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DENTAL TRAUMATOLOGY

Development of clinical and radiographic signs associated with dark discolored primary incisors following traumatic injuries: a prospective controlled study

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Abstract – The purpose was to evaluate late complications of asymptomatic traumatized primary incisors with dark coronal discoloration. The clinical and radiographic signs of 97 teeth of the study group were recorded along a follow-up period that ranged between 12 and 75 months (mean >36 months). Children's age at time of injury ranged between 18 and 72 months (mean 40). The control group consisted of 102 non-discolored maxillary primary central incisors in 51 children older than 54 months with no history of dental trauma. In 50 teeth (52%) the color faded or became yellowish and in 47 (48%) it remained dark. Clinical signs of infection, that were diagnosed 5–58 months after the injury, were associated significantly more with dark than yellowish hues (83 and 17%, respectively). Teeth that had changed their color to become yellow presented more PCO than teeth with black/gray/brown coronal discoloration (78 and 6%, respectively). Arrest of dentine apposition was found in 15 teeth, one had yellow coronal discoloration and the remaining 14 had a dark shade. Eleven teeth showed inflammatory root resorption all with dark discoloration. Two atypical types of root resorption were observed: a surface resorption restricted to the lateral aspects of the apical half of the root while the root length remained unchanged and in the other expansion of the follicle of the permanent successor was observed. Expansion of the dental follicle was observed in 72% of all teeth with no significant difference between the various types of coronal discoloration but only half of the cases were associated with resorption of the root of the primary incisor. The various pathologic findings observed in the study group were either absent or rarely seen in the control group. It can be concluded that more than 50% of the primary incisors that retain their dark coronal discoloration acquired after dental injuries remain clinically asymptomatic till the eruption of the permanent successor even if they present accelerated root resorption. Asymptomatic traumatized primary incisors that retain their dark coronal discoloration may develop a sinus tract and inflammatory root resorption years after the injury. There is still a dilemma: which

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treatment is better for dark discolored primary incisors: early endodontic treatment or follow-up with the risk of development of infection and root resorption that may require extraction?

Tooth discoloration is a common post-traumatic complication (1–6) and is, in many instances, the only clinical evidence of trauma to the tooth (7). The three main shades (pink, yellow and gray) are the external expression of changes in the pulp of teeth following traumatic injuries. While pink and yellow discoloration reflect clear clinical and radiographic conditions (intrapulpar hemorrhage or internal resorption and pulp canal obliteration (PCO), respectively), there is a wide disagreement regarding the condition of the pulp in primary incisors with dark coronal discoloration. This controversy derives from the frequent observation of primary incisors that remain for years without any clinical and/or radiographic pathologic signs despite the dark coronal discoloration (8).

It is widely agreed that appearance of dark gray discoloration of a primary incisor shortly after the injury neither defines the fate of the tooth in the long run nor does it serve as an indication for treatment (1, 9–12). In the course of time the dark gray discoloration may fade with the tooth regaining its original shade (2, 3, 10, 11, 13, 14), it may change to yellow indicating PCO (11, 13), or it may persist (5, 7, 8, 11). Teeth with persisting dark discoloration may remain asymptomatic clinically and radiographically (3–5, 7, 8, 11) or develop periapical osteitis (2, 5, 10). Although dark coronal discoloration is a common finding following traumatic injuries to the primary incisors, only two longitudinal studies were published concentrating on late development of complications in the primary incisors (2, 5).

The purposes of this prospective study was: to evaluate the development of late complications and the fate of traumatized primary incisors that initially presented no clinical and/or radiographic symptoms, except for dark-gray coronal discoloration and were left for follow-up till natural exfoliation or appearance of signs of infection.

Materials and methods

Study group

The study group consisted of children who presented to the emergency clinic of the Department of Pediatric Dentistry at the Hadassah School of Dental Medicine in Jerusalem, Israel, and had at least one primary incisor with dark-gray coronal discoloration following a traumatic injury. Data collected included gender, type of injured tooth, type of injury, child's age at time of injury, and time interval between injury and first observation of discoloration. Only non-carious non-restored primary incisors that were clinically and radiographically asymptomatic except of having a dark discolored crown at the initial recruitment visit were included in the study.

The children were asked to return for periodic recall examinations every 6–12 months till the eruption of the permanent successors. Parents were instructed to return immediately if any untoward changes associated with the injured teeth or surrounding soft tissues were observed before the next scheduled recall visit. The teeth underwent clinical and radiographic examinations at the periodic recall visits. To be included in the study teeth had to have at least 12 months follow-up. Teeth with <12 months follow-up were included only if root canal treatment or extraction were performed because of obvious signs of infection such as sinus tract, external inflammatory root resorption or periodontal breakdown.

Follow-up period began at time of injury when known. When the time of injury was not known, follow-up began when tooth discoloration was first observed. The end of the follow-up period was defined as the time of last recall examination when the involved teeth had not yet exfoliated or the time an involved tooth has been extracted. The follow-up period ended with the eruption of the permanent teeth for patients who were available for examination till that age.

Clinical and radiographic examination

The aim of the clinical examination at each visit was to detect early pathologic signs associated with the injury. These included (i) appearance of the soft tissues adjacent to the affected teeth (swelling, sinus tract), and (ii) sensitivity to palpation at the depth of the vestibule above the affected teeth. The shades of the teeth were also recorded at the recall visits but only the shade observed at the last visit was used for statistical analysis.

Periapical radiographs were taken every 12 months or less if clinical findings had evoked suspicion of pathologic changes in the primary incisors. The radiographic examination intended to detect internal and/or external root resorption, periapical radiolucent defects, arrest of dentin apposition, PCO, expansion of the periodontal ligament (PDL), and any other pathologic sign that may be associated with the dental injury. Radiographic observations of irregular develop-

ments related to the permanent successors were also recorded.

No treatment was performed to the injured teeth unless definite clinical and radiographic signs of infection were detected (i.e. sinus tract, suppuration of pus from the sulcus surrounding the affected tooth, or inflammatory root resorption with a periapical radiolucent area). Teeth showing signs of infection were either extracted or pulpectomized and follow-up continued. When there was evidence or suspicion that the development of the permanent successor is at risk, the affected primary incisors were extracted immediately.

Control group

The control group consisted of the maxillary primary central incisors of children who were older than 54 months when first visited a private dental office. Data was recorded on their first visit when a diagnostic periapical radiograph of the premaxilla was taken as part of their initial examination. In order to be included in the control group the teeth had to meet the following criteria: (i) no history of dental trauma, (ii) no clinical signs of dental injuries such as coronal discoloration, displacement because of luxation, crown fractures (except of enamel crack or minor incisal fractures limited to the enamel), (iii) no advanced physiologic root resorption observed on a periapical radiograph, (iv) no deep carious lesions. Periapical radiographs were evaluated to detect the same parameters as in the study group. The findings were statistically analyzed using the chi-square test with level of significance set at $P < 0.05$.

Results

Study group

A total of 96 children (60 boys and 36 girls) with 127 dark discolored teeth had their initial examination following traumatic injuries to their teeth. Twenty-three children with 30 teeth were excluded because of short-term follow-up period (<12 months) leaving 73 children with 97 teeth that comprised the study group. Forty-six (63%) children of the study group were boys and 27 (37%) were girls. Forty-seven teeth were left maxillary central primary incisors, 48 were right maxillary central incisors and two teeth were maxillary lateral incisors, one of each side. Fifty children had one dark discolored tooth, 22 children had two teeth and one child had three discolored teeth.

Parents of 17 children (11 boys and six girls) with 21 teeth could not recall any injury to the teeth and tooth discoloration was the only drive to look for professional advice. The mean age at time of injury of the other 56 children was 40 months and ranged between 18 and 72 months (median 38). There was no difference between the mean age of boys (40 months) and girls (41 months) at time of injury.

Type of injury

Type of injury was recorded for 76 teeth that were checked shortly after the injury. Thirty-four percent (26 of 76) had concussion, 49% (37 of 76) of the injuries were diagnosed as subluxation, 13% (10 of 76) had lateral luxation and 4% (three of 76) were intruded.

Twenty-one children (15 boys and six girls) with 26 teeth reported on a repeated injury to the teeth and one of these had two repeated injuries during the follow-up period.

Follow-up periods

Three teeth were included in the study group despite the fact that they had <12 months follow-up. Of these, two teeth became infected and presented inflammatory external root resorption and were, therefore, extracted 5 and 8 months after the injury and one tooth had a repeated injury that necessitate extraction 8 months after the initial trauma. Thirty discolored primary incisors were sequentially followed till eruption of their permanent successors. The follow-up period of these teeth ranged between 17 and 75 months with a mean of 42 months. The other 64 teeth were not available for evaluation till exfoliation and eruption of their permanent successors. Their follow-up time ranged between 12 and 61 months with a mean of 34 months.

Clinical findings

Color change

All teeth in the study group had dark discoloration of the crown when follow-up began. A variety of terms were used at time of first examination to describe the dark discoloration of the involved teeth including black, dark, gray, dark-gray, light gray, grayish, brown, light brown and combinations of colors such as gray-brown, black-gray and reddish-brown. The color definition of 47 (48%) teeth at the last recall visit remained one of the dark hues: black, gray or brown. The color observed in the other 50 teeth (52%) has changed to become brighter compared with the original color definition as follows: In four teeth the dark color faded completely, leaving the tooth without any evidence of previous coronal discoloration. In 26 teeth the color

changed into a yellowish hue. Nine teeth presented a gray-yellow hue at the last recall evaluation and the color of 11 teeth was defined as brown-yellow.

Twenty two children had both maxillary primary central incisors with coronal discoloration. In seven of them (14 teeth) the color of both teeth had changed in the same direction or had not changed at all. In 15 children (30 teeth) each tooth showed a different color changes (Fig. 1).

Soft tissue changes

Seventy-six percent of the teeth (74 of 97) teeth in the study group showed no clinical pathological signs of infection associated with an injury to the primary incisors (Table 1). Twenty-three teeth (24%) showed clinical signs of infection such as swelling in the vestibule or sinus tract. The color of the crown of 19 (83%) of the 23 infected teeth was defined as black, gray or brown compared with only 4 (17%) of the teeth with a yellowish shade ($P < 0.001$). Infection was diagnosed 5–58 months after the injury (mean 29 months). One of the gray discolored teeth became infected through exposure of the pulp that occurred after gradual attrition of the incisal edge. The expected response to attrition, i.e. apposition of reparative dentine and recession of the pulp, could not occur in this case, because of necrosis of the pulp that resulted from the injury (Fig. 2). Sixty percent (28 of 47) of the teeth with persistent black/gray/brown discoloration did not present any clinical sign of infection (Table 1) and three of these showed irregular root resorption (Table 2; Fig. 3).

Fig. 1. A clinical view of the maxillary primary central incisors of a 5-year 6-month-old boy. Parents first noticed discoloration 21 months earlier but could not recall any injury to the teeth. The teeth are asymptomatic and have different dark shades.

Table 1. Clinical findings of dark discolored primary incisors

	Tooth color		
Soft tissue	Yellowish shades [*n* (%)]	Black-gray-brown [*n* (%)]	Total [*n* (%)]
Intact	46 (92)	28 (60)	74 (76)
Swelling/sinus tract	4 (8)	19 (40)	23 (24)
Total	50 (100)	47 (100)	97 (100)

Chi-square $P < 0.001$.

Radiographic findings

Table 2 shows the correlation between clinical signs of infection and radiographic signs. Inflammatory root resorption was highly associated with swelling and sinus tract. Teeth with surface root resorption

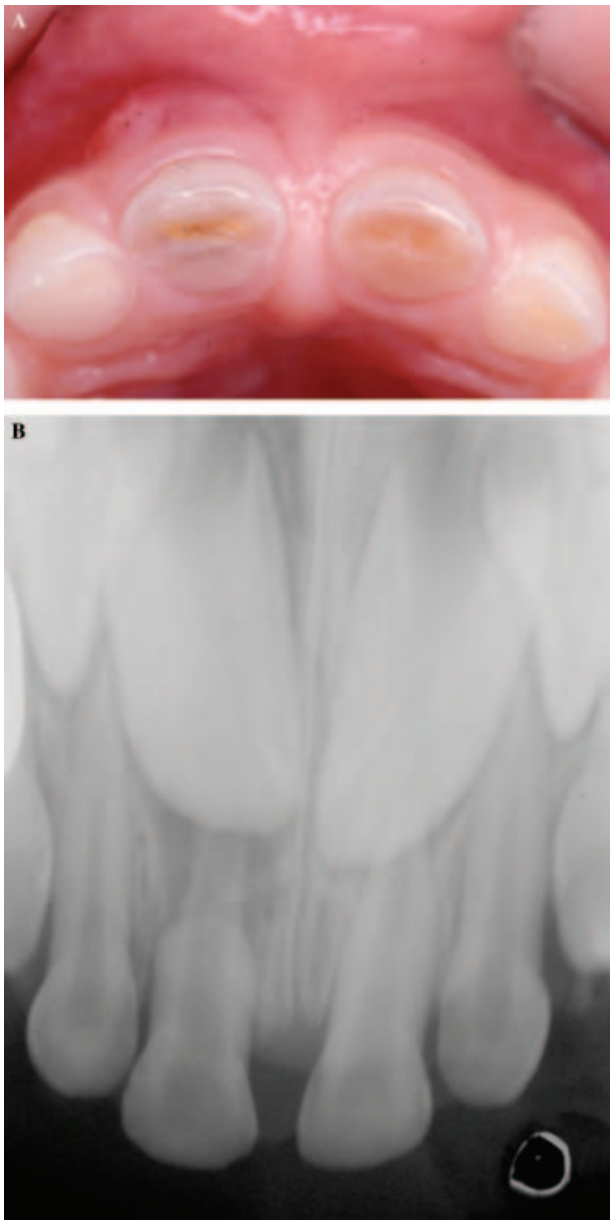

Fig. 2. Maxillary primary central incisors with dark coronal discoloration. (A) Attrition of the right incisor till exposure of the pulp, infection and development of sinus tract. (B) Radiograph showing inflammatory root resorption.

Table 2. Correlation between clinical signs of infection and radiographic signs

Radiographic signs	Clinical signs: Swelling/sinus tract	Clinical signs: Intact/ asymptomatic	Total
External root resorption*			
Inflammatory	8	3	11
Surface	0	31	31
Associated with dental follicle expansion	7	29	36
Physiologic or no resorption	8	23	31
Pulp condition			
Internal resorption	1	1	2
Arrest of dentin apposition	4	11	15
Pulp canal obliteration	3	39	42
Tube-like mineralization	0	7	7
Normal appearing pulp	7	24	31
Expansion of the follicle of the permanent tooth (with and without root resorption)	15	55	70

*Some teeth had more than one characteristic of external root resorption.

and root resorption associated with expansion of the dental follicle of the permanent incisor (see below) were associated with normal clinical appearance in 100 and 81%, respectively.

Pulp condition

The most common radiographic finding that related to dental trauma was PCO observed in the 43% (42 of 97) of the injured primary incisors (Table 3). Teeth that had changed their color to become yellow presented more PCO than teeth with black/gray/brown coronal discoloration (78 and 6%, respectively) ($P < 0.001$). Normal appearing pulp was found in 32% (31 of 97) of all teeth in the study group with the highest prevalence (57%) in teeth with persistent black/gray/brown coronal discoloration. An unusual pattern of pulp canal calcification showing two radio opaque stripes in the pulp canal parallel to the root dentin walls (Fig. 4) was observed in seven teeth. This pattern has been previously described and named 'tube-like mineralization' (15). Arrest of dentine apposition was found in 15 teeth 14 of which had black/gray/brown discoloration.

External root resorption

Thirty-two percent (31 of 97) of the teeth showed physiologic root resorption or no external root resorption at all. Inflammatory root resorption was observed in 11 teeth (11%) all with black/gray/brown discoloration (Table 4). Twenty percent (19/97) of the teeth, with different shades of the crown, showed surface root resorption. Of these, seven teeth showed surface resorption restricted to the sides of the

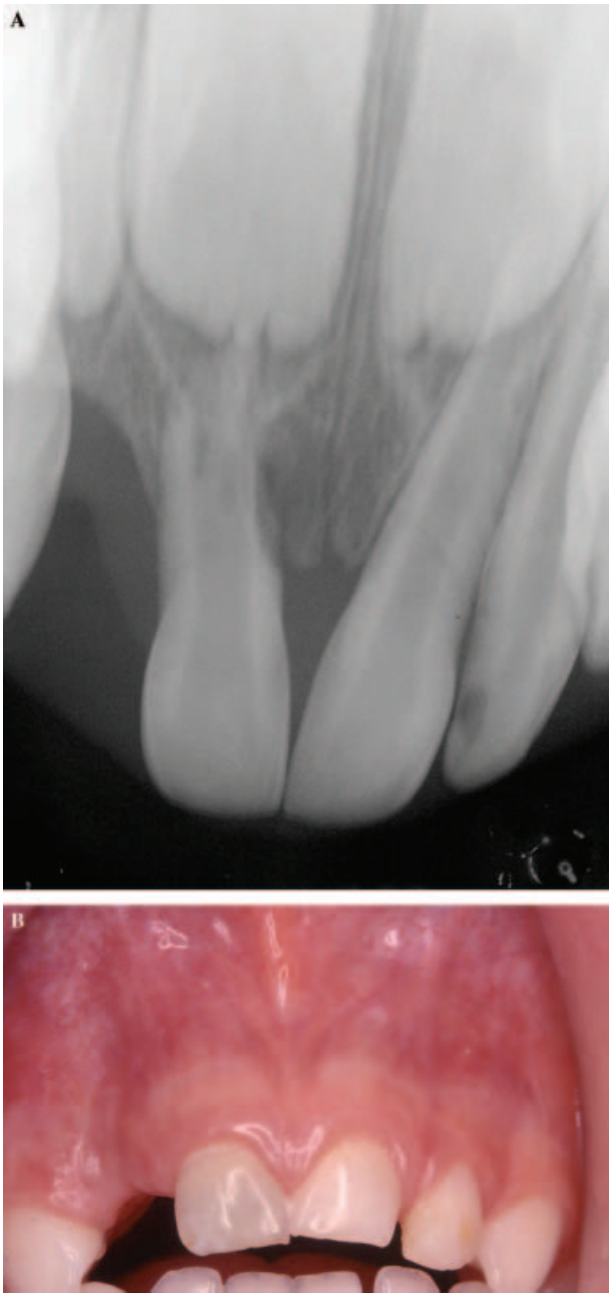

Fig. 3. (A) Irregular external root resorption without accompanying resorption of the adjacent bone in tooth 51. (B) The tooth is clinically asymptomatic.

apical half of the root while the root length and the width of the coronal half, are retained (Fig. 5). This pattern of external root resorption has been previously termed 'circumferentail', 'semilunar' or atypical root resorption (ARR) (16–18). Thirty-six teeth (37%) showed root resorption that could not be attributed to any of the known patterns of external root resorption. This resorption pattern was clearly associated with expansion of the dental follicle of the permanent successors (see below).

Table 3. Distribution of teeth with different crown discoloration and radiographic appearance of the pulp

Radiographic appearance of the pulp	Tooth color: Yellowish shades [*n* (%)]	Tooth color: Black-gray-brown [*n* (%)]	Total [*n* (%)]	Control group [*n* (%)]
Internal resorption	0 (0)	2 (4)	2 (2)	0 (0)
Arrest of dentin apposition	1 (2)	14 (30)	15 (16)	0 (0)
Pulp canal obliteration (PCO)	39 (78)	3 (6)	42 (43)	1 (1)
Tube-like mineralization	6 (12)	1 (2)	7 (7)	0 (0)
Normal	4 (8)	27 (57)	31 (32)	101 (99)
Total	50 (100)	47 (100)	97 (100)	102 (100)

Chi-square $P < 0.001$.

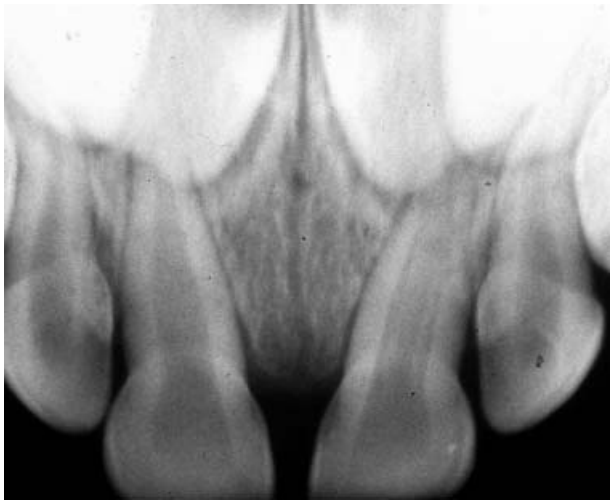

Fig. 4. Radiograph showing two radio opaque strips in the pulp canal of the maxillary left central primary incisor indicating tube-like mineralization of the pulp.

Expansion of the follicle of the permanent successor

In 72% of the injured teeth (70 of 97) the last radiograph taken as part of the follow-up examinations showed expansion of the dental follicle of the permanent successor (Fig. 6). This expansion exceeded 2 mm width and was observed in association with teeth with all types of coronal discoloration with no significant difference between the various types of discoloration ($P > 0.05$) (Table 5). The expansion could also be seen in association with various types of external root resorption (Table 6) and with PCO (Table 7) with no significant difference between the groups ($P > 0.0$). It could also be seen in association with endodontically treated teeth. In 79% of the cases expansion of the dental follicle had no clinical expression, although in three children a hard swelling could be palpated above the discolored maxillary primary incisor. Only about one fifth of the cases of expansion of the dental follicle were associated with infection expressed by fluctuant swelling or sinus tract above the primary incisor.

Correlation between clinical and radiographic findings

Table 2 shows the correlation between clinical and radiographic findings. Seven teeth with normally appearing pulp and three with PCO presented clinical signs of infection (swelling or sinus tract). Eleven teeth remained clinically asymptomatic although arrest of dentine apposition was evident on radiographs.

In three cases with expansion of the follicle of the permanent successor a hard-to-palpate swelling was detected in the vestibule above the affected tooth. This was not associated with redness of the overlaying mucous membrane or sensitivity to palpation nor were the affected teeth mobile or sensitive to percussion.

Fate of the teeth

Table 8 shows the fate of teeth according to crown discoloration at the last follow-up examination. Of

Table 4. Distribution of teeth with different crown discoloration and type of external root resorption

Type of external resorption	Tooth color: Yellowish shades [*n* (%)]	Tooth color: Black-gray-brown [*n* (%)]	Total [*n* (%)]	Control group [*n* (%)]
Inflammatory	0 (0)	11 (23)	11 (11)	0 (0)
Surface	10 (20)	9 (19)	19 (20)	15 (15)
Associated with dental follicle expansion	24 (48)	12 (26)	36 (37)	7 (7)
Physiologic or no resorption	16 (32)	15 (32)	31 (32)	80 (78)
Total	50 (100)	47 (100)	97 (100)	102 (100)

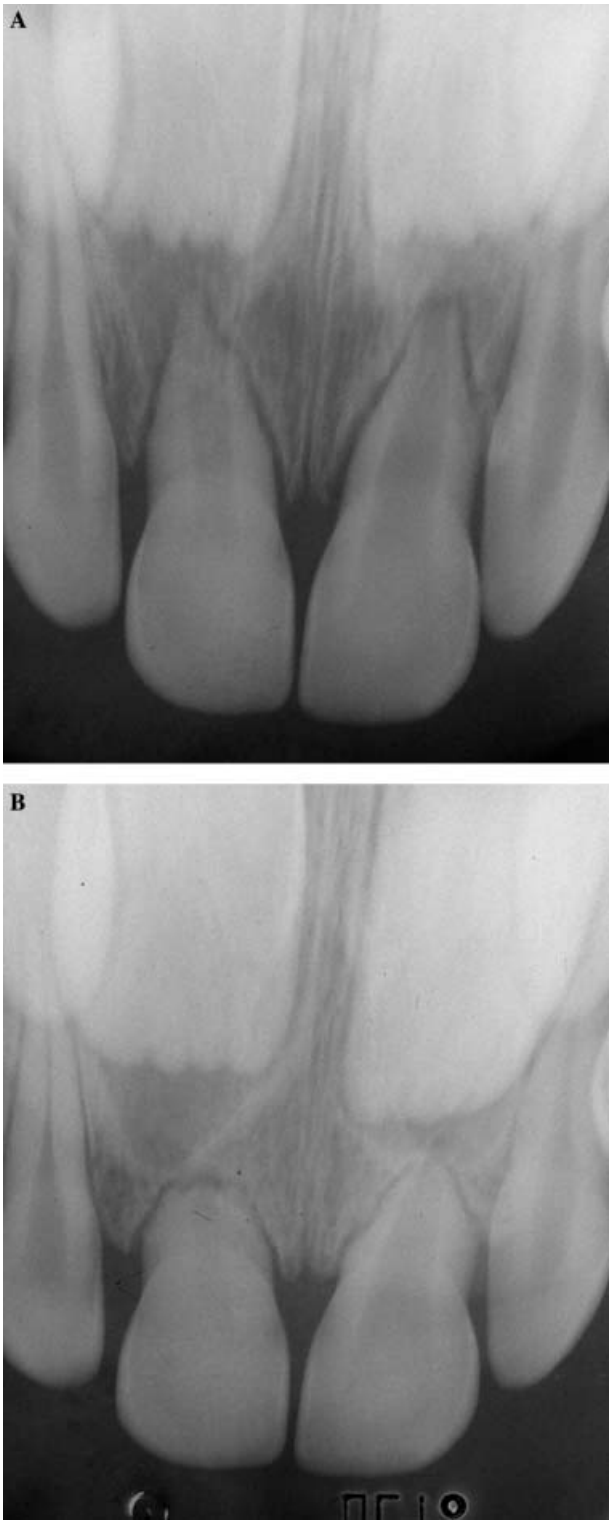

Fig. 5. (A) Atypical external root resorption of both maxillary primary central incisors and PCO of the right central incisor following an injury in a 39-month-old child. (B) Fourteen months later. Notice the progression in root resorption.

Fig. 6. Expansion of the dental follicle of the permanent central incisors, following injury to the primary predecessors. (A) With and (B) without resorption of the root of the primary incisors.

the 97 teeth in the study group 72 teeth (74%) remained asymptomatic and did not present any clinical or radiographic pathologic finding that justified extraction or root canal treatment. Forty-six yellow discolored teeth (92%) remained asymptomatic along the follow-up period compared with only 26 teeth (55%) of the black/gray/brown-discolored incisors ($P < 0.001$). For 11 dark discolored teeth follow-up lasted till eruption of the permanent successors. Follow-up of twenty-seven of the asymptomatic teeth continued till natural

Table 5. Expansion of the dental follicle of permanent teeth after coronal discoloration of primary predecessors

Expansion of dental follicle of the permanent tooth	Tooth color			Control group [*n* (%)]
	Yellowish shades [*n* (%)]	Black-gray-brown [*n* (%)]	Total [*n* (%)]	
Yes	37 (74)**	33 (70)**	70 (72)*	27 (26)*
No	13 (26)	14 (30)	27 (28)	75 (74)
Total	50 (100)	47 (100)	97 (100)	102 (100)

Chi-square *$P < 0.001$; **$P > 0.05$.

Table 6. Expansion of dental follicle associated with different types of external root resorption in discolored teeth

Expansion of dental follicle of the permanent tooth	External root resorption				Total [*n* (%)]
	No [*n* (%)]	Inflammatory [*n* (%)]	Surface [*n* (%)]	Associated with dental follicle expansion [*n* (%)]	
Yes	18 (26)*	6 (9)*	10 (14)*	36 (51)	70 (100)
No	13 (48)*	5 (19)*	9 (33)*	0 (0)	27 (100)
Total	31 (32)	11 (11)	19 (20)	36 (37)	97 (100)

*Chi-square $P > 0.05$.

exfoliation and eruption of the permanent successor. Three of the twenty-seven teeth had to be extracted, as they did not exfoliate when the permanent teeth had erupted (Fig. 7). One tooth was lost because of repeated injuries and twenty-four teeth (25%) became infected 5–58 months after the injury (mean 29). Twelve of the infected teeth were extracted because of extensive inflammatory external root resorption. The other twelve teeth were pulpectomized and a root canal treatment was performed. A necrotic pulp was found in all endodontically treated teeth. Of the root treated teeth three were not available for further follow-up; four were extracted because of failure of the endodontic treatment; one tooth was avulsed because of a repeated injury and four teeth were followed uneventfully till eruption of the permanent successor.

Table 7. Expansion of dental follicle associated with pulp canal obliteration (PCO)

Expansion of dental follicle of the permanent tooth	PCO		Total [*n* (%)]
	Yes [*n* (%)]	No [*n* (%)]	
Yes	36 (73)	34 (71)	70 (72)
No	13 (27)	14 (29)	27 (28)
Total	49 (100)	48 (100)	97 (100)

Chi-square $P > 0.05$.

Table 8. Fate of dark discolored primary incisors

Fate of teeth	Tooth color		Total [*n* (%)]
	Yellowish shades [*n* (%)]	Black-gray-brown [*n* (%)]	
Repeated injury (lost)	0	1	1
Total	0 (0)	1 (2)	1 (1)
Infected			
Extracted	2	10	12
Endodontic treatment	2	10	12
Total	4 (8)	20 (43)	24 (25)
Asyptomatic			
Follow-up	30	15	45
Follow-up till normal exfoliation	16	8	24
Over retained	0	3	3
Total	46 (92)	26 (55)	72 (74)
Total	50 (100)	47 (100)	97 (100)

Chi-square $P < 0.001$.

Control group

One hundred and two maxillary primary central incisors of 51 children (27 boys and 24 girls) who had their first visit in the dental office in a period of 4 years and 4 months comprised the control group. The children's age ranged between 55 and 93 months (mean 67). None of the teeth presented a periapical lesion or pulp changes except one tooth with PCO but no coronal discoloration. Sixty-three teeth (62%) in 34 children did not present any radiographic pathologic sign. Twenty-seven teeth (26%) presented expansion of the dental follicle of the permanent successor and in 12 teeth it was the only radiographic finding. In seven teeth it was associated with accelerated external root resorption. Table 5 shows that while no significant difference exists between black-gray-brown discolored teeth and yellowish teeth regarding expansion of the dental follicle of the permanent teeth ($P > 0.05$), this

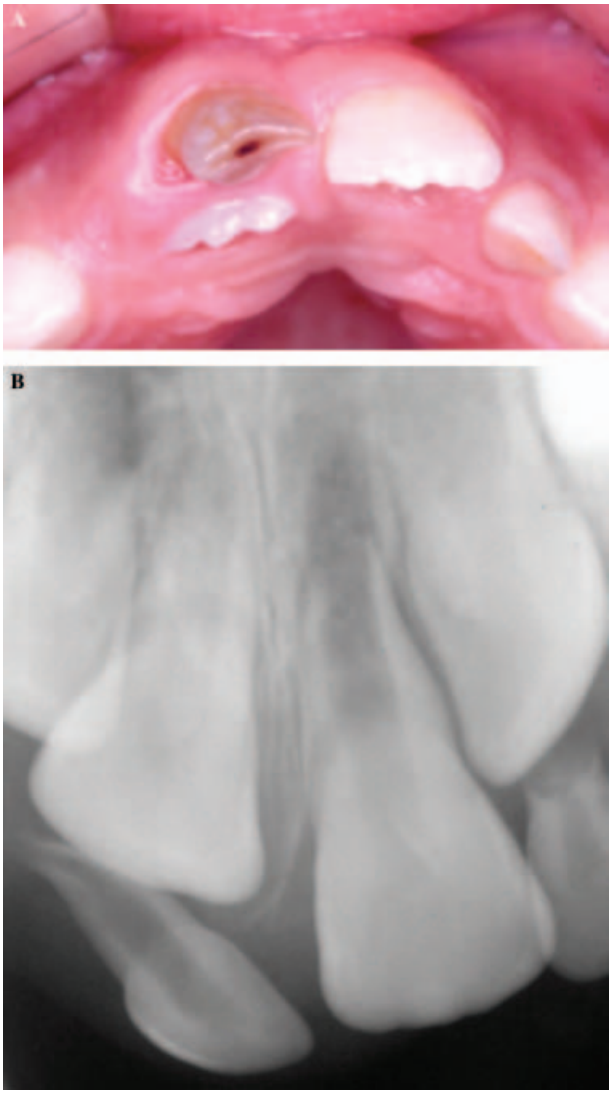

Fig. 7. (A) Ectopic eruption of the maxillary right permanent central incisor with retention of the dark discolored primary predecessor. (B) Radiograph showing failure of root resorption of the primary incisor.

phenomenon appears significantly less in teeth of the control group ($P < 0.001$). None of the teeth presented internal resorption. Eighty control group teeth (78%) presented physiologic root resorption (four teeth) or no root resorption at all (76 teeth). External root resorption was observed in 22 (22%) teeth. Circumferential resorption (ARR) was found in 15 teeth (15%) and resorption associated with expansion of the dental follicle of the permanent tooth appeared in seven teeth. The inflammatory and the replacement types of external root resorption were not found. Table 4 presents the distribution of teeth by types of root resorption and shows a significant difference between the traumatized teeth and the control group regarding pathologic root resorption vs. no or physiologic root resorption.

Two teeth had root fractures, one associated with resorption of the apical fragment of the root and the second without any other radiographic sign. Tables 3–5 show the differences between the radiographic findings of the dark discolored teeth and those of the control group.

Discussion

Observations of discolored teeth in the present study revealed a wide range of shades. Customarily, no differentiation is made between the various dark shades (i.e. gray, brown and black) and they are collectively termed 'dark discoloration'. Intrapulpar hemorrhage that occurs following traumatic injuries results in death of the red blood cells, degradation of the hemoglobin to hemosiderin and hematoidin that seep into the dentinal tubules (19). This mechanism is commonly offered as the mechanism by which a tooth acquires a dark hue. However, it does not provide an answer to the question: why do some children present traumatized primary incisors that changed their color into gray while in others the teeth turn brown or black with various intermediate shades? Moreover, two incisors, in the same child, that were injured at the same time may acquire different dark hues. Assessment of tooth color in the clinical setting is based on impression and not on a calibrated calorimetric scale and is therefore a subjective estimation. The fact that 'dark' discoloration is a generalization of different shades has been recognized by Soxman et al. (4) who distinguished between the various shades by using the Vita Lumin shade guide in an attempt to correlate between pulp pathology and shade of traumatized discolored primary incisors. They did not find any correlation between the degree of discoloration and the histopathologic status of the pulp. Croll et al. (20) were also unable to correlate between clinical and radiographic observations and the histopathologic changes in the pulp of traumatized discolored primary incisors.

The findings of the present study show that the initial dark color of the primary incisors may change into a wide range of shades. The different shades could be roughly divided into two groups: the yellowish and the dark shades. In more than half of the teeth (52%) the dark discoloration completely disappeared or faded into a yellowish hue. Most of these teeth (78%) presented PCO at the last follow-up examination, indicating that the pulp remained vital, while only 8% had developed swelling or a sinus tract indicating a necrotic and infected pulp. On the contrary, a much higher percentage (43%) of the teeth that had retained their dark shades presented signs of a necrotic and infected pulp. Moreover, none of the yellow discolored teeth

presented external inflammatory root resorption compared with 23% of the dark discolored incisors.

Widening of the dental follicle of the permanent successor was found in 72% of the teeth with no significant difference between teeth that remained dark and those turning brighter and between teeth with and without PCO. Expansion of the dental follicle of permanent incisors has been previously reported in association with primary incisors that experienced traumatic injuries and had dark coronal discoloration (21). In this report the expansion of the dental follicle has been diagnosed as a dentigerous cyst. Detection of an expanded dental follicle of an unerupted permanent tooth is not enough to diagnose a dentigerous (follicular) cyst. In many cases even a histopathologic examination does not guarantee correct diagnosis and confusion between a dentigerous cyst and simple expanded dental follicle may occur as both are lined by epithelium (22). They differ by the type of the epithelial layers: whereas dental follicles are lined by reduced enamel epithelium that changes with age to squamous epithelium, dentigerous cysts are lined by stratified squamous epithelium (22). The clinician who faces a wide expansion of a dental follicle of a permanent incisor where the primary tooth had experienced an injury should be aware of the risk that a dentigerous cyst may develop. Suspicion should be evoked when the follicle is asymmetric, the permanent tooth is deflected from its normal alignment, fails to continue its eruption and fails to resorb the root of the primary incisor (21, 22). If all or some of these signs are observed the primary tooth should be removed and the permanent tooth followed to assure proper eruption.

As the expanded dental follicle lies close to the apex of the primary incisor its radioluceny may be erroneously interpreted as periapical osteitis. As most cases of expansion of the dental follicle were not associated with infection, this condition should be differentiated from periapical osteitis as the approach to treatment of these two entities may be different.

The rate of root resorption associated with expansion of the dental follicle seemed to be faster than physiologic root resorption and to differ from the pattern of physiologic root resorption. However, based on the observation of this study, it is still not clear whether it should be considered as physiologic or pathologic external root resorption.

Unlike permanent teeth in which external root resorption is always considered a pathologic process, in primary teeth it may be either physiologic, as part of normal development of the dentition, or pathologic, as a result of inflammation following traumatic injuries or pulpal infection. Some types of external root resorption observed in the present study fit the definitions and description provided for traumatized permanent teeth (6), while other patterns of root resorption seem to appear exclusively in primary teeth. Replacement resorption (ankylosis) although not observed in the present study has been previously found in primary teeth following intrusive luxation (23).

Surface resorption in traumatized permanent teeth begins when the innermost layer of the periodontal ligament, that normally protects the root from osteoclastic activity, is damaged or removed and the mineralized root surface is exposed. This type of resorption stops as soon as the root surface is relined by a layer of PDL cells that again protect the root against further attack of odontoclasts (24). Radiographically, the root of permanent teeth with surface resorption is surrounded by a normally appearing PDL. Traumatized primary incisors may present a similar radiographic picture, although the accelerated resorption process continues (Fig. 5). It has been shown that the process of root resorption in primary teeth consists on three phases: (i) active root resorption, (ii) a resting phase and (iii) root repair by cellular cementum formation (25). Based on the mechanism of root resorption in the permanent dentition, it can be hypothesized that accelerated root resorption, seen in dark discolored primary incisors, occurs when toxins, deriving from the necrotic pulp, have access through the dentinal tubules to the outer surface of the root where they can irritate the surrounding tissue. This becomes possible during the active phase of root resorption when the dentinal tubules are exposed. The situation, in which dark discolored primary incisors that remained asymptomatic for a long time, develop a sinus tract and rapidly progressing resorption of the root and the surrounding bone, requires an explanation. A necrotic pulp is an excellent growth culture for microorganism, which may reach the pulp after an additional injury through the ruptured periodontal ligament, enamel-dentin cracks or from the blood stream by anachoresis. When the pulp becomes infected, invasion of bacterial products through the dentinal tubules sustain a rapidly progressing inflammatory root resorption (26). This can explain the situation in which dark discolored primary incisors that contain a necrotic, but not infected, pulp present root resorption but do not present any clinical pathologic sign (Fig. 3).

For unknown reason progressive root resorption is sometimes confined to the apical half of the root whereas its coronal part retains the original thickness. This pattern of root resorption in primary teeth has been termed 'circumferential' and attributed to digit sucking (16, 17). Indeed the authors

excluded from the study cases with history of traumatic injuries, however, an unknown percentage of trauma to primary teeth go unnoticed. An attempt to correlate this ARR with traumatic injuries to the primary incisors failed (18) and it was found to appear significantly more only in teeth of children with and increased overjet. Children who have the habit of digit sucking may have an increased over-jet, which is a risk factor for injuries to the maxillary incisors (27). Rubel (17) had distinguished between four types of ARR that were found in the present study to be sequential steps of the same type of root resorption were the apical part continues to resorb until it disappears completely leaving the tooth with a shortened root and a wide layer of bone separating it from the permanent incisor (Fig. 5).

Some authors extracted traumatized primary incisors with dark coronal discoloration claiming that such teeth have a necrotic pulp (28, 29) and may develop periapical osteitis (2), while others suggest pulp therapy to prevent periapical involvement (4). Several authors, however, feel that dark discoloration should be used as an adjunct in diagnosis (30) and treatment of such teeth should not be instituted before signs, such as sinus tract or swelling appear (31). This study shows that 55% of the teeth that remained dark did not present any clinical sign of infection along the follow-up period that in some cases ended with the eruption of the permanent tooth. This finding is in accordance with the observations of Sonis (5) who found that 72% of gray-black discolored primary incisors failed to develop any radiographic or clinical evidence of pathology. Reed and Sayegh (3) found about 80% of dark discolored primary incisor to be retained until normal time of exfoliation with no treatment. Such asymptomatic primary incisors with dark coronal discoloration following traumatic injuries were found to contain a necrotic or necrotizing pulp (8). No damage to the permanent successors of such teeth is expected as long as the pathoses are restricted to the pulpal contents (5). Forty-three percent of the dark discolored teeth developed signs of infection during the follow-up period. Some of these teeth had to be extracted because of extensive inflammatory root resorption. Attempt was made to save twelve infected teeth in which root resorption was minimal or completely absent. This attempt failed in four teeth that were finally extracted. Success rate of endodontic treatment of infected primary incisors is expected to be lower than in non-infected teeth. This generates a dilemma to the dentist who faces a young patient with an asymptomatic dark discolored primary incisor. Root canal treatment may have higher success rates if performed at this stage than if postponed till signs of infection appear. Conversely, such endodontic treatment may be unnecessary as there are <50% chances that dark discolored teeth will become infected and require either pulpectomy or extraction. In order to answer this question the two treatment options (immediate pulpectomy and follow-up) must be compared.

The control group was based on children that when examined were older than 54 months in order to allow the period the teeth were exposed to the oral environment to be as long as possible. When comparing the radiographic findings observed in dark discolored teeth to teeth of the control group, it is clear that expansion of the follicle of the permanent tooth and the various types of pathologic external root resorption are associated with dental trauma. The presence of such signs in the control group can be explained by undiagnosed minor injuries to the teeth. Although parents of children of the control group could not recall any event of dental trauma, detection of one tooth with PCO and two teeth with root fracture indicate that parents are not always aware to dental injuries experienced by their children.

Conclusions

1. Dark coronal discoloration that appears in primary incisors following traumatic injuries may fade or possess a lighter yellowish shade in more than 50% of the teeth.
2. Yellowish teeth develop significantly less pathologic signs that require intervention as compared with traumatized primary incisors that retain the dark discoloration.
3. More than 50% of the primary incisors that retain their dark coronal discoloration acquired after dental injuries remain clinically asymptomatic till the eruption of the permanent successor even if they present accelerated root resorption.
4. Asymptomatic traumatized primary incisors that retain their dark coronal discoloration may develop a sinus tract and inflammatory root resorption years after the injury.
5. There is still a dilemma: which treatment is better for dark discolored primary incisors? Early endodontic treatment or follow-up with the risk of development of infection and root resorption that may require extraction.

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