

Endodontic treatment of large periradicular lesions with and without cutaneous sinus tract: Report of two cases and review

Gul Celik Unal, Bulem Ureyen Kaya

Suleyman Demirel University, Faculty of Dentistry, Department of Endodontic, Isparta, Turkey.

Cilde fistülizasyonu olan ve olmayan geniş periradikuler lezyonların cerrahi olmayan endodontik tedavisi: İki olgu sunumu ve derleme

Özet

Bu vaka raporunun amacı cilde fistülizasyonu olan ve olmayan geniş periradikuler lezyonu olan iki olguyu rapor etmek ve tanı ve tedavi prensiplerini sunarak, diğer rapor edilmiş vakaları özetlemektir. Birinci vaka mandibular kesici dişlerinden kaynaklanan çene ucu cilt fistülizasyonu olan 23 yaşında bir kadındı. İki yıldır mevcut olan lezyonda ara sıra pürülan bir sıvı drene olmaktadır. İkinci vaka ise radyografik olarak sol mandibular ikinci premolar diş kökünün apikalinde sınırları belirsiz geniş bir radyolüsent lezyonu olan 46 yaşında bir kadındı. Her iki vakada da, cerrahi olmayan endodontik tedaviyi takiben lezyonlar tamamen iyileşti. İzleyen 3, 6 ve 12 aylarda kontrolleri yapıldı. Birinci vakada fistül iki hafta içinde küçük çukur şeklinde bir skarla iyileşirken, her iki vakada da 1 yılın sonunda radyografik olarak kemik iyileşmesi izlendi.

Anahtar kelimeler: Geniş periapikal lezyon, diş kaynaklı cilt fistülü, cerrahi olmayan endodontik tedavi, taşkın kalsiyum hidroksit

Abstract

The aim of this case report was to report two cases of large periradicular lesions with and without cutaneous sinus tract and to present diagnostic guidelines, treatment guidelines and to summarize other reported cases. The first case was about a 23 year-old woman with a draining cutaneous sinus tract at her chin, which originated from mandibular incisors. She had the lesion for two years and since then it had been occasionally draining purulent fluid. The second case was about a 46 year-old woman with a large non-demarcated radiolucent lesion located in apical portion of the mandibular left second premolar root. The patient had a slight discomfort in the premolar region. In both cases, the lesions completely resolved in the following non-surgical endodontic treatments. Recall visits were performed in 3,6 month and 12-month period. In the first case, the sinus tract completely healed within two weeks with a small dimple-like scar, in both cases bone healing was recorded radiographically after 1 period.

Key words: large periapical lesion, odontogenic cutaneous sinus tract, non-surgical treatment, extruded calcium hydroxide

Yazışma Adresi/Corresponding: Gül Çelik Ünal
Süleyman Demirel Üniversitesi Diş Hekimliği Fakültesi
Endodonti AD, Doğu Yerleşkesi İSPARTA
E-mail adresi: celikunalgul@yahoo.com

Tel: 0246 211 8778

Müracaat tarihi: 16.03.2011
Kabul tarihi: 12.07.2011

Introduction

The majority of periradicular lesions associated with teeth without root canal treatment are caused by pulpal inflammation and/or degeneration. The development of these lesions is largely due to the passage of irritational products, including microorganisms, from root canal space (1). Little correlation exists between

the clinical signs and symptoms and duration of the lesions when compared to the histopathologic findings (2). Periapical lesions classified into 5 main groups: Acute apical periodontitis, chronic apical periodontitis, condensing osteitis, acute apical abscess and chronic apical abscess (3).

In chronic inflammations the local inflammatory destructive process, are severally asymptomatic and their progress is slow through the cancellous alveolar bone along the path of least resistance. They may perforate the thin cortical plate and form a subperiosteal abscess (4,5). This asymptomatic locus of infection may spread into the surrounding soft tissues, its progress is limited only by muscle attachments and fascial planes (6). A subperiosteal abscess, which has established drainage through a sinus tract, is termed chronic apical abscess or suppurative apical periodontitis. The abscess has 'burrowed' through the bone and soft tissues to form a sinus tract stoma on the oral mucosae or sometimes on the skin of the face. Chronic apical periodontitis may also drain through periodontium into the sulcus and mimic a periodontal abscess or pocket (3).

Chronically draining cutaneous sinus tracts are frequently misdiagnosed and incorrectly treated. The differential diagnosis should include a traumatic lesion, local skin injection (carbuncle, infected pilar or epidermoid cyst) (4), foreign body pyogenic granuloma, chronic tuberculous lesion, osteomyelitis, actinomycosis, and the gummo of tertiary syphilis (7). Less frequently, developmental defects of branchial cleft or thyroglossal duct origin, suppurativelymphadentitis (7), and salivary gland duct fistula (8), dacryocystitis and neoplasm (4) should also be taken into account the following clinical cases present non-surgical endodontic treatment of large periradicular lesions with and with utaneous sinus tract.

Case I

A 23-year-old woman with 18-month history of a chronically draining cutaneous lesion, approximately 134 mm², associated with an erythematous base located on the chin was presented. The patient noted periodic drainage of yellowish, awful, sticky fluid on her chin (Figure 1).



Figure 1: Cutaneous sinus tract on the chin region.

She noted that she fell down the stairs 3 years ago and extracted her mandibular right central incisor at a dental clinic one year ago. She stated that when the lesion started to discharge pus she visited a surgeon who prescribed antibiotics. However she stated that she kept on complaining about the continuity of the pus drainage.

She was systemically healthy and, the medical history was not contributory to this complaint. A regional head and neck examination demonstrated palpable lymphadenopathy of the submental node. Physical examination revealed palpable facial nodule with drainage.

A clinical intraoral examination demonstrated missing of tooth # 41 and darkening and discoloration of the teeth # 31 and # 42. The teeth were not mobile. She exhibited sensitivity to percussion. The

results of vitality tests by an electric pulp tester were negative. A periapical dental radiograph revealed diffuse radiolucency at the apices of the mandibular incisors (Figure 2).



Figure 2: The periapical radiograph of the lesion before treatment.

Non-surgical endodontic therapies of the # 31 and # 42 were planned. The access cavity was prepared without application of local anaesthesia. When the canals were opened, a suppurative fluid (pale yellowish) belongs to # 31 was drained through the canal. When the drainage ceased, working lengths were determined according to the radiographic method known as the Ingle Method (9) and the canals were prepared using a step-back technique until an apical preparation size #40 was achieved. During the preparation, 5.25% NaOCl irrigating solution was used between the files. (Figure 3).

A powder of Ca(OH)_2 (Kalsin, Aktu Tic., Bornova, Izmir) mixed with glycerine was packed into the root canals by using lentulo filler. The access cavities were sealed with a temporary filling material (Coltosol, Coltene, Altstätten, Switzerland). Following a periapical dental radiograph showed that Ca(OH)_2 passed beyond the apical foramen into the radiolucent area at the tooth # 42 (Figure 4).



Figure 3: Determination of working length.



Figure 4: Overextension of Ca(OH)_2

Only one week after the non-surgical endodontic treatment, the teeth were asymptomatic, and the clinical examination showed that drainage from the sinus tract had been stopped (Figure 5).

Three week after the first visit, the root canals were obturated with Sealapex (Kerr, Romulus, Italy) and guttapercha using lateral condensation technique (Figure 6).

The sinus tract healed in the next two weeks without additional intervention leaving only slightly hyper-pigmented of the skin and small dimple-like scar (Figure 7).



Figure 5: No drainage from the sinus tract at one week after the treatment

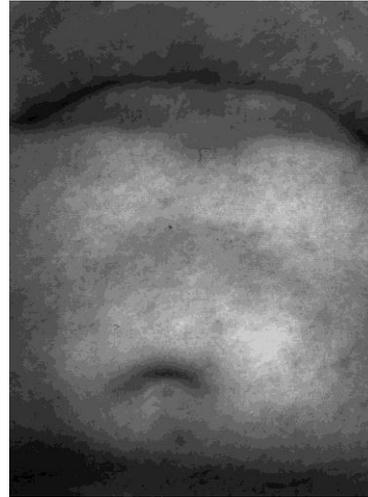


Figure 7: Healed cutaneous sinus tract at three week after the treatment



Figure 6: Filling of the root canal



Figure 8: The periapical radiograph at the end of the first year

At the end of the first year, clinical examinations showed no sensitivity to percussion or palpation, and the soft tissues were healthy. Radiographic examination showed the progressive process of healing (Figure 8).

Case II

A 46-year-old woman attended to the endodontics clinic with a slight discomfort at the left mandibular premolar region. There was no intraoral or extraoral swelling or pain at any time. She was systemically healthy. A regional head and neck examination demonstrated no palpable lymphadenopathy of the

submental node. Intraoral examination of the left mandibular mucosae revealed no abscess formation, discoloration and fistulisation. A good amalgam restoration on the occlusal and distal surfaces of the tooth # 36 and a composite Class V restoration on the buccal face of the tooth # 35 were observed. There was inadequate marginal integrity and discoloration on the marginal surfaces of the composite restoration on the tooth # 35. The teeth were not tender to percussion or painful when biting. The second premolar tooth # 35 did not respond to electrical pulp testing. Radiographic examination demonstrated a large (approximately 206 mm²) diffuse, non-demarcated radiolucent

lesion located through the root of the tooth # 35 (Figure 9,10).

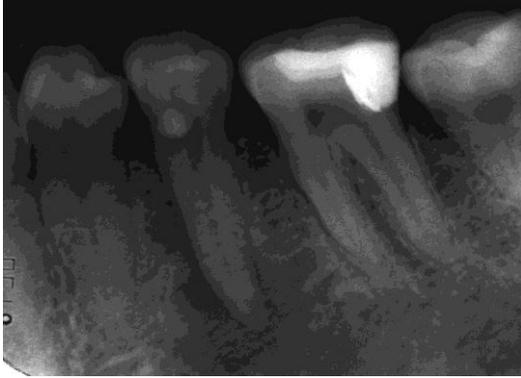


Figure 9: The periapical radiograph of the tooth #35 before endodontic treatment



Figure 10: Panoramic radiograph of the tooth #35 before endodontic treatment

Treatment procedure was similar in both cases. At the first visit, the access cavity of the tooth # 35 was prepared. Working length was determined and biomechanical preparation root canals were performed with copious 5,25% sodium hypochlorite irrigation. A powder of $\text{Ca}(\text{OH})_2$ mixed with glycerine was packed into the root canals by using lentulo. The class V composite restoration was replaced. After three weeks, the tooth was asymptomatic, and the root canal was obturated with Sealapex (Kerr, Romulus, Italy) and gutta-percha using lateral condensation technique. Radiographic examination showed the progressive process of healing in three months (Figure 11).

The periapical radiograph taken at first year revealed complete disappearance of the radiolucent lesion (Figure 12).



Figure 11: The periapical radiograph after three months.



Figure 12: The periapical radiograph at the end of the first year.

Discussion

Microbial, mechanical or chemical irritation of pulpal and periradicular tissues results in inflammation. Dental caries and microorganisms in canals constitute the main sources of microbial irritants of the dental pulp and the periradicular tissues (3).

The second case exhibited the periradicular lesion has a composite restoration with inadequate marginal integrity and discoloration. This microbial irritation of the pulp and subsequent necrosis is the etiology of the periradicular inflammation. Pulpal necrosis is a frequent sequel of trauma and if microbial infection occurs, this will result in the development of a periradicular lesion (8). The impact injury could be the reason of the periradicular lesion in the first case.

There are a number of studies that point out a correlation between radiographic lesions that increase in size and the incidence of a periradicular cyst (10-12). Many practitioners have an indelible impression that whenever a periradicular lesion is radiographically large, and/or exhibits a radio-opaque border, a periradicular cyst is unquestionably present and surgical removal is essential (13). Simon (14) has outlined two different types of periradicular cysts: a) a 'bay' cyst which is an epithelial walling off of irritants originating from the root canal and in which the lumen of the cyst has direct communication with the root canal system b) a 'true' cyst which is a three-dimensional, epithelium lined cavity where there is no connection between cyst lumen and the root canal system.

Treatment options to manage large periapical lesions range from nonsurgical root canal treatment and/or apical surgery to extraction (15-18). Non-surgical treatment of the majority of root canals will result in complete repair of the largest periradicular lesions, including the bay cyst and in some cases the true cyst (1). In the presented cases the periradicular lesions were 134 mm² and 206 mm² but lacked radio-opaque border. Both of the periradicular lesions healed completely with non-surgical endodontic treatment.

Current literature reviews note 3 findings that are consistent with dental sinus tract (19). 1- Palpable facial nodule with or without drainage 2- Dental radiographs demonstrate a periapical pathosis 3- as often noted palpable introoral cord.

Cutaneous sinus tracts result from the spreading of the infection into the surrounding soft tissues. The major factors influencing the spread of cutaneous sinus

tracts are bacterial virulence, body resistance of the patient, lower resistance of the connective tissue in the facial spaces, and the position of the apex of the affected tooth relative to muscle attachments (4, 20). The mandibular muscle attachments posteriorly and facial anteriorly determine the sites of the drainage of such sinus tracts (6).

Only a few studies have dealt with prevalence of sinus tracts. Mortensen et. al. (21) investigated 1600 teeth with periapical lesions; 136 (9,0%) teeth had sinus tracts while Gupta & Hasselgren (22) found 29 (18,1%) of 160 infected teeth had sinus tracts.

Cutaneous sinus tracts of dental origin most commonly arise on the chin or lower jaw. Cioffiet al. (6) reported that most of the cutaneous sinus tracts are associated with mandibular teeth (80% mandibular teeth, 40% mandibular anterior teeth). Pubmed was searched with odontogenic cutaneous sinus tract and extraoral fistula keywords from 1986 to 2011. The examination of 42 sinus tract cases between 1986-2011 years showed 81% involved mandibular, of which 62% was anterior teeth (Table 1).

Symptoms and/or location may not suggest a dental pain and the stoma of the sinus tract may not to be adjacent to the involved teeth (23). Nonvital may appear sound and may be completely asymptomatic (44). The sinus tract prevents swelling or pain from pressure build-up because it provides drainage of the odontogenic primary site (24). So that patients, assuming the cutaneous sinus tract to be unrelated to dental infection, often seek treatment from a dermatologist or from their family physician. In literature between 1986 and 2011, about 64% of the patients with sinus

Table 1. Cutaneous Sinus Tract Cases after 1986 (f: female, m: man, a.b.: antibiotic, rct: root canal treatment, ppd: persistent pus drainage)

Authors	Age/sex	Area	Reason of infection	Initial operator	Initial therapy	Reason of attending to dentist	Treatment
Cioffi et al. 1986 (6)	20/f	submental area	41	physician	excision, a.b, topical steroid	-	rct
	19/m	posterior mandible	46	-	-	-	rct
	18/m	posterior mandible	46	-	-	-	rct
Bianchi et al. 1986 (23)	-	midline chin	residual odontogenic cyst left lower teeth	dentist	24,25,26 extraction a.b.	ppd	surgical extraction
	58/m	anterior neck		physician		ppd	
McWalter et al. 1988 (24)	18/f	right mandible	46	dentist	-	ppd	rct
Held et al. 1989 (25)	37/f	chin	42	physicians	incision, a.b.	ppd	rct
	52/m	right mandible	47	physician	psoriasis therapy	-	extraction
Hodges et al. 1989 (26)	23/m	chin	32	physician	pyogenic granuloma	mild sensitivity swelling	rct
Foster et al. 1992 (19)	30/m	right mandible	45	dentist			rct
Al-Kandari et al. 1993 (27)	32/m	chin	43	physician	excision	ppd	rct
Caliskan et al. 1995 (16)	15/f	left cheek	36	-	-	ppd	rct
	32/f	chin	31,41	physician	incision, drainage, a.b	referred to dermatologist	rct
	22/m	chin	31	physician	incision, drainage	-	rct
Urbani&Tintinelli 1996 (28)	32/f	chin	31,41	physician	a.b.	ppd	rct
	65/f	chin	42	physician	excision, a.b, topical steroid	-	rct
Cheung et al. 1996 (29)	28/m	on left alar area	22	physician	drainage, cauterisation	ppd	rct, apicalotomy
Tidwell et al. 1997 (30)	37/m	chin	33	physician	mgs ₄ , a.b.	pain	rct
Chan et al. 1997 (31)	37/m	on left cheek	36	physician	excision, a.b	ppd	extraction
Nakamura et al. 1999 (32)	22/f	left cheek	36	plastic surgeon	-	ppd	rct
Palacio et al. 1999 (33)	79/m	nasolabial fold area	-	physician	a.b	-	
	33/f	on ala of the nose	22	physician	a.b	tumorlike growth	rct
Bradford RJ et al. 1999 (34)	23/f	chin	lower incisor	physician	cauterization	ppd	rct
	45/f	right chin	43	dentist	-	-	rct
	20/m	left mandible	36	physician	a.b		rct
	59/m	on ala of the nose	23	physician	a.b, steroid, excision	ppd	rct
Hwang et al. 2000 (35)	46/m	on the left nasolabial area	remnant root	physician	incision, drainage, excision	ppd	extraction
Gulec et al. 2001 (36)	47/f	chin	41	physician	-	-	extraction
Mukerji& Jones 2002 (37)	38/m	right cheek	46	-	-	-	extraction
Cohenca et al. 2003 (38)	19/m	left cheek	third molar	surgeon	a.b., drainage	ppd	extraction
	15/m	chin	23,24,25	-	-	swelling	rct
	24/m	right cheek	46	-	-	ppd	rct
Mittal & Gupta 2004 (39)	25/m	left cheek	26	physician	excision	ppd	rct
	10/f	right cheek	46	dentist	pulpectomi	ppd	rct
Sheehan DJ et al. 2005 (40)	44/f	rightcheek	22,23	physician	a.b	ppd	extraction
Qazi SS et al. 2006 (41)	38/f	leftcheek	25	dentist			rct
Soares JA et al. 2007 (42)	17/m	chin	41	dentist	Rct		
	35/m	chin	42	dentist	Rct	ppd	retreatment
	21/m	chin	41	dentist	Rct		
Pasternak-Júnior B et al. 2009 (43)	37/f	submental area	32	dermatological surgeon	surgery	no healing	rct
	18/m	right mandible	46	surgeon	surgery	no healing	rct
	29/m	leftmandible	35	dermatological surgeon	surgery	no healing	rct

tracts in the skin of dental origin sought for treatment for their physician (plastic surgeon, dermatologist etc.) (Table 1).

In the past, the treatment decision is generally extraction for tooth having large lesions with or without cutaneous sinus tract. Cioffiet *al* (6) reported that the lesions relating with sinus tract, 77% of the 137 cases were treated by extraction, 10 % by surgical root canal therapy, and 10% by conservative non-surgical root canal therapy. Increased knowledge of the root canal anatomy and the availability of better endodontics, instruments and materials mean that severely diseased teeth can be saved by conservative endodontic treatment. Literature reviews related to sinus tract cases between 1986-2011 years, all of the cases were treated with root canal treatment except nonrestorable due to caries, remnant and vertical root fracture (Table 1).

Earlier studies have reported that sinus tracts are lined with epithelial tissue, providing the basis for the surgical treatment (45,46). However, there is a misconception that the tract is lined with epithelium. Grossman (13) and Bender & Seltzer (47) reported an absence of epithelial tissues and they also described that such tracts are generally lined with granulation tissue. There is no evidence that on epithelium-lined sinus tract will not heal after endodontic therapy (46). It has been established that only root canal treatment of the tooth with a necrotic pulp can eliminate the need for surgical treatment (6, 16, 25 and 32). After treatment, the sinus tract usually heals in 5 to 15 days, with a small, dimplike scar that over the next few months will become inconspicuous (48). Cosmetic surgical treatment may be required later if the area heals with a residual tract that results in cutaneous retraction or dimpling.

A cutaneousodontogenic sinus tract is a localized entity and is not an indication for antibiotics. Systemic antibiotic administration is neither necessary nor recommended in patients with a cutaneous

odontogenic sinus tract who have an intact immune system, no sign or symptoms of systemic involvement, and no other systemic condition that requires prophylactic antibiotic coverage (6, 30).

Complete debridement and irrigation of the root canal during the first appointment, followed by the application of a calcium hydroxide dressing for 1 week or more (49, 50). Obturation of the root canal is then performed at the second or a later appointment (50). The length of time that Ca(OH)₂ is left in the root canal can affect its effectiveness depending on its ability to rapidly disassociate into hydroxyl ions and calcium ions. In the present research, the medication was left in the root canal for 21 days because some clinical situations have indicated the presence of this medication in the root canal for several days, well beyond a reasonable period of penetration and disassociation into hydroxyl ions and calcium ions (51).

Clinically, though satisfactory antiseptic results have been obtained with Ca(OH)₂-based pastes for up to 7 days (50), studies of hydroxide ion diffusion through dental structure suggest that the minimum time should be 2-3 weeks (52). This is required to reach the threshold alkalinity in order to inactivate or eliminate bacteria and fungi remaining in the root canal system and possibly periapical biofilm microorganisms as well (53, 54). Calcium hydroxide dressing of root canals was performed at the first appointment and root canals were obturated after 3 weeks in the presented cases.

In the literature, there are number of reports relating the beneficial effects of calcium hydroxide preparation (49-55). Calcium hydroxide in a paste form is a widely used medicament in endodontics because of its high alkalinity, antibacterial activity and ability to induce hard tissue deposition (50, 56-60). It has been established that overfilling of root canals with calcium hydroxide has been advocated, because of its direct effect on inflamed tissue and epithelial cystic linings

also beneficial for osseoinductive reasons (17, 57, 58, and 61). Sahli (62) suggested that the necrotising ability of calcium hydroxide might destroy any epithelium present and this function allowing a connective tissue invagination into the lesion with ultimate healing. However, it may be accidentally extruded during filling procedures like in the first case. In the first case, complete resorption of the paste did not occur although the periapical radiolucency around the paste disappeared. It was considered that this could be due to the barium sulphate content of the paste, which was added to provide opacity. This makes radiographic interpretation of osseous heal more difficultly (63). In the first presented case, periapical repair took more than 6 months to be completed, this finding confirms the findings of Vernieks& Messer (64) who suggested that extrusion of calcium hydroxide beyond the apex may be a factor for the lack of early healing of periapical lesions.

In conclusion; odontogenic cutaneous sinus tracts are still misdiagnosed by dentists and physicans. Large periradicular lesions with or without sinus tracts may heal with non-surgical root canal treatment by means of calcium hydroxide dressing. In addition, although many case reports of dental originated extraoral sinus tracts to heal only by non-surgical root canal treatment were reported in literature, these cases are still termed as a complicated therapy by many dentists. Therefore, postgraduate education on such cases should be highlighted.

References

1. Maalouf EM, Gutmann JL. Biological perspectives on the non-surgical endodontic management of periradicularpathosis. *Int Endod J* 1994; 27: 154-162.
2. Morse DR, Seltzer S, Sinai I, Biron G. Endodontic classification. *J Am Dent Assoc* 1977; 94: 685-689.
3. Torabinejad M, Walton RE. Principles and Practice of Endodontics. 2nd ed. Philadelphia: W. B. Saunders Co., 1996.29-51.
4. Kaban LB. Draining skin lesions of dental origin: the path of spread of chronic odontogenic infection. *PlastReconstrSurg*1980; 66:711-717.
5. Donohue WB, Maisonneuve C. Sinus tracts of odontogenic origin in the face and neck. *J Can Dent Assoc*1984;1:199-102.
6. Cioffi GA, Terezhalmly GT, Parlette HL. Cutaneous draining sinus tract: an odontogenicetiology. *J Am AcadDermatol* 1986; 14:94-100.
7. Scott MJ Jr, Scott MJ Sr. Cutaneousodontogenic sinus.J *Am AcadDermatol* 1980; 2:521-24.
8. Sundqvist G. Bacteriological studies of necrotic dental pulps. *Odontological Dissertations no: 7 Umea, Sweden: Umea University* 1976.
9. Ingle JI. Endodonticinstrumentsandinstrumentation. *DentClin North Am* 1957;11:805.
10. Wais FT. Significance of findings following biopsy and histologic study of 100 periapical lesions. *Oral Surg Oral Med Oral Pathol* 1958; 11: 650-653.
11. Linenberg WB, Waldron CA, Delaune GF. A clinical, roentgenographic, and histopathologic evaluation of periapical lesions. *Oral Surg Oral Med Oral Pathol* 1964;7: 467-472.
12. Lalonde ER, Luebker. The frequency and distribution of periapical cysts and granuloms. *Oral Surg Oral Med Oral Pathol*1968;25: 861-8.
13. Grossman LI. Endodontic Practise. 10th ed. Philedelpia. PA. USA: Lea &Febiger , 1981, 106.
14. Simon JH. Incidence of periapical cysts in relation to the root canal. *J Endod* 1980; 6: 845-848.
15. Walker TL, Davis MS. Treatment of large periapical lesions using cannulization through the involved teeth. *J Endod* 1984; 10: 215-220.

16. Caliskan MK, Turkun M. Periapical repair and apical closure of a pulpless tooth using calcium hydroxide. *Oral Surg Oral Med Oral Pathol* 1997;84,:683-687.
17. De Moor RJ, De Witte AM. Periapical lesions accidentally filled with calcium hydroxide. *Int Endod J.* 2002; 35: 946-958.
18. Oztan MD. Endodontic treatment of teeth associated with a large periapical lesion. *Int Endod J*2002 ;35 :73-8.
19. Foster KH, Primack PD, Kulild JC. Odontogenic cutaneous sinus tract. *J Endod*1992; 18: 304-306.
20. Azaz B, Taicher S. Facial sinus tracts of dental origin in children. *J Dent Child* 1976;43:167-171.
21. Mortensen H, Winther JE, Birn H. Periapical granulomas and cysts. An investigation of 1,600 cases. *Scand J Dent Res* 1970;78:241-50.
22. Gupta R, Hasselgren G. Prevalence of odontogenic sinus tracts in patients referred for endodontic therapy. *J Endod* 2003;29:798-800.
23. Bianchi MA, Rosenberg SL, Murphy JB. Cervical necrosis and sinus tract formation secondary to a dentoalveolar infection: report of a case. *J Oral MaxillofacSurg* 1986;44: 894-896.
24. McWalter GM, Alexander JB, del Rio CE, Knott JW. Cutaneous sinus tracts of dental etiology. *Oral Surg Oral Med Oral Pathol* 1988; 66:608-614.
25. Held JL, Yunakov MJ, Barber RJ, Grossman ME. Cutaneous sinus of dental origin: a diagnosis requiring clinical and radiologic correlation. *Cutis* 1989; 43: 22-24.
26. Hodges TP, Cohen DA, Deck D. Odontogenic sinus tracts. *AmFam Physician* 1989; 40:113-116.
27. Al-Kandari AM, Al-Quoud OA, Ben-Naji A, Gnanasekhar JD. Cutaneous sinus tracts of dental origin to the chin and cheek: case reports. *Quintessence Int*1993;24:729-733
28. Urbani CE, Tintinelli R. Patent odontogenic sinus tract draining to the midline of the submental region: report of a case. *J Dermatol* 1996; 23:284-286.
29. Cheung LK, Samman N, Lee E. An unusual facial sinus. *Aust Dent J* 1996;41: 6-8.
30. Tidwell E, Jenkins JD, Ellis CD, Hutson B, Cederberg RA. Cutaneous odontogenic sinus tract to the chin: a case report. *Int Endod J* 1997;30:352-355.
31. Chan CP, Chang SH, Huang CC, Wu SK, Huang SK. Cutaneous sinus tract caused by vertical root fracture. *J Endod*1997;23:593-595.
32. Nakamura Y, Hirayama K, Hossain M, Matsumoto K. A case of an odontogenic cutaneous sinus tract. *Int Endod J* 1999;32: 328-331.
33. Palacio EJ, Altemus DA, Christensen ED, Sorensen GW. Unusual recurrent facial lesion. *Arch Dermatol* 1999;135:595-598.
34. Bradford RJ, Nijole AR, Joseph EV. Diagnosis and treatment of cutaneous facial sinus tracts of dental origin. *J Am Dent Assoc* 1999;130: 832-836.
35. Hwang K, Kim CW, Lee SI. Cutaneous sinus tract from remaining tooth fragment of edentulous maxilla. *J CraniofacSurg* 2000;11: 254-257.
36. Gulec AT, Seckin D, Bulut S, Sarfakoglu E. Cutaneous sinus tract of dental origin. *Int J Dermatol*2001; 40:650-652.
37. Mukerji R, Jones DC. Facial sinus of dental origin: a case report. *Dent Update* 2002;29: 170-171.
38. Cohenca N, Karni S, Rotstein I. Extraoral sinus tract misdiagnosed as an endodontic lesion. *J Endod* 2003;29:841-3.
39. Mittal N, Gupta P. Management of extra oral sinus cases: a clinical dilemma. *J Endod* 2004; 30:541-547
40. Sheehan DJ, Potter BJ, Davis LS. Cutaneous draining sinus tract of odontogenic origin: unusual presentation of a challenging diagnosis. *South Med J.* 2005 Feb;98(2):250-252.
41. Qazi SS, Manzoor MA, Qureshi R, Arjumand B, Hussain SM, Afridi Z. Nonsurgical endodontic management of

- cutaneously draining odontogenic sinus. *J Ayub Med Coll Abbottabad*. 2006 Apr-Jun;18(2):88-89.
42. Soares JA, de Carvalho FB, Pappen FG, Araújo GS, de Pontes Lima RK, Rodrigues VM, de Toledo Leonardo R. Conservative treatment of patients with periapical lesions associated with extraoral sinus tracts. *Aust Endod J*. 2007 Dec;33(3):131-135.
43. Pasternak-Júnior B, Teixeira CS, Silva-Sousa YT, Sousa-Neto MD. Diagnosis and treatment of odontogenic cutaneous sinus tracts of endodontic origin: three case studies. *IntEndod J*. 2009 Mar;42(3):271-276.
44. Caliskan MK, Sen BH, Ozinel MA. Treatment of extraoral sinus tracts from traumatized teeth with apical periodontitis. *Endod& Dental Traumatol*1995;11:115-120.
45. Thoma KH. *Oral Surgery*, 4th ed. St Louis, USA: Mosby. 1963; 733.
46. Harrison JW, Larson WJ. The epithelized oral sinus tract. *Oral Surg Oral Med Oral Pathol*1976;42: 511-517.
47. Bender IB, Selzer S. The oral fistulas: its diagnosis and treatment. *Oral Surg Oral Med, Oral Pathol*1961;14:1367-1376.
48. Spear KL, Sheridan PJ, Perry HO. Sinus tract to the chin and jaw of dental origin. *J Am AcadDermatol*1983;8:486-492.
49. Byström A, Claesson R, Sandqvist G. The antibacterial effect of camphoratedparamonochlorophenol, camphorated and calcium hydroxide in the treatment of infected root canals. *Endod& Dental Traumatol*1985;1:170-175.
50. Sjögren U, Figdor D, Spanberg L, Sundqvist G. The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing. *Int Endod J*1991;24:119-125.
51. Lambrianidis T, Kosti E, Boutsoukis C, Mazinis M. Removefficacy of variouscalciumhydroxide/chlorhexidinemedicamentsfromtherootcanal. *Int Endod J* 2006;39:55-61.
52. Siqueira Jr JF, Lopes HP. Mechanisms of antimicrobial activity of calcium hydroxide: a critical review. *IntEndod J* 1999;32:361-369.
53. Soares JA, Leonardo MR, TanomaruFilho M, Silva LAB, Ito IY. Effect of biomechanical preparation and calcium hydroxide pastes on the anti-sepsis of root canal systems in dogs. *J Appl Oral Science* 2005;12:110-117.
54. Leonardo MR, Silva LAB, Rossi MA, Ito IY, Bonifácio KC. Evaluation biofilm and microorganisms on the external root surface in human teeth. *J Endod* 2002;28:815-818.
55. Stanley HR. Pulp capping: conserving the dental pulp--can it be done? Is it worth it? *Oral Surg Oral Med Oral Pathol*1989;68: 628-39.
56. Webber R.T. Traumatic Injuries and the expanded endodontic role of calcium hydroxide. In: Gerstein H. *Techniques In Clinical Endodontics*, Philadelphia, Saunders., 1983,238-39.
57. Cvek M. Treatment of non-vital permanent incisors with calcium hydroxide. I. Follow-up of periapical repair and apical closure of immature roots. *Odontol Revy* 1972;23:27-44.
58. Tronstad L, Andreasen JO, Hasselgren G, Kristerson L, Riis I. Ph changes in dental tissues after root canal filling with calcium hydroxide. *J Endod* 1981;7:17-21.
59. Ghose LJ, Baghdady VS, Hikmat YM. Apexification of immature apices of pulpless permanent anterior teeth with calcium hydroxide. *J Endod*1987;13: 285-90.
60. Sonat B, Dalat D, Gunhan O. Periapical tissue reaction to root fillings with Sealapex. *Int Endod J*1990;23:46-52.
61. Orstavik D, Kerekes K, Molven O. Effects of extensive apical reaming and calcium hydroxide dressing on bacterial infection during treatment of apical periodontitis: a pilot study. *Int Endod J*1991;24:1-7.
62. Rotstein I, Friedman S, Katz J. Apical closure of mature molar roots with

the use of calcium hydroxide. Oral Surg Oral Med Oral Pathol 1990;70:656-60.

63. Sahli CC. L'hydroxide de calcium dans le traitement endodontique des grandes lésions périapicales. Rev Fr Endodon 1988;7: 45-51.

64. Vernieks AA, Messer LB. Calcium hydroxide induced healing of periapical lesions: a study of 78 non-vital teeth. J Br Endod Soc 1978;11:61-69.