal root, and it was found that the amount of bone lost was too extensive to perform an apicoectomy. An osteotomy of the bony bridge between the apical and marginal bone destruction was performed with a round bur # 14 in a slow-speed hand piece. The mesial root was resected in the furcation area with a steel fissure-bur in a high-speed hand-piece with copious amounts of sterile saline as coolant (figure 8). The root was removed with an elevator. The resection was performed under a Zeiss Opmi-pico® microscope. The bony crypt was covered with Stryfnon®-gauze. The resected area was acid-etched for 15 seconds, rinsed with saline, and blow-dried. Syntac-sprint® one step-bonding was applied and a thin layer of Tetric-flow® composite was placed and light-cured (figure 9).

Figure 8

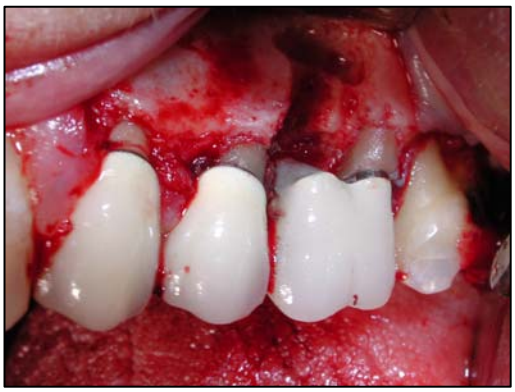


Figure 9

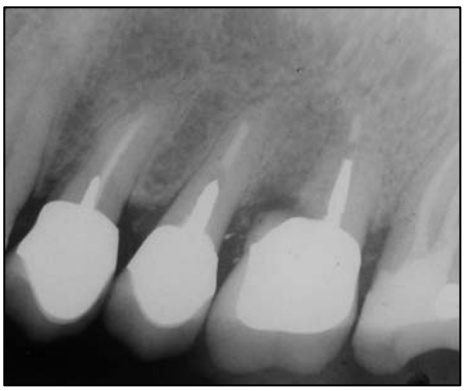


Figure 10

An X-ray of the procedure was taken (figure 10). The surgical site was debrided with copious amounts of saline. Five sutures were placed after keeping the flap in place with light finger-pressure for five minutes. The patient was given instructions for the first 10 days after surgery. A prescription of Apocillin 660 mg x 30 and Ibuprofen® 600mg x 30 was given. Oral hygiene was to be maintained with Corsodyl® mouthwash twice a day. She was given an icepack to hold to her cheek at intervals the first hours.

Second visit, June 18th 2003
(one week later)

The patient told of pain rated as seven on a scale of one to ten the following two days after surgery. She did not go to work the day after surgery. Healing was progressing uneventful, but much by secondary intention (figure 11).The five sutures were removed (figure 12). She was given hygiene instructions in the use of inter dental micro brushes and Superfloss®.

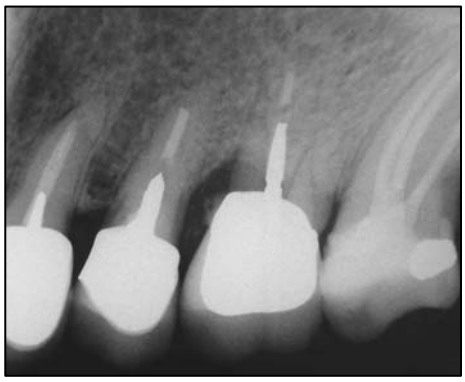
Figure 11

Figure 12

Prognosis

The prognosis was assumed to be questionable.

Follow-up



The patient did not attend her three months follow-up. At the twelve months follow-up the patient was asymptomatic. The radiograph showed a radiolucent halo in the area of the resection (figure 13). She found keeping the area free of food-debris and plaque a daily challenge.

Figure 13

Two years after treatment the tooth had been extraxted (figures 14 and 15). In the journal from the Oral Surgery Department the following diagnoses had been registered prior to extraction: Pathologic resorption, cervical caries and fracture of tooth 26.

Figure 14

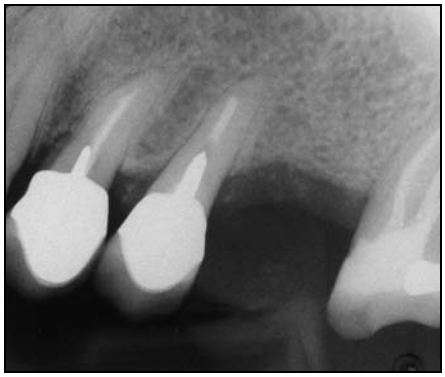


Figure 15

Discussion

At the time of surgery antibiotics was routinely prescribed after periapical surgery involving removal of bone, at the Postgraduate Endodontic Clinic. That practise has later been terminated (1). In a randomized clinical trial of 256 patients needing periapical surgery it was found that the infection rate of the surgical site was 1, 6 % (two patients) in the antibiotic-group and 3, 2 % (4 patients) in the placebo group and the difference was not statistically significant. The overall infection rate was 2, 3 %, which must be rated as low.

Success of initial treatment of apical periodontitis, based on radiographs, indicates a success-rate of 83-94% from well-controlled studies and 61-77% from epidemiologic studies (2). The level of rootfilling is more critical to outcome in teeth with apical periodontitis (3). Even in obliterated canals there is a pulp-remnant with the potential to late infection and development of a periapical lesion. Here it is tempting to speculate that this might have been the case after loss of retention and placing a new crown. Seing the outcome of treatment it is possible that a skilfullt performed apical resection of the MB apical area with retrograde MTA or IRM would have been a better choice. It certainly would have made home care easier for the patient.

Treatment outcome after hemisectioning/radisectomy varies from 79-93% success (4). More mandibular molars were treated, which may imply that case selection is paramount. The importance of a stringent periodontal maintenance regimen (home care and recall-system) was stressed. In the present case this may not have been adhered to.

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Case 18

Treatment of maxillary left and right central incisor with root fracture due to trauma

Patient

A twentyfour year old Caucasian male (figure 1) was referred from his general dental practitioner (GDP) to my private dental practise November 2004 for endodontic treatment of the maxillary right and left central incisor after trauma (tooth 11, 21).

Figure 1

Medical history

His medical history was non-contributory.

Dental history and chief complaint

March 2004 the patient experienced a vasovagal syncope during local anaesthesia at his doctor's office. He injured his teeth and upper lip hitting the floor. He received emergency dental treatment from an oral surgeon at the local hospital. The involved teeth were diagnosed as follows:

Maxillary right lateral incisor:	luxation
Maxillary right central incisor:	luxation and root fracture
Maxillary left central incisor:	luxation and root fracture
Maxillary left lateral incisor:	subluxation

Tooth 11 and 21 were repositioned and all four incisors were fixated with orthodontic ligature. The patient was put on Apocillin® 660mg x 4 for seven days, informed of the prognoses, and recommended close follow-up by his GDP. The patient was back for a control at the local hospital June 2nd 2004. The patient did not seem to contact his GDP until July 29th 2004 when the orthodontic ligature was removed. At that time teeth 12, 11 and 21 gave no response to EPT. September 6th endodontic treatment of 11 and 21 was started. Both coronal and apical parts were instrumented and dressed with Ultracal®. Teeth were reopened and inter-appointment dressing changed four times in the period from September 6th to November 2nd due to pain and/or persistent bleeding from the canal. The patient was then referred to me.

Clinical examination

Figure 2

Figure 3

The photos (figures 2 and 3) show the region from the maxillary right lateral incisor to the maxillary left lateral incisor. These teeth had no previous caries-experience. There was a healthy periodontium with pocket probing depth within normal limits. At the palatal aspect temporary fillings covering the access-cavities of teeth 11 and 21 were observed. The buccal oral mucosa overlying the roots of the central incisors had oval areas of mucosa with signs of rubor. These inflammatory signs were anticipated to be at the level of the root-fractures. Both coronal fragments of 11 and 21 were mobile grade I, but not tender to percussion or palpation apically. Some tenderness was found in the midroot-area of both central incisors. Teeth 11, 12 and 21 gave negative response to EPT. The right lateral incisor was also somewhat tender to percussion.

Radiographic examination

Figure 4

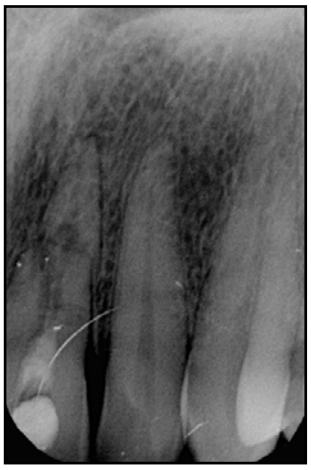

Figure 5

The radiographs (figure 4, 5) show the region from the upper right lateral incisor to the upper left lateral incisor. The radiographs have white artefacts from scratches in the Digora®-sensor. Tooth 12 had no restorations. The PDL-space was hard to discern in the apical area. The normal-anatomy of the apical area seemed changed; with radiographic loss of the structure of dentine. Dental products with the radiodensity of temporary materials were seen coronally of 11 and 21. Tooth 11 had a horizontal root fracture in the cervical third; the width of the diastasis between the fragments was estimated at 1,5mm. The edges of the fractured area were rounded, but no apparent radiolucency was seen at the lateral aspects of the diastasis. A radiolucent zone the width of a PDL- space was seen next to the dentinal edges of the fracture-site. The less radiodense line representing the pulp space was extraordinary wide in the coronal part. The PDL-space was apparent at the mesial and distal aspect of the fragments. The alveolar bone supporting the coronal fragment was approximately two

millimetres at the distal side and four millimetres at the mesial side. Tooth 21 had a root fracture in the middle third. By the mottled appearance on the radiograph, a contusion-like damage was suspected. The coronal fragment was ill-aligned with the apical part. The PDL-space was followed around the periapical area. Tooth 22 had no restorations and the PDL-space could be followed all the way around the root.

Diagnosis

Tooth 12: Symptomatic Apical Periodontitis with external inflammatory root resorption
Tooth 11: Cervical root-fracture with luxation of the coronal fragment.
Tooth 21: Root-fracture, middle third with subluxation of the coronal fragment.

Treatment plan

The treatment plan was influenced by two parameters:

- The treatment already given to the teeth
- The patient was taking a contingent for Unifor in the Balticum with duration of more than six months.

Tooth 12:
Treatment of necrotic pulp
Tooth 11:
Treatment of necrotic pulp of coronal fragment
Surgical removal of apical fragment
Tooth 21:
Treatment of necrotic pulp of coronal fragment
Surgical removal of apical fragment

Treatment

First visit, November 18th 2004

One carpule of Xylocain-Adrenalin® was administered. The temporary fillings were removed and rubberdam applied to both 11 and 21 with clamps on canines and ligature of dental floss to keep the rubberdam in place. The clamps and dam were disinfected with Chlorhexidine 5mg/ml in 70% Ethanol-solution.

Tooth 11

Under the temporary filling a cotton pellet was found. Remnants of pulpal tissue were removed from the access cavity and coronal part of the root canal. There was bleeding from the fracture-site that compromised visual inspection with the microscope. The referring dentist had informed of instrumentation of apical fragment to a size of #50/24,5mm. Further instrumentation of the apical fragment was not attempted because of the space between the coronal and apical fragment. A permanent root filling of the apical part was assumed to be a futile attempt. The apexlocator (Root ZX®) could not determine correct length to the fracture-site probably due to bleeding. The coronal fragment was irrigated with 1 % NaOCl and EDTAC 17 %. Calcium hydroxide-paste was packed with paperpoints, and a temporary filling of Cavit-G® was placed.

Tooth 21

Under the temporary filling a cotton pellet was found. Remnants of pulpal tissue were removed from access cavity and coronal part of canal. Three pale and hard fragments were found near the fracture-site, two of these were removed. The remaining one the operator was unable to loosen. They were presumed to be dentine or cancellous bone from the traumatic event. There was bleeding from the fracture-site that compromised visual inspection. The apexlocator (Root ZX) could not determine correct length to the fracture-site, probably due to bleeding. The referring dentist had informed of instrumentation of apical fragment to a size #50/25,5mm. I was not capable of finding the pulpal passage in the apical fragment in this tooth. The coronal fragment was irrigated with 1 % NaOCl and EDTAC 17 %. Calcium hydroxide-paste was condensed with sterile paper-points. A Cavit-G® temporary filling was placed. A periapical X-ray was taken to check the density of the dressings (figure 6). For tooth 11 packing of the inlay had not produced the wanted radiological density.

The GDP was informed of the pathology of tooth 12. She wanted to perform the endodontic procedure herself. She was told of the importance of quick onset of treatment.

Figure 6

Second visit, November 24th 2004

Figure 7

Both maxillary central incisors were asymptomatic. The redness in the buccal mucosa had subsided (figure 7). Rubberdam was applied and disinfected as previously described. The temporary fillings were removed and the dressing washed out with with 1 % NaOCl and EDTAC 17 %. No bleeding was observed from the fracture-sites in 11 or 21. The apexlocator indicated the fracture-site to start at 12mm in tooth 11 and at 15mm in tooth 21. MTA was placed 1mm short of this and condensed stepwise with a plugger. X-rays were exposed several times to control homogeneity and apical extent of the MTA.

The appearance of the MTA on radiographs changed minimally from one X-ray to the next, even though increasing pressure was applied in the condensation (figures 8, 9 and 10). The soft tissue of the fracture-site seemed to offer support to the MTA applied and the fillings were left at that though unsatisfactory appearance on radiographs. Moist cotton-pellets were placed over the MTA and covered with Cavit-G temporary filling.

Figure 8

Figure 9

Figure 10

Third visit, December 2nd 2004

Figure 11

The temporary restorations and cotton pellets were removed after application and disinfection of the rubberdam. Proper hardness of the MTA was confirmed with an explorer. A diamond bur was brushed against the walls of the access cavity. The dentin and enamel were acid-etched and bonded with All-bond-2®, and Z-110® A2 composite was light cured in layers (figure 11).

February 5th 2005

Eight weeks after the MTA-placement the patient was back for an evaluation of treatment. The patient had no subjective symptoms from the maxillary central incisors. Tooth 12 had been root canal treated by the GDP. Teeth 11 and 21 were not tender to palpation or percussion. The coronal fragment of tooth 11 was still mobile grade I.

Three X-rays were taken (figures 12, 13 and 14). Surgical removal of the apical fragments by a surgeon at the local hospital was scheduled two days later. An orthodontist would place a splint from the upper right lateral incisor to the upper left lateral incisor at suture-removal one week after surgery.


Figure 12


Figure 13

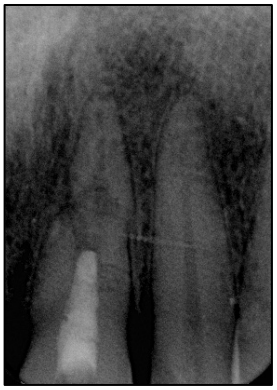
Figure 14

February 8th 2005

The surgeon called to let me know that the apical fragments had not been removed since the patient was asymptomatic and there was no periapical pathology found on X-ray.

Prognosis

The prognosis was uncertain.

Follow-up

I have not been able to get in contact with the patient. The patient has not been back to the referring dentist either.

Discussion

Cvek et al 2002 have classified the injuries to the coronal fragments related to root-fractures (1).
-Concussion when the tooth was only tender to percussion.
-Subluxation when coronal fragment also was mobile but not displaced.
-Luxation when coronal fragment also displaced. The types of healing that can be expected are classified by Andreasen & Hjörting-Hansen 1967 (2):
A. Healing with interposition of hard tissue. B. Healing with interposition of hard and soft tissue (PDL) between fragments. C. Healing with interposition of soft tissue (PDL). D. No healing. Cvek et al studied 94 incisors with transverse fractures limited to the cervical third of the root and oblique fractures involving both the cervical and middle parts of the root. They found healing of the fracture with hard tissue in 18%, healing with PDL and sometimes hard tissue in 66% and no healing with radiolucency adjacent to the fracture in 16%. Positive sensibility at the time of injury was significantly related to both healing and hard tissue repair. They also found that immature teeth had a better chance of healing than those with closed apices. The same applied to concussion or subluxation compared with dislocation of the coronal fragment, as well as optimal compared to suboptimal reposition of displaced coronal fragments. 44% of teeth with transverse fractures and 3% of those with oblique fractures were lost after healing had taken place. Mobility and bacterial contamination from the gingival crevice seem paramount to failure of healing and loss of the teeth with transverse cervical fractures.

References:

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2. Andreasen JO, Hjörting-Hansen E. Intraalveolar root fractures. Radiographic and histologic study of 50 cases. J Oral Surg 1967;25:414-26.

Case 19

Chronic apical periodontitis of a central incisor
Medically compromised patient

Patient

A fortytwo year old white female (figure 1) was referred from her general dental practitioner (GDP) to the Graduate Endodontic Clinic, Faculty of Dentistry at the University of Oslo, for apical surgery of the upper right central incisor (tooth 11).

Figure 1

Medical history

The patient was institutionalized as a result of her medical condition. Suffering from Munchausen syndrome accompanied by manic and depressive periods, she was unable to tend for herself. She has bronchial asthma. She had a considerable daily intake of drugs:

Cipralex® 30mg x 1
Sobril® 25mg x 3
Nozinan® 350mg x 1
Seroquel®300mg x 1
Apodorm® 10mg x 1
Orifiril Retard® 300mg
Rivotril® 8mg x 1
Seretiject® inh. x 2
Ventoline® inh. 0,1mg x 4
Atrovent® 20mg/dose x 8
Aerius® 10mg x 1
Zadyten® eyedrops 0,25mg x 2
Nasonex® nasal spray 50mg x 1
Losec® 40mg
Ydo-E® 100mg x 1
Pinex Forte® x 3
Fungizone® 40mg x 1
Fluoride® 0,5mg x 3

Dental history and chief complaint

Prior to my first meeting with the patient I discussed the problems I might meet with the GDP. He stressed the importance of keeping radiographic findings in mind when listening to the patient and not playing into her hands when performing the clinical examination. The patient presented with an array of pains and problems described in her own words. Her descriptions of signs and symptoms of disease in the oro-anthral regions were detailed and including medical terms. Most of it sounded plausible. That ended when she told me that her grandfather, Terje Haapasalo, was a professor with the faculty, and that he had told her that she would need major surgery. Assuming that she referred to Professor Markus Haapasalo, I knew that to be a lie. I consulted her physician. All medication was to be prescribed through her.

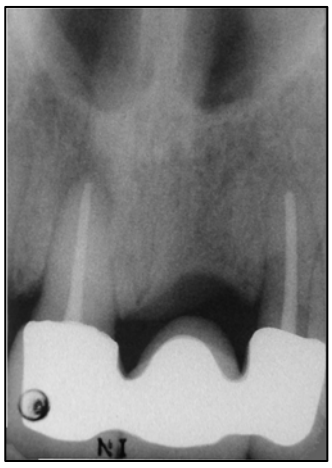
Clinical examination

The photos (figures 2 and 3) show the region from the upper right lateral incisor to the upper left lateral incisor. Tooth 12 had a mesial composite filling and signs of chronic caries in the marginal area. There was a fixed partial denture from tooth 11 to tooth 22 where the left central incisor was missing. Pocket probing depth was 5 mm mesial and distal to both abutment teeth. With the hand retracting her lip, I tried to blind her to what tooth was tested. Tooth 11 was tender to percussion and palpation. Tooth 12, 13 and 23 responded positive to electrical pulp testing.

Figure 2

Figure 3

Radiographic examination



The pre-treatment radiograph (figure 4) shows the region from the upper right central incisor to the upper left lateral incisor. There was a coronal restoration extending from 11 to 22 with the radiographic appearance of a metal-ceramic fixed bridge. A radioopaque root filling material was present in the roots of both abutment teeth. There was marginal bone loss of 3mm distal to the central incisor in conjunction with a bony pocket. The margial bone loss at the mesial aspect og tooth 22 exceeded half of the roots length. The lamina dura could be followed around the apex of the left lateral incisor. The lamina dura of tooth 11 tapered into a radiolucent area of approximately 4mm.

Figure 4

Diagnosis

Chronic marginal periodontitis
Chronic apical periodontitis of the upper right central incisor

Treatment plan

Retreatment of tooth 11, surgical intervention if needed

Treatment

First visit, April 27th 2005

She demanded premedication due to anxiety of dental treatment. When I told her that her dentist said that wasn't true, she agreed to go through with the scheduled treatment anyway. The porcelain of the prosthetic crown was negotiated with a round diamond bur in a high speed handpiece. The metal and dentine was removed by steel fissure-burs.

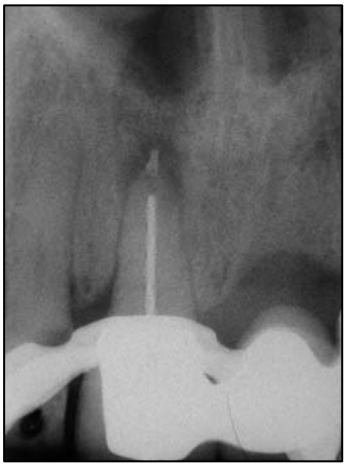


Figure 5

Some guttapercha was removed with a round bur in a slow speed handpiece before applying rubberdam and disinfecting the field of work with 5% chlorhexidine in 70% ethanol (CHX). The guttapercha was removed using H-files and microscope. The last 2mm of gp was not retrievable. The Root ZX® apexlocator indicated being in the periapical tissues at 20mm. A working-length radiograph was taken with a K-file 50/18mm (figure 5). The canal was then instrumented to a K80/18.5mm. The canal was irrigated with sodium hypochlorite (NaOCl) 1 % and EDTA 15 % followed by 5ml chlorhexidine-digluconate (CHX-2-G) left in canal for 5 minutes. A dressing of calciumhydroxide ($Ca(OH)_2$) mixed with CHX-2-G was placed in the canal with a lentulo spiral filler. The access-cavity was washed clean of dressing and the tooth was temporized with Cavit-G®.

Second visit, May 10th 2005

The patient came back complaining of symptoms, and the right upper central incisor was tender to percussion. It was then decided that apical surgery would be performed since some gp persisted in the periapical area. Rubberdam was applied and disinfected with CHX. Cavit-G® was removed with round bur in a slow speed handpiece. Irrigation was done with copious amounts of NaOCl 1 % and EDTA 17 %. Some bleeding was encountered apically when drying the canal with paper points. MTA® was packed using a K-file 80 and sterile paperpoints exposing X-rays in the procedure (figures 6 and 7) to check satisfactory radiographic density of the MTA®. A cotton pellet moistened with sterile saline was placed over the MTA® and a temporary restoration of IRM® was placed in the access cavity. Figure 7 was taken the day of surgery.

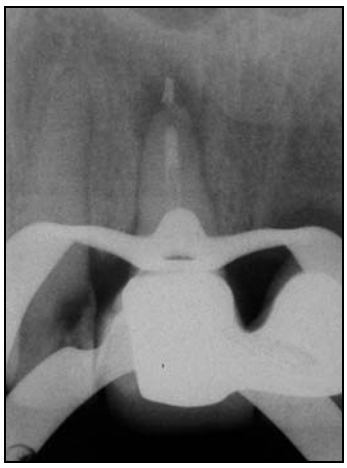


Figure 6

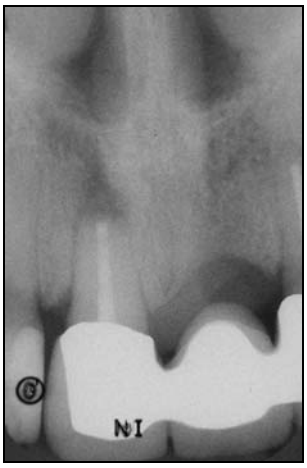


Figure 7

Third visit, May 24th 2005

Endodontic surgery:

Prior to surgery the patient's physician had been consulted. She doubled the patient's amount of Sobril® the day of surgery. She approved of Xylocain-Adrenalin® (X/A) as local anaesthetic.

The patient came premedicated with 150 mg Sobril®. The area was checked for symptoms and found still tender to palpation and percussion though blinding of the patient to what was done. Rubberdam was applied and disinfected as previously described. The IRM and cotton pellet was removed. MTA was probed and found to have hardened properly. Access cavity was acid-etched and bonded with Syntac Sprint®. One layer of Filtec flow® followed by layers of Z250® A3 were placed and light-cured. Rubberdam was removed and the occlusion checked. Three carpules of X/A were administered and the patient asked to rinse with 10ml of Corsodyl-solution for 60 seconds. A sterile field of operation was established. A marginal incision was made with a #15 scalpel-blade from 13d to 22d with a releasing incision at the canine in the first quadrant (figure 8).

Figure 8

A full thickness flap was elevated and a fenestration of the cortical bone could be seen apically of tooth 11 (figure 9). A minor osteotomy was needed to view the periapex (figure 10). Granulation tissue and the guttapercha remnant were removed using a curette. A fissure bur in a slow speed hand piece with sterile saline as coolant was used to remove the apical 2.5-3mm of the root (figure 11). An X-ray was taken to check the apicoectomy (figure 12). A copious amount of saline was used to rinse the area, the flap firmly held in position for 5 minutes before 6 sutures were placed (figure 13). The patient received an ice-pack and instructions regarding the 7 days postoperative. Instructions were also given to the person accompanying her.

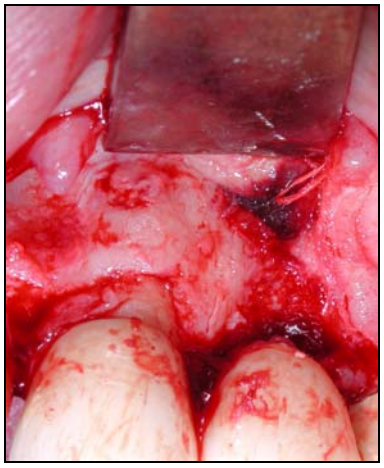


Figure 9

Figure 10

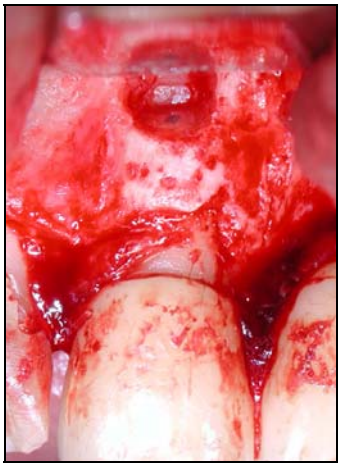


Figure 11

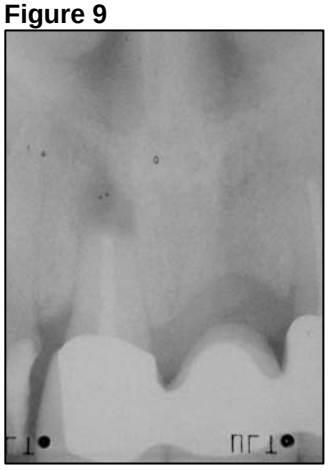


Figure 12

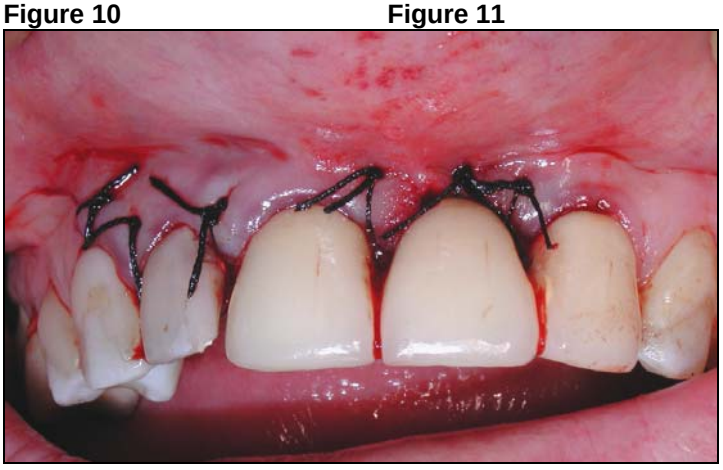


Figure 13

May 31st 2005

A nurse from the patient's institution called and cancelled the appointment because the patient felt too sick to leave the hospital.

June 7th 2005
(fourteen days after surgery)

The patient seemed out of sorts when she came to the rescheduled appointment. She told of problems sleeping because of the pain. The pain was described as continuous throbbing day and night. She complained of “sewer and pus” emanating from the area where the surgery had been performed. The clinical examination gave no proof of pus, but the area representing the flap was clearly tender to palpation. She was still on extra Pinex Forte®, Sobril® and Ibuprofen®. The sutures were removed and the patient scheduled for a 3-month follow-up.

June 14th 2005

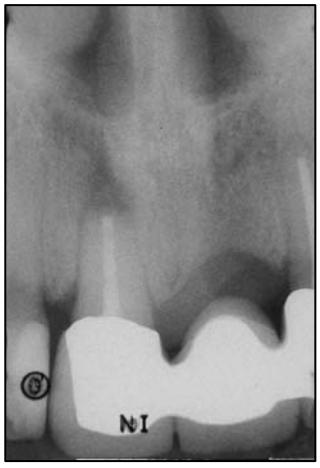
The patient’s physician called to let me know she had administered apocillin for 7 days because the patient felt ill. A blod-test told of wbc 11.7, neutrophils 7.5 and CRP 53. These were indicative of a bacterial infection. No one was able to accompany the patient to me, so the physician would look for pus or a sinus tract in the area.

Prognosis

The prognosis was assumed to be good.

Follow-up

Three months follow-up, August 31st 2005



The patient’s cell counts had proved to be a sinusitis. It had been treated with tetracyclines. The absence of symptoms was taken as a sign of improvement. A periapical radiograph was taken (figure 14).

Figure 14

One-year follow-up, March 21st 2006

The patient was recalled to evaluate the outcome of treatment. During the clinical examination she reported tenderness to percussion and palpation in the vestibulum in the area of the upper right central incisor. The condition of the soft tissue is shown (figure 15). No sinus tract was detectable intraorally or extraorally. PPD was 5mm mesial and distal to tooth 11 with bleeding on probing. She complained about stuffed nose and sinuses. The radiographic findings were consistent with periapical healing in progress. Still, the radiolucent zone towards the nasal cavity persisted (figures 16 and 17). Taking her complaints of a stuffed nose into consideration, her physician was contacted. She would look into the matter and refer the patient on if needed.

Figure 15

Figure 16

Figure 17

Discussion

Munchausen syndrome is a psychiatric disorder that causes an individual to self-inflict injury or illness or to fabricate symptoms of physical or mental illness, in order to receive medical care or hospitalization. The syndrome is characterized by a triad of features: simulated illness; pathological lying; and wandering from place to place in seek of treatment (1). Munchausen syndrome takes its name from Baron Karl Friederich von Munchausen, an 18th century German military man known for his tall tales. The disorder first appeared in psychiatric literature in the early 1950s, when it was used to describe patients who sought hospitalization by inventing symptoms and complicated medical histories, and/or inducing illness in, and injury to, themselves. Categorized as a factitious disorder (a disorder in which the physical or psychological symptoms are under voluntary control), Munchausen's syndrome seems to be motivated by a need to assume the role of a patient.

The exact cause of Munchausen syndrome is unknown. It has been theorized that Munchausen patients are motivated by a desire to be cared for, a need for attention or a need to suffer. Factors that may predispose an individual to Munchausen's include a serious illness or neglect in childhood, or an existing personality disorder. The Munchausen patient presents a wide array of physical or psychiatric symptoms, usually limited only by their medical knowledge. Many Munchausen patients are very familiar with medical terminology and symptoms. There is no clearly effective treatment for Munchausen syndrome. Extensive psychotherapy may be helpful with some Munchausen patients. If Munchausen syndrome co-exists with other mental disorders, such as a personality disorder, the underlying disorder is typically treated first (2).

There are few reports in literature describing oral presentations of the syndrome, but patients with the disorder are known to also describe symptoms mimicking facial and oral pain or inflicting injury to teeth or surrounding tissues (3). Assessment of the treatment outcome was complicated by the need to take her statements with a pinch of salt. They could not be all together overlooked either. The reason why the treatment seems to be failing, in spite of the radiographic findings to the contrary, has not been established. No report had come back from the physician when this went in the print. Possible explanations may be improper curettage of the periapical granulation tissue leaving a residual cyst with possible sinus tract to the nasal cavity. The pain may also come from mobility of the bridge caused by periodontal breakdown, or even from the patients own jiggling of the restoration. All this can only be mere speculation at present.

The microbial flora in failed cases is significantly different from those of necrotic pulps. In failed cases the flora is dominated by Gram positive facultative anaerobes. Enterococcus faecalis is found more often in failed cases, and it is not unusual to find them in mono-cultures (4, 5). Therefore, a very selective environment may exist within the root canal system that favours the survival of E. faecalis and Candida albicans (6).

The use of chlorhexidine (CHX) as a mixing vehicle for $Ca(OH)_2$ during retreatment has until now mostly been founded on in vitro studies (7). CHX possesses broad-spectrum antimicrobial activity, biocompatibility with periodontal tissues, and substantivity. Mixed with $Ca(OH)_2$, the CHX precipitates leaving <1% in aqueous solution. The in vivo effect of the combination of $Ca(OH)_2$ and CHX-digluconate (CHX-2-G) in water has been investigated. Fourty teeth were treated with $Ca(OH)_2$ either alone (control) or in

combination with CHX-2-G. The control medication disinfected 12 of 20 teeth (60%) including 2 of 4 teeth initially containing enterococci. The experimental medication disinfected 16 of 20 teeth (80%) and none of the four teeth initially harbouring enterococci showed growth. The difference between the two types of medication was not statistically significant (8). The findings can not be disregarded either, as this would likely not have been the case with a larger sample. The study indicates two important things in retreatment cases: Enterococci are susceptible to $Ca(OH)_2$ –treatment in vivo, and combined with CHX-2-G calciumhydroxide seems more potent.

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Case 20

Pain management

Patient

A fortyfive year old White female (figure 1) was referred from her general dental practitioner (GDP) to the Postgraduate Endodontic Clinic at the University of Oslo June 2005 for evaluation and possible treatment of chronic orofacial pain related to the upper right central incisor (tooth 11). She was described as a tender subject.

Figure 11

Medical history

The patient reported being of good health and that she was taking no medications. She was diagnosed as depressive by her physician, but she would not take the prescribed medication.

Dental history and chief complaint

Twenty years ago she had gotten prosthetic crowns on her four maxillary incisors. Root canal treatment had been performed on the upper left and right central and lateral incisors prior to that. Cast posts had been luted to support the crowns. Approximately five years later she had apicectomies performed to both central incisors while in Poland. She had the past few years experienced an increasing discomfort in the area of the central incisors. She also had pain from her temporo-mandibular joint (TMJ) on the right side. The discomfort had elevated to a level of now unbearable pain that in periods disturbed her quality of life. Four dentists and two doctors had tried treating her to no avail. Physiotherapy and acupuncture did not seem to help. Previous root canal treatment of tooth 47 and extraction of tooth 46 three years ago had not relieved her symptoms. The pain was said to have been at the present level for about 12 months with a change to the worse after receiving apical curettage with retrograde IRM® to 11 and 21 February 2005. The surgery had been performed at the Department for Oral Surgery and Medicine at the University of Oslo. In the period before surgery she had been given a set of exercises to relax the muscles in the head and neck. An extensive radiological examination including a CT-scan of her temporo-mandibular joints had been done, and arthrosis was found in the right TMJ. She was back for multiple consultations post surgery. She described severe pain to light touch of the skin in the area under her right eye. When the exercises didn't help she was prescribed Vival® to be taken when needed. Tegretol® was also prescribed on the suspicion of trigeminus neuralgia. She was referred to a neurologist. He said her pain was not typical for neuralgia. After a while she fell out with the personnel at the Department of Oral Surgery. She felt she was not taken seriously and not properly cared for.

Clinical examination

Figure 12

October 3rd 2005. To the clinical examination came a delicate woman at the verge of tears. She poured out her description of the problem so fast it was hard to keep track. When asked if she took any prescription drugs, she said her physician had given her NSAIDs and antidepressants. She took none of them. Her husband took out legal separation in 2004 and she had been through a divorce the first months of 2005. She felt she, in periods, couldn't properly care for her two sons, age 13 and 16, because of the grave pain. She had periods of migraine starting September 2004 and she was at present off sick from her daytime job. The pain was described as continuous grave discomfort with periods of excruciating pain. The pain was sometimes provoked by palpation of the cheek or opening her mouth wide open. There was no trouble opening her mouth, but there was deviation to the left doing so. The TMJ was tender to palpation on the right side, and clicking while opening. Musculus pterygoideus medialis was tender bilaterally and m. temporalis was tender to palpation on the right side. The pain characteristic was of burning modality. It was worst during the day but sometimes disturbing her sleep. The upper central incisors felt too long, she related that to the apical surgery performed February 2005. Tooth 11 also felt loose. The pain had worsened after surgery, and the painful periods were more frequent. She described severe pain to light touch of the skin in the area under her right eye. There was redness of skin in the same area, and the right eye was red. Scar-tissue was observed in the buccal mucosa overlying the incisors. The central and lateral incisors all had prosthetic crowns with signs of adjustments having been made in occlusion palatally. Of the upper anterior teeth all save 13 were negative to Endo-Ice®. Tooth 11 was tender to percussion in vertical direction. There was tenderness to palpation of 11. Moving the crown of tooth 11 in distal direction was clearly painful to the patient. Pocket probing depth (PPD) was within normal limits (WNL) for all incisors of the upper jaw. Occlusion was checked with no apparent signs of supra-occlusion for tooth 11.

Radiographic examination

The radiographs (figures 3, 4, 5 and 6) show the region from the upper right canine to the upper left canine. The right canine was superimposed on the first premolar, but the periapical area was visible with a traceable lamina dura. 12, 11, 21 and 22 had coronal restorations with radioopacity in line with posts and metal-ceramic crowns. 12 had a radioopaque root filling material resembling guttapercha. The PDL-space was visible around the periapex. 11 and 21 had root filling material with radioopacity comparable to IRM. The anatomy in the periapical area of the central incisors was indicative of previous apicectomies. The lamina dura was traceable around the root of tooth 11 in figure 5 and around tooth 21 in figure 6. There was a radiolucent zone resembling a void of 3mm between the post and root filling material in tooth 22. The PDL was visible all the way around the root.

Figure 13

Figure 14 ortho-radial

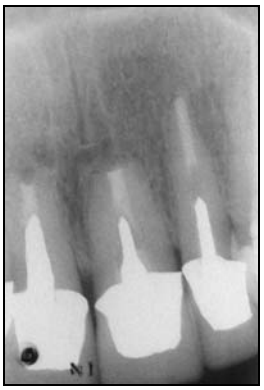

Figure 15 disto-radial

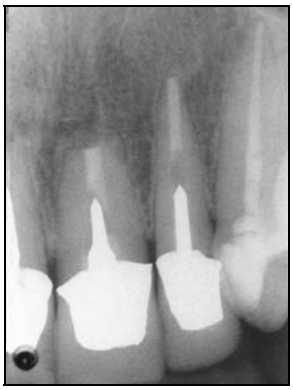

Figure 16

Diagnosis

TMJ-dysfunction
Tentative diagnosis of vertical root fracture of tooth 11
Some of her described problems could indicate neuropathic pain.

Treatment plan

To establish a relation to a prosthodontist and maybe an oral surgeon to replace tooth 11 if it was lost. Remove the crown and post of tooth 11 to look for a vertical root fracture.

Treatment

The patient wanted all treatment to be performed at the Faculty of Dentistry. Progression of treatment and planning thereof was slow due to the fact that I was unable to find a Postgraduate student or employee at the Department of Oral Surgery willing to treat her, or a Postgraduate student at the Prosthodontics Department with the time to assist her. In the process I spent hours with her on the phone trying to comfort her. She cried a lot. I contacted her physician and he could inform me that she refused taking the prescribed medication meant to treat her depressive state.

October 20th 2005

The patient was back for impressions of both jaws prior to removal of the crown and post of tooth 11. She could not hold back her tears. She was on Ibuprofen 600mg x 4 but the pain was unchanged. She had contacted a dentist with special interest in TMJ-disorders and occlusal adjustments. She wanted to postpone the planned treatment until after consulting him.

October 27th 2005

I talked to the dentist that would look into her occlusion. He had found anterior discus displacement on the right side. They had made a new stabilisation splint. He said anterior discus displacement lead to anterior movement of the lower jaw that sometimes led to loss of the buccal bone plate that might create mobility of the involved tooth. After relieving the patient of her symptoms by the stabilisation splint he recommended total oral rehabilitation. He recommended a vitamin-B screening to rule out burning-mouth syndrome from vitamin-B deficiency. I contacted her physician. Vitamin-B-screening had already been done and her vitamin-B levels were within normal limits.

October 31st 2005

The patient could tell that her symptoms were alleviated from the stabilisation splint. Her spirit was better and she did not cry on the telephone as she usually did. She would call me if she needed more assistance.

December 14th 2005

The patient called. She was no longer happy with the treatment she had gotten from the dentist she had consulted about her TMJ-disorder. She said she had paid a lot of money for a brief consultation, and that he was arrogant. She would discontinue the treatment there even though she felt some improvement. I would arrange for a prosthodontist being a private practitioner to accept treating her before removing the crown and post.

January 16th 2006

A prosthodontist at her homeplace was willing to take on the challenge of giving her teeth she could be content with. He said the teeth adjacent to 11 were not fit for a fixed partial denture. If the right central incisor was lost this would imply an implant. She had started with antidepressants (Vival®). She was a totally different person. Her spirit was up and she had dressed in a nice manner. Impressions were made of both jaws. PPD of tooth 11 was within normal limits. It was still painful when the crown was jiggled. A new pariapical X-ray was taken (figure 7). No apparent changes since October were seen.

Figure 17

The crown of tooth 11 was negotiated with a diamond-coated bur (figures 8 and 9). The crown was removed exposing a separately luted post (figure 10). The coronal part of the post was reduced with a bur (figure 11), and the radicular part came out immediately with no aid from ultrasound or other devices. The root was isolated with rubberdam and disinfection was done with 5 mg/ml chlorhexidine in 70% ethanol. The root was inspected for signs of a fracture aided by the Zeiss Opmi Pico® microscope and methylene-blue. No clear sign of root-fracture was found. A radiograph was taken (figure 12). Some of the apically placed IRM was removed (figure 13). The tooth was irrigated with NaOCl 1%, EDTA 15%. Chlorhexidine-digluconate was left in the canal for five minutes. The canal was dried and new IRM® placed on top of the residing IRM®. An X-ray was taken (figure 14). Ca(OH)2-slurry placed with a lentulo-spiral and a Cavit-G® placed well into the canal-orifice. A temporary crown was made of ProTemp® and luted with Temp-bond® (figure 15). It was adjusted in occlusion. When biting down she immediately said the occlusion felt more right.

Figure 8

Figure 9

Figure 10

Figure 11

Figure 12

Figure 13

Figure 14

Figure 15

January 24th 2005

The patient was back for a check-up. She felt much better and had an appointment with the prosthodontist coming up. She was no longer off from work. She was happy finally seeing a change for the better.

Prognosis

The prognosis of the endodontic treatment was deemed uncertain. Despite the negative clinical findings, I still wouldn't rule out vertical root fracture as a differential diagnosis. The part of the orofacial pain assumed to be neuropathic was not expected to go away but may be held in check by medication.

Discussion

The aetiology of TMD is obscure. At best it can be stated that it is multifactorial. Stress and occlusal disharmonies have been mentioned in this respect. Different "schools" have presented their interpretation of the aetiology and a plausible treatment related to that. Known treatment alternatives are physiotherapy (home exercises) (1), analgesics (2), splint therapy (3) and occlusal adjustments (4, 5). The actual evidence for the efficacy of physiotherapy is weak because of the very limited number of randomized clinical trials (RCT) available in literature (1). It is non-invasive and reversible. There seems to be no evidence in literature to support the common use of analgesics in TMD (2). Regarding splint-therapy and occlusal adjustments, all review articles based on RCT state that there is no evidence that these treatment modalities cure or prevent TMD (3, 4)

Neuropathic pain is a disorder affecting up to 5 % of the adult population (6). After pulpal amputation 3-5 % of the patients develop neuropathic pain in the form of phantom pain (7). The diagnosis is often preceded by numerous dental procedures without altering the pain sensation. The high prevalence of orofacial pain of dental origin and the dramatic similarities between neuropathic orofacial pain and odontogenic and other pathologic pains in the region frequently lead to incorrect diagnoses and, more importantly, inappropriate treatments that are frequently invasive and irreversible. The records of patients presenting with neuropathic pain at a university pain clinic were reviewed to gain insight into dental factors as they related to the etiology, presentation, diagnosis, and management of neuropathic pain of the trigeminal system. Relative to etiology, the records review revealed that most onsets were associated with a specific dental treatment or odontogenic symptom that resulted in a dental diagnosis or treatment. Initial treatment modalities that either caused the pain or were used to address painful symptoms commonly included replacement of restorations, endodontic therapy, apicectomy, extraction, splint therapy, and occlusal equilibration (8). It is not uncommon for patients with orofacial neuropathic pain to have erythema and swelling of the affected area accompanied by stuffed nose and secretion of tears. These objective findings are easily confused with signs of inflammation (7). It is not uncommon that every intervention gives a short period, 14 days or more, with less pain. After that the pain is often back in full, or even worse (7).

It seems that tricyclic antidepressants (TCAs) are effective treatments for the treatment of neuropathic pain (9). The patient in the present case had been through a similar circle of treatment before the antidepressants. Antidepressants are connected with shame and resignation. Most people would, as she, be reluctant to accept that the problem is in the brain and not the body.

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