The diagnostic value of coronal dark-gray discoloration in primary teeth following traumatic injuries

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Abstract

The diagnostic value of dark-gray discoloration of the crown of primary incisors following traumatic injury as a predictor of pulp vitality is controversial. This retrospective study attempted to examine the condition of the pulp of primary incisors with dark-gray discoloration following traumatic injuries. Forty-eight gray caries-free, traumatized primary incisors were examined clinically and radiographically prior to treatment. All teeth included in the study were free of any clinical and/or radiographic signs of pulp necrosis. Crown discoloration was the only clinical or radiographic sign of pulp involvement. Pulpectomy was performed in five teeth within the first month after trauma, in 16 teeth during the second month, 11 teeth between 2 and 6 months, seven teeth between 6 and 12 months, and three teeth after more than a year. In six teeth the time of injury was unknown. Endodontic treatment was initiated without local anesthetic under rubber dam, access to the pulp chamber with high speed, and debridement with a barbed broach. Assessment of the pulp condition was made upon opening the pulp chamber, as follows: Vital — pulp bled when exposed; partial necrosis — bleeding disclosed at the apical area during debridement; total necrosis — no bleeding was observed. Pulp necrosis was found in 37 teeth (77.1%), 10 teeth (20.8%) presented partial necrosis, and only one tooth (2.1%) had a vital pulp. Dark-gray discoloration of primary incisors could be interpreted as an early sign of pulp degeneration that would deteriorate into necrosis. Moreover, discolored primary incisors can be necrotic even without presenting tenderness to percussion, increased mobility, and periapical osteitis. (Pediatr Dent 18:224-27, 1996)

Crown discoloration of primary incisors is a common post-traumatic complication. In many instances it is the only evidence of trauma to the tooth, and is best observed by transillumination. Attempts have been made to correlate discoloration to pathologic radiographic and histological pulp changes of injured primary incisors. However, some of these attempts failed to demonstrate any correlation, while others resulted in a variety of findings.

Different shades observed in traumatized primary incisors have been interpreted as the expression of different pulpal conditions. Both pink and yellow discolorations do not reflect devitalization of the pulp, and no treatment is indicated according to most authors. However, follow-up is necessary as developing pulp necrosis might occur later.

The most conflicting type of discoloration is the dark-gray (also defined as grayish-blue, grayish-black, or dark-brown) with wide disagreement concerning the diagnostic meaning and suitable treatment of teeth with this type of discoloration. Some authors feel that gray or bluish color is a diagnostic sign of pulp degeneration. Schroder reinforced this argument demonstrating that 82% of gray-discolored traumatized primary incisors developed periapical osteitis less than 1 month after the injury. Conversely, Croll et al. in a study on traumatized primary incisors trying to correlate the histological findings to the clinical and radiographic observations, found that of 51 gray-black teeth, only 33 had a necrotic pulp.

Hargreaves and Craig warned against interpretation of gray discoloration of the crown as pulp death, and provided another explanation for the color change. According to these authors and to Hawes, in these cases, hemorrhage invades the dentinal tubules followed by hemoglobin breakdown, with no possibility of final absorption of the hemosiderin deposits, or disappearance of the stain.

Another factor related to tooth discoloration is the time interval between the injury and the discoloration. According to Auslander and Andreasen, pink discoloration becomes evident 1 to 3 weeks after the trauma, and the tooth returns to its natural color within 2 to 3 months. Conversely, Johnson stated that a pink primary tooth indicates internal resorption. A late yellow discoloration, on the other hand, represents pulp obliteration. Croll describes a dark-gray discoloration that oc-
curs soon after the injury and persists, reflecting a ne-
crotizing pulp with poor chances to heal. McTigue stated that color changes that occur weeks or months after the injury are more indicative of a necrotic pulp than those that occur after 1 or 2 days. This general statement, however, does not refer to any specific type of discoloration.

The purpose of this retrospective study was to ex-
amine the condition of the pulp of primary incisors with dark-gray discoloration following traumatic injuries.

**Methods and materials**

Records of patients with 88 pulpectomized primary incisors treated by the principal investigator over a period of 52 months were examined. To be included in the study the teeth had to meet the following clinical and radiographic criteria:

1. Present dark-gray discoloration of the crown prior to root canal treatment
2. Be caries free or have only initial caries
3. Present no or only slight mobility, not greater than 1 mm
4. Present no or only slight sensitivity to percus-
5. Be surrounded by healthy soft tissue
6. Present normal appearance of the pulp with no internal resorption or pulp obliteration
7. Include sufficient data in their records
8. Present normal width of the periodontal liga-

Teeth with periapical osteitis, which was defined as slight widening of the PDL limited to the periapex, also were included in the study.

Forty-eight teeth were found to be suitable for evaluation. The remaining 40 teeth were excluded from the study for the following reasons: caries exposure of the pulp (19 teeth); both trauma and caries exposure (six teeth); or clinical or radiographic evidence of pulp necrosis (swelling, mobility greater than 1 mm, sinus tract, wide periapical lesion, or pathologic inflammatory root resorption (10 teeth). Pathologic inflammatory root resorption was defined as bowl-shaped radiolucent areas on the lateral aspects of the root surface. Another five teeth were excluded due to insufficient data in their records.

The data collected included gender, patient’s age at treatment, time elapsed between the injury and the first time discoloration was observed, time elapsed between trauma and treatment, clinical condition of the tooth (increased sensitivity to percussion and mobility), and radiographic findings (periapical radiolucency, presence and degree of physiologic or pathologic external root resorption). These data described the tooth condition either just before pulpectomy was performed or up to 1 week earlier. Data recorded during treatment included the patient’s complaint of pain during extirpa-
tion, the use of a local anesthetic, and the state of pulp vitality. The latter was defined as follows:

Vital — bleeding was noticed immediately while penetrating the pulp chamber
Partial necrosis — no bleeding was evident in the coronal part, but limited bleeding was disclosed at the apical area while debriding the root ca-
nal with a barbed broach
Total necrosis — no bleeding was observed dur-
ing pulpal removal.

The teeth included 22 right and 26 left maxillary primary central incisors, in 19 girls and 24 boys (five children had both teeth treated). The children’s ages at time of treatment ranged between 23 and 69 months (mean 40.4 months).

The operator’s standard protocol for pulpectomy of primary incisors suspected of having a necrotic pulp included rubber dam isolation without a clamp, and palatal approach to reach the pulp chamber using a #330 tungsten bur on a high-speed handpiece with water spray. Local anesthesia was not routinely infiltrated to the mucosa. If the patient complained of pain during insertion of the broach, intrapulpal anesthesia might have been added. Data related to patients’ report of pain, and infiltration and intrapulpal administration of local anesthesia during the dental procedure also were recorded.

**Results**

In 46% of the cases, coronal discoloration was ob-
erved within 2 weeks after injury (Table 1). The time interval between the injury and pulpectomy ranged between 2 weeks and 2 years (Table 2). In six cases, no specific
tervals were recorded.

**TABLE 1. TIME INTERVALS BETWEEN INJURY AND FIRST OBSERVATION OF TOOTH DISCOLORATION**

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>Number of Teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 days–14 days</td>
<td>22 (45.8%)</td>
</tr>
<tr>
<td>15 days–1 month</td>
<td>4 (8.3%)</td>
</tr>
<tr>
<td>&gt;1 month–1 year</td>
<td>8 (16.7%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>14 (29.2%)</td>
</tr>
<tr>
<td>Total</td>
<td>48 (100.0%)</td>
</tr>
</tbody>
</table>

**TABLE 2. THE STATE OF PULP VITALITY AT THE DIFFERENT TIME INTERVALS BETWEEN THE INJURY AND PULPECTOMY**

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>Number of Teeth</th>
<th>NCR</th>
<th>Part NCR</th>
<th>VTL</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 weeks to 1 month</td>
<td>5</td>
<td>4</td>
<td>80 %</td>
<td>1</td>
</tr>
<tr>
<td>&gt;1–2 months</td>
<td>16</td>
<td>13</td>
<td>81 %</td>
<td>3</td>
</tr>
<tr>
<td>2–6 months</td>
<td>11</td>
<td>11</td>
<td>64 %</td>
<td>3</td>
</tr>
<tr>
<td>6–12 months</td>
<td>7</td>
<td>5</td>
<td>71 %</td>
<td>2</td>
</tr>
<tr>
<td>12 month to 2 years</td>
<td>3</td>
<td>3</td>
<td>100 %</td>
<td>0</td>
</tr>
<tr>
<td>Unknown</td>
<td>6</td>
<td>5</td>
<td>83 %</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>48</td>
<td>37</td>
<td>77.1 %</td>
<td>10</td>
</tr>
</tbody>
</table>

NCR = total necrosis of the pulp; Part NCR = partial necrosis of the pulp; VTL = vital pulp.
injury could be associated with the color change, since the parents either denied any injury to the teeth or reported multiple injuries. In eight other cases, parents were unable to recall the first time discoloration was observed or were not even aware of the change in the color of the tooth. Pulp necrosis was found in 37 teeth (77.1%), partial necrosis in 10 teeth (20.8%), and vital pulp in only one tooth (2.1%). Of 21 teeth treated during the first two months, 80% presented a necrotic pulp. Of the teeth treated 2–6 months after injury, 64% presented a necrotic pulp, and in those treated 6–12 months post injury, the pulp was nonvital in 71%. These differences were statistically analyzed using the Fisher’s exact test; however, no significant difference was found ($P = 0.257$).

The clinical and radiographic conditions of the teeth are presented in Table 3. Of the 37 necrotic teeth, 27 (73%) were not sensitive to percussion despite the dark-gray discoloration. Moreover, half of the necrotic discolored teeth were completely stable, and 46% of the teeth with a necrotic pulp did not present periapical osteitis. Since Table 3 is only descriptive, no statistical analysis has been applied on the figures.

One patient received local infiltration of lidocaine 2% with epinephrine 1:100,000 in advance, and the pulp was found to be partially necrotic. In two other patients, anesthesia was administered intrapulpally and pulp necrosis was disclosed. The explanation for this deviation from the protocol was the operator’s desire to avoid disruptive behavior. The distribution of reported pain by condition of the pulp and administration of local anesthesia are presented in Table 4.

**Discussion**

The major shortcoming of trauma studies in children is the fact that many injuries to primary teeth remain unnoticed. Many obvious clinical and/or radiographic signs of a previous injury to the teeth, especially concussions and subluxations, are not supported by the dental history due to poor recall of the parents. In addition, multiple injuries to a single tooth mask the consequences of each individual trauma. In our study, the time of injury was not known for six teeth, and in two of them color change was reported to have existed for several months.

We postulated that we would find more vital or partial necrotic pulps in teeth treated shortly after the injury than those pulpectomized after a longer time interval. No such correlation was found. As one can see in Table 2, 80% of a total of 21 teeth treated within 2 months after the injury presented a necrotic pulp. Teeth treated after more than 2 months and up to 1 year after presented lower percentages, which is in contrast to our hypothesis.

Penetrating the pulp chamber without a local anesthetic is frequently employed when pulp vitality is doubtful. The lack of sensitivity and/or bleeding during extirpation, in these circumstances, confirms that the pulp is necrotic. If pain is eventually experienced, most adult patients react favorably after receiving local anesthetic. The same procedure could lead to disruptive behavior in an apprehensive child. For this reason, the teeth were anesthetized in three patients despite the suspicion of pulp necrosis. In only four cases of necrotic pulp, the patient complained of pain or discomfort during debridement, possibly due to overinstrumentation. Diagnostic periapical radiographs were used to estimate the root canal length. This, by itself, cannot rule out apical bleeding from resorbed (and nonresorbed) roots. It is therefore possible that some teeth with a totally necrotic pulp were diagnosed as partial necrotic due to over, instrumentation. If this is the case, the finding that 77% of the teeth had a necrotic pulp may be even regarded as underestimated.

<table>
<thead>
<tr>
<th>Pulp Condition</th>
<th>Yes (N-Y-IP)</th>
<th>No (N-Y-IP)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Necrotic</td>
<td>4 (2-0-2)</td>
<td>33 (31-0-2)</td>
<td>37</td>
</tr>
<tr>
<td>Partial Necrotic</td>
<td>9 (1-1-7)</td>
<td>1 (1-0-0)</td>
<td>10</td>
</tr>
<tr>
<td>Vital</td>
<td>1 (0-0-1)</td>
<td>0 (0-0-0)</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>14 (3-1-10)</td>
<td>34 (32-0-2)</td>
<td>48</td>
</tr>
</tbody>
</table>

N = no local anesthesia; Y = infiltration of local anesthesia; IP = intrapulpal anesthesia.
of time in the past, these teeth may not have presented any clinical or radiographic evidence of pulp necrosis. Some of the discolored teeth in our study might eventually develop pathologic evidence of necrosis if left untreated. Such pathologic reactions were considered contraindications for pulpectomy with extraction being the treatment of choice. This led to the operator’s decision to treat endodontically teeth with dark-gray discoloration of the crown even before clear evidence of pulp necrosis existed.

Partial or total necrosis was found in discolored asymptomatic teeth, 6 and even up to 24 months after the reported injury or color change. This might be attributed to a slow degenerative process occurring in the pulp chamber. Andreasen stated that sterile necrosis can occur without any radiographic sign of periodontal involvement. Sonis observed that 72% of primary incisors with gray-black discoloration following concussion or subluxation failed to develop radiographic and/or clinical evidence of pathosis. However, an aseptic necrotic pulp is an excellent growth culture medium for anaerobic bacteria and can become infected in cases of bacteremia. This infection may deteriorate into a periapical rarefaction and a sinus tract. The teeth examined in this study belonged to a selected group presenting dark-gray discoloration of the crown, and some of them were found to be nonvital during pulpectomy. Therefore, they do not represent the overall effect of traumatic injuries to primary incisor dental pulp.

Dark-gray discoloration may fade and the tooth regain its normal shade or may become yellow, reflecting calcification of the pulp. Schroder et al. reported 11 of 75 dark-gray discolored primary incisors, became yellow or returned to normal color. In our study, the tooth in which the pulp was found to be vital might have changed its dark-gray discoloration to normal or yellow if not treated endodontically. A relatively high percentage of teeth were found to be necrotic. One may assume that this is due to the fact that the teeth included in this study were selected for endodontic treatment. However, of the 37 necrotic teeth, 27 (73%) were not sensitive to percussion despite the dark discoloration. Moreover, half of the necrotic discolored teeth were completely stable, and 46% of teeth with a necrotic pulp did not present periapical osteitis. These findings show that discolored primary incisors can be necrotic even without presenting tenderness to percussion, increased mobility, and periapical osteitis.

It is often difficult to distinguish periapical osteitis from the radioluency of the dental sac of the permanent incisor over the apical area of its primary predecessor.

The results of this study agree with previous studies. Schroder found that 82% of injured primary incisors with gray discoloration developed periapical osteitis less than 1 month after injury, indicating pulp necrosis. Auslander stated that “color change into darker and darker hues when accompanied by no response on the pulp tester almost certainly indicates pulpal degeneration.” Bennett confirmed this by saying that in most instances coronal discoloration—especially a gray or bluish color—is diagnostic of pulp death.

**Conclusions**

1. Dark-gray discoloration of primary incisors could be interpreted as an early sign of pulp degeneration that would deteriorate into necrosis
2. Dark-gray discolored primary incisors can be necrotic even without presenting tenderness to percussion, increased mobility, and periapical osteitis.

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