Clinical Article

CASE-CONTROL

Radiographic Changes Associated with Pulp Infection in Primary Incisor Roots and in Their Developing Permanent Dental Follicles

Malka Ashkenazi, DMD[1] • Efrat Hershkovitz, DMD[2] • Limor Afek, DMD[3]

Abstract: ***Purpose:*** *Early diagnosis and treatment of pulp infection in primary incisors are important for preventing systemic infection and damage to the permanent tooth bud. The purpose of this retrospective case-control study was to identify radiographic changes associated with pulp infection in primary incisor roots and their developing permanent dental follicles.* ***Methods:*** *Oblique occlusal radiographs of 102 primary maxillary incisors with sinus tracts were compared to 390 radiographs of incisors from healthy same-age children.* ***Results:*** *Compared with controls, teeth with sinus tracts had more inflammatory root resorption (odds ratio [OR]=50.7, P<.001), pulp pathosis (OR=13.0, P<.001), deep caries (OR=3.9, P<.001), past traumatic injury (OR=3.4, P<.001), enlarged dental sac (OR=10.8, P<.001), loss of lamina dura of the dental sac (OR=27.5, P<.001), and increased radiolucency of the dental sac of the corresponding permanent tooth bud (OR=19.5, P<.001). In multivariate logistic regression, 2 variables were associated with radiographic changes in the primary root (inflammatory resorption, P<.001; and pulp pathosis, P=.004). Two variables associated with changes in the permanent tooth bud (loss of lamina-dura and increased radiolucency) were independently associated with the presence of a sinus tract, P<.001.* ***Conclusion:*** *Deep caries, inflammatory resorption, pulp pathosis, loss of lamina-dura, and increased radiolucency of the dental sac were statistically associated with presence of a sinus tract.* *(Pediatr Dent 2012;34:397-402) Received February 4, 2010 / Last Revision June 11, 2011 / Accepted September 30, 2011*

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Pulp infection of the primary maxillary incisors is very common and develops primarily as a consequence of pulp necrosis caused by deep caries or traumatic injury.[1,2] Appearance of a sinus tract is definitive evidence of pulp infection.[3,4] The presence of dental caries in primary molars was found to be associated with pulp infection and a sinus tract.[5] Such association has not been documented in primary incisors, however, despite the high prevalence of sinus tracts in carious primary incisors.

Traumatic injury, usually to the maxillary incisors,[6] is another common etiology of pulp infection. Traumatic injuries have been reported in up to 30% of primary teeth.[7] In young children, most of these comprise luxation injuries,[8] which are often associated with nonreversible damage to the blood supply of the pulp, resulting in pulp necrosis.[8] Notwithstanding, pulp necrosis can also develop following less complicated traumatic injuries, such as complicated or uncomplicated crown fracture.[8]

Pulp infection sometimes spreads rapidly through the bone and periosteum and disseminates through the blood toward more distant structures, such as intracranial, retropharyngeal, pulmonary pleura, and valves of the heart (endocarditis).[9,10] Moreover, it also may spread to the orbit and lead to orbital abscess or loss of vision.[11,12] Untreated decayed teeth were shown to be 10 times more prone to cause sepsis in 5-year-old children than were teeth with fillings and no decay.[3]

Pulp infection in primary teeth may also induce alterations in tooth eruption and in the development of successor permanent tooth buds, resulting in: hypoplasia; morphological alteration on the dental crown; or total arrest of radicular formation.[1,13-16]

Drs. [1]Ashkenazi, [2]Hershkovitz, and [3]Afek are in private practices, all in Tel-Aviv, Israel. Correspond with Dr. Ashkenazi at malka.ashkenazi@gmail.com

Developmental disturbances are characterized mainly by color changes, with or without defects in the enamel surface.[1,13-16] These disturbances result from the close proximity between the primary tooth apex and the permanent dental bud and the absence of hard tissue between them.[14]

Matsumiya observed destruction of the enamel or interference with enamel formation in 28 (~23%) of 120 dogs with infected teeth.[15] Valderhaug exposed the pulp of 52 primary teeth in monkeys. After 360 days, 45 teeth developed periapical inflammation. Pathological changes in the hard tissue of permanent successor tooth buds were detected in 16 teeth with periapical inflammation.[13] McCormick and Filostrat evaluated 452 teeth of first- and second-grade children, among whom 60% had radiographic evidence of periapical bone destruction. Damage to permanent successors was detected in 19 (23%) of 82 severely abscessed teeth that were not treated until exfoliation vs 4% of teeth without evidence of dental abscess.[16]

Primary teeth with sterile pulp necrosis do not necessitate immediate intervention, but rather long-term clinical and radiographic follow-up for early detection of pulp infection and consequent prevention and minimization of complications. The establishment of radiographic criteria for diagnosis of pulp infection prior to the appearance of a sinus tract is of prime importance. Little is known, however, about radiographic changes that occur in the primary root or dental follicle in response to chronic pulp infection of primary teeth.[6] Such information can assist clinicians in diagnosing and treating early pulp infection before such complications as sinus tracts or cellulitis arise.

The purpose of this study was to characterize radiographic changes in primary incisor roots and in their developing permanent dental follicles subsequent to chronic infection in primary incisors.

Methods

This was a retrospective case-control study. The Ethics Committee of Tel Aviv University approved this study. Data were accessed from clinical and radiographic records of children examined between 2005 and 2008 by students from the Department of Pediatric Dentistry of Tel Aviv University, Tel Aviv, Israel, and by 2 pediatric dentists in private practice.

Files of patients were screened for written documentation of presence of a sinus tract in the maxillary incisors. Radiographs of involved teeth, taken from the same day of the clinical diagnosis, were subsequently obtained. Only radiographs that contained data about the primary root and its corresponding developing permanent dental buds were included in the present study. We identified 102 oblique occlusal radiographs of primary maxillary incisors with sinus tracts from 85 children: 49 boys and 36 girls (17 children had 2 teeth with sinus tracts); 61 incisors were from males and 41 from females. Of them, 97 were central incisors and 5 were lateral incisors.

For the control group, we screened files of patients with no documentation of a sinus tract with oblique occlusal radiographs of primary maxillary incisors. The circumstances for taking a radiograph of maxillary incisors with no evidence of presence of a sinus tract varied. These circumstances included: as a routine procedure to exclude presence of impacted anterior supernumerary teeth; as a routine procedure for treating children under general anesthesia; to evaluate the extent of dental caries; and for routine follow-up after traumatic injury.

We established a control group comprising 390 oblique occlusal radiographs of healthy "equally old" incisors from 156 children: 65 girls and 91 boys. Of these, 239 were of central incisors and 151 lateral incisors. We note that several control teeth were included from the experimental group from children with adjacent healthy incisors. In 23 children, 67 incisors (41 centrals and 26 laterals) were adjacent to teeth with sinus tracts and served as controls. In 10 children, 1 adjacent incisor served as the control; in 5 children, 2 adjacent teeth served as controls; and in 8 children, 3 teeth were suitable as controls.

The maximum ages for inclusion in the study were 5 years for central incisors and 6.5 years for lateral incisors. These ages, which precede the ages for estimated normal eruption by approximately 2.5 to 3 years, were determined to exclude radiographic changes associated with the exfoliation process. Moreover, only teeth with minimal or no physiological resorption were included in the study (ie, the length of the contralateral healthy root was twice that of the crown). Physiologic resorption was defined by the following conditions: irregularity of the outline of the primary incisor root; extension of resorption comparable to that of the healthy contralateral/adjacent primary incisors; and an intact lamina dura without radiolucency in the adjacent bone. By contrast, surface (not physiologic) resorption was defined by disproportional resorption of the primary tooth root relative to the dental age of the adjusted healthy incisors and by a lack of resorption in the bone adjacent to the intact lamina dura (Figures 1-3).

Dental files containing a high quality oblique occlusal radiograph of central or lateral incisors were analyzed according to a structured form. Demographic and clinical data were collected, including: age; gender; tooth location; date of traumatic injury (when documented); presence of radiographic pathology; and clinical pathology. Deep proximal caries was defined by the lesion exceeding half of the distance between the dentino-enamel junction (**DEJ**) and the pulp border. Clinical records of presence of a sinus tract were collected as well as records of crown discoloration, and crown fracture indicated previous traumatic injury. Radiographs were classified as having no pathological changes, including obliteration of the dental pulp, or as having pathological changes in the roots of the primary teeth: presence of surface root resorption; inflammatory external resorption (associated with radiolucency in the adjacent bone); internal root resorption; and cessation of pulp chamber development.

In addition, radiographs were analyzed for changes in the dental sac of the corresponding developing permanent dental follicle. The criteria used to determine pathology in the dental sac were: enlargement by more than 2 mm compared to the adjacent intact tooth[2,27]; radiolucency of the dental sac that was increased more than that of the pulp of the primary incisor; and loss of or incomplete lamina dura (loss of the compact bone lining the border of the dental sac of the developing permanent tooth bud; Figures 1-6.

The X-rays were evaluated by all 3 investigators separately using a back light and magnifying glass (3x). Initially, all 3 investigators analyzed a sample of 25 oblique occlusal radiographs to set criteria for the study, prior to correlation to clinical findings. Later, at 2 separate meetings, all 3 investigators evaluated the radiographs according to the established classification while being blinded to information regarding presence or absence of a sinus tract. They discussed lesions about which they disagreed until they reached a unanimous decision.

Intrarater reliability. In a pilot study, 25 to 48 oblique occlusal radiographs were evaluated by 3 authors over a 1-week interval or longer. The mean intraexaminer reliability for scoring of loss of lamina dura was approximately 93%, for increased radiolucency 98%, for increased size of the dental sac 98%, for inflammatory resorption 98%, and approximately 99% for surface resorption.

Statistical analysis. Student *t* test and chi-square tests were used to calculate the statistical significance of differences between continuous and nominal variables, respectively. For chi-square tests of dichotomous variables, the continuity correction was applied. Analyses were carried out using the standard statistical package (SPSS 10.0, SPSS Inc, Chicago, Ill., USA). Odds ratios (**ORs**) were calculated to estimate the probability of a tooth developing a sinus tract after a traumatic injury or experiencing deep caries or when there was radiographic evidence of: inflammatory resorption; internal pulp resorption; and/or cessation of pulp chamber development, enlarged dental sac, increased radiolucency of the dental sac, and loss of lamina dura.

Interactions between the presence of a sinus tract and radiographic changes were evaluated by 2-way analysis of variance (**ANOVA**). We performed 2 multivariate analyses to investigate the variables associated with radiograph changes in the primary root and in the dental sac. The level of significance was set at 5%.

Results

Patient characteristics. The age range of the children was 1.5 to 6.6 years old (mean=3.4±1.0 years old). The distribution of sinus tracts, according to children's age, is presented in Figure 7. There was no statistically significant difference between the mean ages of the 85 children with sinus tracts (4.3±0.9 years old; range=1.5-6.0 years old) and the 156 children without sinus tracts (4.5±1.0 years old; range=1.6-6.6 years old) by

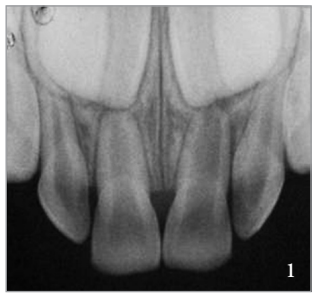












Figure 1. An oblique occlusal radiograph of maxillary incisors showing a healthy dental sac of the developing permanent central incisors and cessation of development of the pulp chamber of the primary left central incisor (necrosis).

Figure 2. An oblique occlusal radiograph of maxillary incisors showing a healthy dental sac of the permanent teeth buds of the central incisors and surface root-resorption of the primary central incisors.

Figure 3. An oblique occlusal radiograph of maxillary incisors showing an enlarged dental sac (>2 mm compared to the adjacent intact tooth) of a developing permanent central incisor and surface root resorption of the primary central incisors.

Figure 4. An oblique occlusal radiograph of maxillary incisors showing increased size (>2 mm compared to the adjacent intact tooth) and radiolucency (more than the pulp chamber) of the dental sac of the permanent central incisors while the lamina dura of the dental sac is intact.

Figure 5. An oblique occlusal radiograph of maxillary incisors showing external inflammatory root resorption of the primary central incisors with both increased size and radiolucency of the dental sac of the permanent central incisors, but with intact lamina dura.

Figure 6. An oblique occlusal radiograph of maxillary incisors showing primary left central and left lateral incisors with inflammatory root resorption and both increased size and radiolucency of the dental sac as well as loss of the lamina dura of their corresponding permanent central incisors. The root of the primary right incisor and its corresponding permanent dental bud are within the normal range.

t test. Sinus tract was significantly more common in central than in lateral incisors (98/337 vs 4/155, respectively; *P*<.001—by chi-square test). No correlation was found between the presence of a sinus tract and gender.

Clinical examination (Table 1). A previous injury was documented in 180 (~37%) teeth. Of them, 61 (~34%) also had sinus tracts. Teeth subjected to a traumatic injury had significantly more sinus tracts (61/180, ~34%) than teeth not subjected to traumatic injuries (41/312, ~13%; *P*<.001, OR=3.4).

No caries, including caries extending up to half the distance between the DEJ and the pulp border, was detected in 278 and 108 incisors, respectively. Dental caries not exceeding beyond the DEJ was detected in 18 incisors. Of them, 2 (~11%) had sinus tracts and 16 (~89%) did not. Dental caries extending up to half the distance between the DEJ and the pulp border were detected in 47 incisors. Of them, 8 (17%) had sinus tracts and 39 (83%) did not. Deep proximal caries (the lesion exceeded beyond the halfway point between the DEJ and the pulp border) was detected in 106 incisors. Sinus tract was significantly more prevalent in teeth with deep caries (49/106, ~46%) than in teeth without caries or with proximal caries extending up to half the distance between the DEJ and the pulp border (57/386, ~15%; *P*<.001; OR=3.9).

Sinus tract was significantly more prevalent in teeth with both deep proximal caries lesions (the lesion exceeded beyond the halfway point between the DEJ and the pulp border) and evidence of previous traumatic injury (26/46 ~57%) than in teeth with only deep carious lesions (31/107, 29%) or only with evidence of previous traumatic injury alone (35/134, ~26%; *P*<.001).

Radiographic findings related to primary incisor roots (Table 1). External inflammatory root resorption was diagnosed radiographically in 131 teeth. Teeth with external inflammatory root resorption had significantly more sinus tracts (88/131, ~67%) than teeth without external inflammatory root resorption (14/361, ~4%; *P*<.001; OR=50.7).

Presence of pulp necrosis, diagnosed by cessation of pulp chamber development or by internal inflammatory resorption, were diagnosed radiographically in 7 and 29 teeth, respectively. Fifty-five teeth showed radiographic evidence of pulp obliteration. Sinus tracts were significantly more prevalent in teeth with pulp necrosis or internal inflammatory root resorption than in teeth without any sign of pulp pathosis or with pulp obliteration (26/36, ~72% vs 76/456, ~17%, respectively; *P*<.001; OR=13.0).

Surface root resorption was diagnosed radiographically in 23 teeth. Of them, 3 had sinus tracts. No significant differences were detected between the presence of surface root resorption and the presence of sinus tracts.

An enlarged dental sac was diagnosed radiographically in 189 teeth. Sinus tract was diagnosed radiographically in significantly more teeth with enlarged dental sac than in teeth without (82/189, ~43% and 20/303, ~7%, respectively; *P*<.001, OR=10.8).

Increased radiolucency of the dental sac of corresponding permanent teeth was diagnosed radiographically in 183 teeth. Teeth radiographically diagnosed with increased radiolucency of the dental sac had significantly more sinus tracts than teeth without (88/183, ~48% and 14/309, ~5%, respectively; *P*<.001; OR=19.5).

Loss or discontinuity of the lamina dura of the dental sac of the corresponding permanent tooth bud was diagnosed radiographically in 145 teeth. Teeth in which there was loss or discontinuity of the lamina dura of the dental sac of the

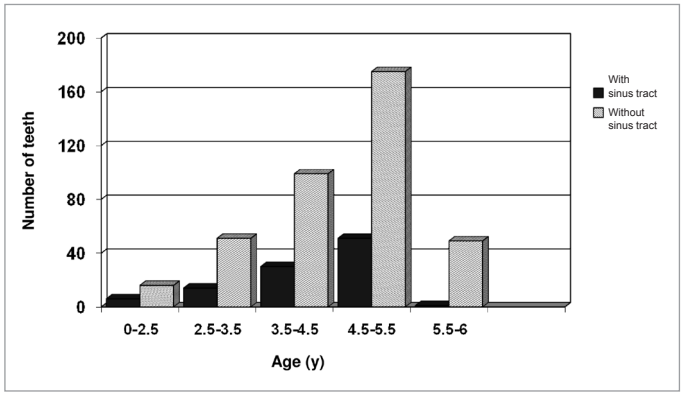


Figure 7. Distribution of teeth with and without sinus tract according to child's age. In some children, several incisors were analyzed.

corresponding permanent tooth bud had significantly more sinus tracts than teeth without (85/145, ~59% and 17/347, ~5%, respectively; *P*<.001; OR=27.5).

Multivariate regression analysis. Multivariate analysis revealed 3 of the variables related to the primary tooth to be independently correlated with the presence of a sinus tract: deep caries; external inflammatory root resorption; and pulp necrosis with or without internal inflammatory root resorption.

In a separate multivariate analysis, 2 of the variables associated with the dental sac of the developing permanent teeth were found to be independently correlated with the presence of a sinus tract: increased radiolucency and loss of the lamina dura of the dental sac (Table 2).

Discussion

Timely treatment of a pulp infection is crucial to minimize pain, severe systemic infections, and damage to secondary successors.[3,6,9,11-13,15,16,20] Postponing treatment of infected teeth may decrease success rates of root canal treatment in infected primary incisors.[2]

The currently available clinical signs for infection development (presence of a sinus tract, mobility, and sensitivity of the tooth to percussion) are usually associated with late diagnosis. Moreover, it has been shown that pulp infection can exist without clinical evidence of a sinus tract,[26] and mobility or sensitivity to percussion can develop after a new traumatic injury and not just as a consequence of pulp infection.

Radiography is another commonly used tool for diagnosing infection in dentistry and in medicine.[33] A literature search (PubMed®) for studies containing the keywords "pulp infection," "dental sac," "infection," and "radiographic signs," however, yielded only 3 references. The only one that was relevant discussed the clinical interpretation of enlargement of the dental sac.[27] Neither did we find data related to our findings in oral pathology, oral radiology, pediatric dentistry, or dental traumatology textbooks,[6,28-32] except for one reference to expansion of the follicle of permanent teeth following dental trauma.[32]

The present study showed that deep caries, inflammatory resorption (resorption of root and adjacent alveolar bone), and pulp pathosis were related to the presence of pulp infection. Traumatic injuries, however, were not significantly correlated statistically with infection in logistic regression analysis. This may be due to our inability to classify traumatic injuries according to their severity, in contrast to dental caries. Interestingly, although the relation between a sinus tract and deep caries is obvious to most clinicians, the present study is one of the few to show correlation between deep caries and the development of a sinus tract in children.

The positive relation between inflammatory resorption and pulp infection agrees with previous studies, which showed that pulp infection in permanent teeth extending into the periodontal ligament space depends on the total microbial content and virulence of the micro-organism involved and duration of the infection.[29] If the infection is not treated, the periapical pathosis becomes radiographically visible.[3,6,18,19,22-24] These findings are also in accordance with an evaluated study of full-mouth radiographs of 616 individuals, which showed dental caries to be associated with an increased risk for apical periodontitis.[25]

In primary incisors, pulp infection was mainly expressed by inflammatory root resorption and less by apical periodontitis, as is usually expressed in permanent incisors. This may be due to the fact that, in primary roots, the cementum is usually damaged by the physiological root resorption process, even without traumatic injury.[6]

In the present study, we evaluated 3 radiographic changes associated with the developing permanent dental bud in the presence of infection, namely: enlargement of the dental sac; increased radiolucency; and loss of the lamina dura. The criterion "enlargement of the dental sac more than 2 mm relative to the adjacent teeth" was established by Rocha and Cardoso.[2] In the absence of established criteria in previously published articles or textbooks, the other 2 criteria were determined according to

Table 1. ASSOCIATION OF DIFFERENT VALUES WITH THE PRESENCE OF A SINUS TRACT USING UNIVARIATE ANALYSIS

Variables			No. of teeth with sinus tract		Odds ratio of a sinus tract (95% confidence interval)	Range	*P*-value
			No	Yes			
Associated with medical history	Traumatic injury	No	271	41	3.4	2.2-5.3	<.001
		Yes	119	61			
Associated with clinical examination of the teeth	Deep caries	No	333	53	3.9	2.5-6.1	<.001
		Yes	57	49			
Associated with the affected root of the primary tooth	External inflammatory resorption	No	347	14	50.7	26.6-96.9	<.001
		Yes	43	88			
	Internal pulp resorption and/or cessation of pulp chamber development	No	380	76	13.0	6.0-28.1	<.001
		Yes	10	26			
Associated with the dental bud of corresponding permanent tooth bud	Enlarged dental sac	No	283	20	10.8	6.3-18.6	<.001
		Yes	107	82			
	Increased radiolucency of the dental sac	No	295	14	19.5	10.6-35.9	<.001
		Yes	95	88			
	Loss of lamina dura	No	330	17	27.5	15.3-49.6	<.001
		Yes	60	85			

Table 2. SIGNIFICANT VARIABLES ASSOCIATED WITH PRESENCE OF A SINUS TRACT BY MULTIVARIATE LOGISTIC REGRESSION ANALYSIS*

	Variables	Odd ratio of a sinus tract (95% confidence interval)	Range	*P*-value
A	External inflammatory resorption	37.3	19.2-72.7	<.001
	Internal pulp resorption and/or cessation of pulp chamber development	4.2	1.6-11.3	.004
	Deep caries	2.9	1.5-5.4	<.001
B	Increased radiolucency	4.1	1.9-9.1	<.001
	Loss of lamina dura	10.7	5.1-22.6	<.001

* Variables associated with the infected primary molar (A) and with the dental bud of the corresponding permanent tooth (B).

our own previous clinical experience and were based on the knowledge that infection is usually associated with bone resorption. According to the present study, radiographic evidence of loss or discontinuity of the lamina dura or increase in radiolucency of the dental sac of the developing tooth bud indicate a presence of infection. Therefore, such teeth should be treated by extraction or by endodontic treatment, but should not be followed without intervention.

The treatment options depend on several factors, such as patient cooperation, restorability, financial costs, and whether the infection penetrated the dental sac of the corresponding permanent tooth bud. Another factor to be considered is that root canal treatment in infected primary incisors with periapical radiolucency was shown to induce 2 to 3 times more developmental defects in their successor teeth than in healthy teeth or infected teeth treated by extraction.[35] Further clinical studies should be conducted to evaluate the effect of the duration of these radiographic changes in the dental sac on the type and severity of developmental disturbances in erupted permanent teeth.

In the current study, multivariate analysis did not show an increase in the size of the dental sac to correlate with the presence of infection. These results concur with previous studies showing that a dental sac greater than 2 mm presented in: 72% of the injured teeth; all types of coronal discoloration; occlusal trauma caused by thumb-sucking; obliteration; and endodontically treated teeth.[2,27] Moreover, a sinus tract was present in only 21% of the teeth with expanded dental sacs.[34] Accordingly, an enlarged dental sac may indicate presence of breakdown products from necrotic, uninfected pulps. Though this does not necessitate operative intervention, it may require continuous clinical and radiographic follow-up.

The main limitations of the present study were due to its retrospective design. For one, oblique occlusal radiographs were not all exposed using the same radiographic conditions. Nevertheless, radiographic changes were evaluated in comparison to adjacent unaffected teeth that were obviously exposed to the same radiographic conditions. In addition, teeth with deep buccal caries but without proximal lesions were not always included in the group of deep caries.

Conclusions

Based on this study's results, the following conclusions can be made:

1. Primary incisors with radiographic evidence of internal or external inflammatory root resorption are statistically associated with presence of a sinus tract, and therefore should be treated immediately by root canal treatment or by extraction;
2. Primary incisors in which there is loss or discontinuity of the lamina dura and increased radiolucency of the dental sac of the corresponding permanent tooth bud are statistically associated with presence of a sinus tract, and therefore should be treated immediately by root canal treatment or by extraction;
3. Primary incisors with deep caries or with cessation of pulp chamber due to pulp necrosis, but without any other pathologic signs, are at high risk for infection, and therefore should be followed by periodic radiographic examination to exclude development of infection; and
4. Primary incisors in which there is enlargement of the dental sac of the corresponding permanent tooth do not necessitate operative intervention, but should be followed by periodic radiographic examination to exclude development of infection.

References

1. Johnson BR, Remeikis NA, Van Cura JE. Diagnosis and treatment of cutaneous facial sinus tracts of dental origin. J Am Dent Assoc 1999;130:832-6.
2. Rocha MJ, Cardoso M. Federal University of Santa Catarina endodontic treatment of traumatized primary teeth: Part 2. Dent Traumatol 2004;20:314-26.
3. Pine CM, Harris RV, Burnside G, Merrett MC. An investigation of the relationship between untreated decayed teeth and dental sepsis in 5-year-old children. Br Dent J 2006;200:45-7.
4. Sadeghi S, Dibaei M. Prevalence of odontogenic sinus tracts in 728 endodontically treated teeth. Med Oral Patol Oral Cir Bucal 2010 [Epub ahead of print].
5. Ledezma-Rasillo G, Flores-Reyes H, Gonzalez-Amaro AM, Garrocho-Rangel A, Ruiz-Rodriguez Mdel S, Pozos-Guillen AJ. Identification of cultivable micro-organisms from primary teeth with necrotic pulps. J Clin Pediatr Dent 2010; 34:329-33.
6. Andreasen JO, Andreasen FM, Andersson L, eds. Textbook and Color Atlas of Traumatic Injuries to the Teeth. 4th ed. Copenhagen, Denmark: Blackwell Munksgaard; 2007:66, 180, 457.
7. Andreasen JO. Challenges in clinical dental traumatology. Endod Dent Traumatol 1985;1:45-55.
8. Borum MK, Andreasen JO. Sequelae of trauma to primary maxillary incisors: Complications in the primary dentition. Endod Dent Traumatol 1998;14:31-44.
9. Jiménez Y, Bagán JV, Murillo J, Poveda R. Odontogenic infections, complications, systemic manifestations. Med Oral Patol Oral Cir Bucal 2004;9(suppl):139-47.
10. Brook I. Brain abscess in children: Microbiology and management. J Child Neurol 1995;10:283-8.

11. Kim IK, Kim JR, Jang KS, Moon YS, Park SW. Orbital abscess from an odontogenic infection. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:e1-6.
12. Rosen D, Ardekian L, Abu el-Naaj I, Fischer D, Peled M, Laufer D. Orbital infection arising from a primary tooth: A case report. Int J Paediatr Dent 2000;10:237-9.
13. Valderhaug J. Periapical inflammation in primary teeth and its effect on the permanent successors. Int J Oral Surg 1974;3:171-82.
14. Andreasen JO. The influence of traumatic intrusion of primary teeth on their permanent successors: A radiographic and histologic study in monkeys. Int J Oral Surg 1976;5:207-19.
15. Matsumiya S. Experimental pathological study on the effect of treatment of infected root canals in the deciduous tooth on growth of the permanent tooth germ. Int Dent J 1968;18:546-59.
16. McCormick J, Filostrat DJ. Injury to the teeth of succession by abscess of temporary teeth. J Dent Child 1967;34: 501-4.
17. Eidelman E, Ulmansky M, Michaeli Y. Histopathology of the pulp in primary incisors with deep dentinal caries. Pediatr Dent 1992;14:372-5.
18. Bystrom A, Happonen RP, Sjogren U, Sundqvist G. Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis. Endod Dent Traumatol 1987;3:58-63.
19. Korzen BH, Krakow AA, Green DB. Pulpal and periapical tissue responses in conventional and monoinfected gnotobiotic rats. Oral Surg Oral Med Oral Pathol 1974;37: 783-802.
20. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. J Clin Periodontol 2002;29:663-71.
21. Langeland K. Tissue response to dental caries. Endod Dent Traumatol 1987;3:149-71.
22. Rocha CT, Rossi MA, Leonardo MR, Rocha LB, Nelson-Filho P, Silva LA. Biofilm on the apical region of roots in primary teeth with vital and necrotic pulps with or without radiographically evident apical pathosis. Int Endod J 2008;41:664-9.
23. Dwyer TG, Torabinejad M. Radiographic and histologic evaluation of the effect of endotoxin on the periapical tissues of the cat. J Endod 1981;7:31-5.
24. Balto K, White R, Mueller R, Stashenko P. A mouse model of inflammatory root resorption induced by pulpal infection. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;93:461-8.
25. Kirkevang LL, Vaeth M, Hörsted-Bindslev P, Bahrami G, Wenzel A. Risk factors for developing apical periodontitis in a general population. Int Endod J 2007;40:290-9.
26. Sasson LM, Fidel R, Faveri M, Fidel S, Figueiredo L, Feres M. Microbiological evaluation of primary endodontic infections in teeth with and without sinus tract. Int Endod J 2008;41:508-15.
27. Holan G. Development of clinical and radiographic signs associated with dark discolored primary incisors following traumatic injuries: A prospective controlled study. Dent Traumatol 2004;20:276-87.
28. Stewart RE, Barber TK, Troutman KC, Wei Shy. Pediatric Dentistry: Scientific foundations and clinical practice. 1st ed. St. Louis, Mo: Mosby Company; 1982:918, 946, 951-52.
29. Regezi JA, Sciubba JJ, Jordan RCK. Oral Pathology: Clinical Pathologic Correlations. 4th ed. St. Louis, Mo: WB Saunders Company; 2003:1-74, 309-318, 330.
30. White SC, Pharoah MJ. Oral Radiology: Principles and Interpretation. 6th ed. St. Louis, Mo: Mosby Company; 2009:256-69, 325-8.
31. McDonald RE, Avery DR, Dean JA. Dentistry for the Child and Adolescent. 8th ed. St. Louis, Mo: Mosby Company; 2000:105-6, 390-410, 470-85.
32. Pinkham, Casamassimo PS, Fields HW, McTigue DJ, Nowak AJ. Pediatric Dentistry: Infancy Through Adolescence. Philadelphia, Pa: Saunders Company; 1988:204, 237-54.
33. Miyashita N, Sugiu T, Kawai Y, et al. Radiographic features of mycoplasma pneumoniae pneumonia: Differential diagnosis and performance timing. BMC Med Imaging 2009;9:7.
34. Holan G. Long-term effect of different treatment modalities for traumatized primary incisors presenting dark coronal discoloration with no other signs of injury. Dent Traumatolog 2006;22:14-7.
35. Holan G, Topf J, Fuks AB. Effect of root canal infection and treatment of traumatized primary incisors on their permanent successors. Endod Dent Traumatol 1992;8:12-5.