# Calcific metamorphosis: A challenge in endodontic diagnosis and treatment

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Calcific metamorphosis (CM) is seen commonly in the dental pulp after traumatic tooth injuries and is recognized clinically as early as 3 months after injury. Calcific metamorphosis is characterized by deposition of hard tissue within the root canal space and yellow discoloration of the clinical crown. Opinion differs among practitioners as to whether to treat these cases upon early detection of CM or to observe them until symptoms or radiographic signs of pulpal necrosis are detected. In this review, the clinical, radiographic, and histopathologic appearance of CM is described; a review of the literature is presented to address these issues in an attempt to establish a sound rationale for treatment. Approximately 3.8% to 24% of traumatized teeth develop varying degrees of CM. Studies indicate that of these, approximately 1% to 16% will develop pulpal necrosis. Most of the literature does not support endodontic intervention unless periradicular pathois is detected or the involved tooth becomes symptomatic. It may be advisable to manage cases demonstrating CM through observation and periodic examination. (*Quintessence Int 2001,32:* 447–455)

Key words: calcific metamorphosis, calcification, pulp canal obliteration, pulp disease, trauma

Calcific metamorphosis (CM) is defined as a pulpal response to trauma that is characterized by deposition of hard tissue within the root canal space.<sup>1</sup> It has also been referred to as pulp canal obliteration.<sup>2–5</sup> CM is seen commonly after traumatic tooth injuries<sup>6</sup> and is recognized clinically as early as 3 months after injury, but in most instances it is not detected for about 1 year.<sup>7–9</sup> Although the exact mechanism of CM is still unknown, damage to the neurovascular supply of the pulp is probably related significantly to this process.<sup>10–12</sup> Teeth presenting with radiographic evidence of CM are considered to be undergoing pathologic changes by some clinicians.<sup>9,13</sup> In this respect, the diagnostic status and treatment planning decisions regarding teeth with CM remain controversial.<sup>8,14–16</sup>

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## ETIOLOGY AND INCIDENCE

Calcific metamorphosis occurs commonly in young adults because of trauma. It is evident usually in the anterior region of the mouth and can partially or totally obliterate the canal space radiographically, 78:13:14,17-19 The clinical picture of CM has been described by Patterson and Mitchell<sup>13</sup> as a tooth that is darker in hue than the adjacent teeth and exhibits a dark yellow color because of a decrease in translucency from a greater thickness of dentin under the enamel (Fig 1). These authors considered CM a pathologic condition and recommended either root canal treatment or extraction of the tooth. It was reasoned that the pulp tissue involved should be regarded as a potential focus of infection and therefore should be removed. Histologically, however, no evidence of pulpal pathosis could be demonstrated as a result of poor fixation of the pulp tissue. Table 1 summarizes studies that indicated the frequency of pulp necrosis following CM.

Holcomb and Gregory<sup>14</sup> examined 881 patients and found that 41 teeth in 34 patients exhibited CM, representing an incidence of 3.8%. Over a 4-year period, only three of the 41 teeth (7.3%) developed periradicular rarefactions. They concluded that CM may be a pathologic deviation from the normal pulp. As a result, the only definitive criterion for choice of treatment for these teeth was the appearance of a periradicular rarefaction.

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TABLE 1 Frequency of necrosis following calcific metamorphosis (CM) in permanent teeth

Study	Mean observation period (y)	No. of units studied	No. of teeth with CM	No. of teeth with pulpal necrosis
Holcomb and Gregory 196714	4	88 patients	41	3 (7%)
Andreasen 19707	1-12 (3.4)	189 luxated teeth	42	3 (7%)
Stälhane and Hedegard 1975*	13-21	76 teeth with CM	76	12 (16%)
Jacobsen and Kerekes 197720	10-23 (16.0)	122 traumatized teeth	122	16 (13%)
Andreasen et al 198717	1-10 (3.6)	637 teeth	96	1 (1%)
Robertson et al 1996*	7-22 (16)	82 traumatized teeth	82	7 (8.5%)

Andreasen' conducted a follow-up study of 189 luxated permanent teeth with a mean observation period of 3.4 years. Pulp canal obliteration was found in 42 teeth (22%) and was related significantly to the variable stage of root development. Calcific metamorphosis was more common in teeth with incomplete root development and crown fracture and was related to the type of luxation injury. Calcific metamorphosis was considered to be an accelerated deposition of dentin in response to trauma, and early endodontic intervention was not supported. In a similar study with a 5-year follow-up of 637 permanent incisors, only 15% developed CM, and only 1% of those developed pulp necrosis.<sup>17</sup>

Stälhane and Hedegard<sup>5</sup> conducted a long-term study on 76 teeth with CM following trauma. The teeth were examined 3 to 21 years after the accident. Twelve of the 76 teeth (16%) developed a periradicular rarefaction during the observation period. The authors stated that in making treatment decisions, the success rate enjoyed by modern endodontic treatment must be weighed against the percentage of teeth that become necrotic secondary to CM. The possibility that additional trauma, subsequent caries, or orthodontic movement could contribute to the periradicular changes in these cases was highlighted. Jacobsen and Kerekes<sup>20</sup> conducted a follow-up study of 122 traumatized teeth with radiographic evidence of hard tissue formation in the pulp cavity for a mean of 16 years after injury. Partial canal obliteration was identified in 36% of the cases, with total canal obliteration in 64%. Of the total population studied, 13% eventually developed pulp necrosis. These findings support those of Holcomb and Gregory<sup>14</sup> in that the rationale for endodontic treatment should be the development of a periradicular radiolucency.

A retrospective study of 517 traumatized teeth by Rock and Grundy<sup>a</sup> showed that 16% of the teeth developed CM, with CM mostly occurring in the younger age group and resorption occurring in the older age group. Root canal treatment was recommended as soon as narrowing of the pulp chamber shadow is seen radiographically, based on two clinical parameters: (1) Once the guidance afforded by the pulp canal is lost, it is more difficult to prepare a post hole without penetrating the periodontal ligament; and (2) should necrosis occur in the remaining apical tissue, the only possible access may be surgical intervention.

One of the most recent studies that addressed CM consisted of 82 traumatized permanent incisors that were followed for a period of 7 to 22 years (mean 16

years).4 In that study, a clinical finding of yellow discoloration of the teeth was observed frequently. During the observation period, periradicular bone lesions suggesting pulp necrosis developed in seven teeth (8.5%). The 20-year pulp survival rate was 84%. and no higher frequency of pulp necrosis was observed in teeth with CM that were subjected to caries, new trauma, orthodontic treatment, or complete-crown coverage when compared to intact teeth. The conclusion that the incidence of pulp necrosis in teeth displaying CM seems to increase over time was not supported, and routine endodontic intervention on teeth with CM was not justified.

# **RADIOGRAPHIC INTERPRETATION**

The radiographic appearance of CM is partial or total obliteration of the pulp canal space with a normal periodontal membrane space and intact lamina dura.6 A thickening of the periodontal ligament space or periradicular radiolucency may be observed with or without subjective symptoms (Fig 2). Complete radiographic obliteration of the root canal space, however. does not necessarily mean the absence of the pulp or canal space; in the majority of these cases there is a pulp canal space with pulpal tissue.8,15,19,21

## HISTOPATHOLOGY

Histopathologic studies designed to assess the pulpal status of teeth with CM have failed to show any inflammatory component indicative of a pathologic process.8.13,19,22 This may be a result of multiple causes, including but not limited to poor tissue fixation, specimen sectioning, and the investigator's interpretation. On the other hand, Patterson and Mitchell13 considered CM a pathologic process but could not prove it histologically because they based their conclusion on one case with a poorly fixed pulpal tissue.

The pulps of 20 maxillary permanent incisors were evaluated microscopically by Lundberg and Cvek.19 The teeth were treated endodontically because of progressive hard tissue formation in the canal space. The tissue changes were characterized by a varied increase in collagen content and a marked decrease in the number of cells. Osteoid tissue with included cells was found adjacent to mineralized areas in the pulp, with only one pulp showing moderate lymphocytic inflammatory infiltrate because of further trauma. They concluded that tissue changes in the pulps of teeth with CM do not indicate the necessity for root canal treatment.

Torneck\* described CM as a tertiary dentin response to trauma that is highly irregular in pattern Fig 2 Periradicular radiograph of multiple mandibular incisors with varying degrees of CM demonstrates different outcomes. The mandibular right lateral incisor shows complete obliteration and normal lamina dura.

as well as complete pulpal obliteration and development of a periradicular radiolucency. The mandibular left central incisor shows partial pulpal obliteration and development of a periradicular lesion. (Case courtesy of Dr Anthony McNaught )

and calcification and contains a maze of small irregular spaces and cul-de-sacs that extend from the pulp chamber to the apical foramen. Such irregularities were less common when the rate of calcification was slower, with the dentin deposition occurring only on the periphery of the pulp space. The dentin structure was also more regular, being principally of a tubular type that is supported at times by a small but identifiable pulp in the more central portion of the root.

Fischer<sup>21</sup> indicated that CM was a response to trauma with progressive hard tissue formation, with maintenance of vital tissue and a pulp space observed up to the apical foramen (Fig 3). He argued, however, that such cases require root canal treatment because of reduced cellular content leading to decreased ability for healing, therefore making the pulpal tissue more susceptible to infection.

The histopathologic appearance of pulp canal obliteration in traumatized primary incisors shows three types of calcific tissue occluding the pulp lumen: dentin like, bone like, and fibrotic.23 Recently, Holan<sup>6</sup> described tube-like structures that extended along the entire length of the pulp canal. These were separated from the root dentin by normal pulp tissue but connected to the dentin in some of the sites evaluated. The





structures had a histologic appearance of osteodentin, with cellular inclusions in ring-like formations (Fig 4).

## MECHANISM OF HARD TISSUE FORMATION

The mechanism of hard tissue formation during CM is not yet clear. Several hypotheses have been proposed to explain this phenomenon. Torneck<sup>8</sup> hypothesized that the deposition of hard tissue is either as a result of stimulation of the pre-existing odontoblasts or by loss of their regulatory mechanism. On the other hand, Andreasen and Andreasen<sup>2</sup> described CM as a response to severe injury to the neurovascular supply to the pulp, which after healing leads to accelerated dentin deposition and is closely related to the loss and re-establishment of the pulpal neural supply. Neither mechanism has been proven or studied, and further investigation is required to provide an evidence-based understanding of this occurrence.

Calcific metamorphosis is characterized by an osteoid tissue that is produced by the odontoblasts at the periphery of the pulp space or can be produced by undifferentiated pulpal cells that undergo differentiation as a result of the traumatic injury. This results in a simultaneous deposition of a dentin-like tissue along the periphery of the pulp space (root canal walls) and within the pulp space proper (root canal). These tissues can eventually fuse with one another, producing the radiographic appearance of a root canal space that has become rapidly and completely calcified. Fig 3 (*ieft*) Histologic section of a tooth with evidence of complete pulp canal obliteration radiographically shows irregular deposition of tertary denth along the length of the pulp coronal to the apical loramen. Note the gaps indicating that complete obliteration did not exist. (Original magnification ×100, hematoxylineosin stain.)

Fig. 4 (below) Transverse section of a tooth shows concentric deposition of tertiary dentin, with entrapped cells giving rise to an osteodentin-like appearance. (Original magnification ×16; hematoxylin-eosin stein.)



Ten Cate<sup>24</sup> identified this process as the deposition of tertiary or reparative dentin in response to irritation or trauma. Reparative odontoblasts are somehow able to differentiate from dental pulp cells in the absence of any epithelial influence. During the development of the tooth, the undifferentiated ectomesenchymal cell of the dental papilla divides into two daughter cells. One daughter cell is influenced by the epithelial cells and differentiates into an odontoblast, while the second daughter cell that is not exposed to the epithelial influence persists as a subodontoblast cell, which under certain influences differentiates into odontoblast-like cells and deposits dentin-like hard tissue.

Reparative dentin or tertiary dentin is deposited at specific sites in response to injury, and rate of deposition depends on the degree of injury. The more severe the injury, the more rapid the rate of dentin deposition, with possibly as much as 3.5 µm in a single day. This results in accelerated hard tissue formation that traps some pulpal cells and gives the histologic appearance of osteodentin with an irregular tubular pattern (Figs 5 and 6). Evidence indicates that reparative dentin is produced by odontoblast-like cells and incorporates type I and III collagen in its matrix, which exhibits diminished phosphophoryn content.<sup>24</sup>

The cells constituting this hard tissue originate from cell divisions in deeper layers of the pulp, and the type of cells that divide has not been established. They might be undifferentiated perivascular cells, pulpal fibroblasts, or cells formed from the odontoblast lineage but not exposed to the final epithelial influence.<sup>24</sup>



These newly differentiated cells first express a mixture of collagen (including types I, II, III, and IV), which forms a matrix surface. The deposition of fibronectin on predentin provides the mechanism for positioning the cells that then produce a matrix of type I and II collagen that accepts mineral in the absence of phosphophoryn. There is much discussion as to whether the mineralized tissue so formed is truly dentin, because the original odontoblasts express type I collagen and phosphophoryn.<sup>25</sup>

## MANAGEMENT OF CANALS WITH CALCIFIC METAMORPHOSIS

The management of canals with CM is similar to the management of pulpal spaces with any form of calcification. Usually, the teeth involved with CM are anterior teeth that were subjected to trauma at a young age. The literature supports the fact that most teeth with the radiographic appearance of CM exhibit a persisting narrow pulp canal space that is not usually detectable radiographically.<sup>8,14-16,20,21,26</sup> Even under these circumstances, most canals can be located and negotiated.<sup>14,19</sup>

To locate the calcified orifice, the practitioner first mentally visualizes and projects the normal spatial relationship of the pulp space onto a radiograph of the calcified tooth. Then, the two-dimensional radiographic image is correlated with the three-dimensional morphology of the tooth.<sup>27</sup> Thereafter, access preparation is initiFig 5 (left) Irregular linear calcilication with highly irregular tubular pattern and gnarled appearance of the reparative dentin. (Original magnification ×100; hematoxylin-eosin stain.)

Fig 6 (below) Histologic section shows local deposition of reparative dentin as a response to a localized injury to the pulp. Note the nature of localized deposition compared to calcific metamorphosis, in which the reparative dentin formation is more linear and involves the entire pulp canal. (Original magnification ×25, Brown-Brenn stain.)



ated, with the rotary instrument directed toward the presumed location of the pulpal space. This approach requires knowledge of the normal pulp chamber location, tooth canal anatomy, and the long axis of the roots. Accurate radiographs are essential for preoperative visualization and periodic assessment of bur penetration and orientation. Finally, the practitioner must be able to recognize the calcified orifice when it has been reached.

#### Normal root anatomy

In the past, textbooks on root canal morphology have often overlooked an important anatomic fact: The canal space is always located in the cross-sectional center of the root. Similarly, the pulp chamber is (or was, before calcification) located in the cross-sectional center of the crown<sup>27</sup> (Fig 4).

In a tooth with a calcified pulp chamber, the distance from the occlusal surface to the projected pulp chamber floor is measured from the preoperative radiograph. An access cavity of normal size and shape is created in the crown to a depth equal to that of the pulp chamber floor in a noncalcified tooth.

A second important aspect of normal root canal anatomy is the geometric pattern of canal orifices found in the pulp chambers of teeth with multiple canals.<sup>28</sup> These geometric patterns and their potential variations must be mentally projected onto the calcified pulp chamber floor, with consideration for the direction of the canals as they leave the pulp chamber. This requires an astute integration of two-dimensional radiographic findings with three-dimensional tooth anatomy, coupled with a safe and dexterous movement of the rotary instrument on the pulpal floor.

## Maxillary central and lateral incisors and canines

In maxillary incisors, the root canal is located in the cross-sectional center of the root<sup>29</sup> (Fig 4). If esthetics and structural integrity were disregarded, the ideal location of the access preparation would be through the incisal edge; however, the standard access preparation for this tooth is in the exact center of the palatal surface of the crown buccolingually and incisogingivally. At an angle of roughly 45 degrees to the long axis, bur penetration of 3 to 4 mm will generally intersect with the pulp chamber in average-sized teeth. In a calcified chamber, however, continued penetration at 45 degrees to the long axis will eventually pass over the canal entirely and result in perforation of the labial root surface below the gingival attachment. Therefore, when the chamber is calcified and the canal has not been located after 3 to 4 mm of penetration, the bur must be rotated to be as parallel to the long axis of the tooth as possible to prevent perforation. Penetration proceeds down the lingual aspect of the access preparation, with frequent exploration for the orifice with the DG-16 endodontic explorer. In deep excavations, the bur may be changed to a longshank No. 2 round bur with frequent visual and radiographic reassessment of direction.

## Mandibular incisors and canines

The most common canal morphology for these teeth is a single canal; however, a second canal, if present, will almost invariably be found lingual to the first.29,30 In incisors and canines, second canals are particularly difficult to locate (even where minimal calcification is present) because of angulation of the anatomic crown or the location of the standard access cavity on the lingual aspect. After the main canal is located and debrided, it is important to widen the orifice lingually and probe for the second orifice using a No. 8 or 10 K file with an abrupt curve placed 1 or 2 mm from the tip of the file. If the canal is not located with this technique, the use of No. 2, 3, and 4 Gates-Glidden drills on the lingual surface may be very helpful in uncovering the orifice of a lingual canal. The drill is used in the manner of the round bur and is drawn up the lingual surface in a sweeping motion. With the advent of canal-orifice shapers, the technique of increasing the orifice has been enhanced.

# Location and penetration

The most important instrument for orifice location is the DG-16 explorer. In firm probing during excavation of the pulp chamber floor, the explorer will not penetrate and "stick" in solid dentin: however, if an orifice is present, firm pressure will force the instrument slightly into the orifice, and it will resist dislodgment. or stick. To minimize perforation, reconfirm the location of the canal radiographically, leaving the explorer in place. At this point, a fine instrument, usually a No. 8 or 10 K file, is placed into the orifice, and an attempt is made to negotiate the canal. Some practitioners prefer to use a No. 6 K file initially to negotiate the canal: however, these instruments are very fine and lack stiffness in their shafts. If the canal is highly calcified or packed with necrotic debris, the No. 6 K file will bend and curl instead of penetrating. An alternative option is to use instruments with reduced flutes, such as a Canal Pathfinder (IS Dental), or instruments with greater shaft strength, such as the Pathfinder CS (Kerr), which are more likely to penetrate even highly calcified canals. Here also, the canal Orifice Shapers (Dentsply) will enhance rapid canal penetration.

When faced with trying to locate the canal orifice, many practitioners have chosen to use magnification in the form of enhanced glasses or a microscope. Although it may be advantageous to be able to see the position of the orifice under magnification, this approach will not aid the clinician who does not know where to look for the orifice. The anatomic features of the pulp chamber floor are an essential tool for locating the orifices and should be preserved as much as possible Examining the color changes in the floor with high magnification will aid in locating canal orifices.

Because of curvature in the coronal 1 or 2 mm of many canals, it is necessary to remove the cervical ledge or bulge.31 If the orifice still cannot be negotiated with a fine instrument, drill 1 or 2 mm into the center of the orifice with a No 2 round bur on slow speed and use the explorer to re-establish the canal orifice. When counter sinking or troughing in an area where an orifice is located, he sure that the pulp chamber is dry. The slow-speed rotating bur will remove white dentin chips that accumulate in the orifice. After a light stream of air is blown into the chamber, these dentin chips appear as white spots on the dark floor of the chamber and serve as markers for exploration or further counter sinking. Upon entry, the file is carefully rotated and teased apically around the canal curves. Chelating agents such as REDTA (Roth Drug), RC-prep (Premier Dental Products), and Glyde (Dentsply) are seldom of value in locating the orifice but can be useful during canal negotiation.32

## Penetration and negotiation

Once the orifice has been located, it is advisable to enlarge the coronal third to improve tactile sensation and better apical penetration. A 21-mm No. 8 K file is the initial instrument of choice to negotiate the calcified canal. A No. 10 K file is usually too large, and a No. 6 K file is too weak to apply any firm apical pressure, particularly if curved. Likewise, the use of nickel-titanium files is contraindicated for this purpose because of lack of strength in the long axis of the file. The 21-mm-long K file is flexible enough to negotiate around curvatures of calcifications. If the canal is longer than 21 mm, it is simple to change to a 25-mm instrument once 21 mm of penetration has been achieved. Before the file is inserted into the canal, a small curve is placed in its apical 1 mm. The point on the rubber stop is then aligned with the curve. In negotiating the fine-curved canal, the curved instrument must be directed along the pathway the canal is most likely to follow; consequently, it is vitally important to know what direction the curve in the instrument is pointed. This is easily accomplished by observing the rubber stop on the instrument shaft

#### Helpful considerations

- Irrigate copiously at all times with 2.5% to 5.25% NaOCI, which enhances dissolution of organic debris,<sup>33</sup> lubricates the canal, and keeps dentin chips and pieces of calcified material in solution.
- · Advance instruments slowly in calcified canals.
- Clean the instrument on withdrawal and inspect it before reinserting it into the canal.
- Do not remove the instrument when it has reached the approximate canal, rather, obtain a radiograph to ascertain the position of the file.
- Do not use acids (hydrochloric acid) or alkalis (sodium hydroxide) to aid in canal penetration.
- Use chelating pastes or solutions to assist in canal penetration.
- Use ultrasonic instruments in the pulp chamber to loosen debris in the canal orifices.
- Flare the canal orifice in a crown-down fashion and enlarge the negotiated canal space to improve tactile perception in continued canal penetration.
- Use of newer, nickel-titanium rotary orifice-penetrating instruments should be considered when possible.

#### Prognosis

The prognosis of teeth with CM treated with nonsurgical root canal treatment has been investigated by Cvek et al.<sup>18</sup> They examined 54 nonvital incisors with posttraumatically reduced pulpal lumina with a follow-up of 4 years. Healing was found in 80%. They filled the canals with resin-chloroform and gutta-percha points. This could be another factor involved in decreasing the success rate, because considerable shrinkage in gutta percha occurs when chloroform evaporates.<sup>34</sup> There have been no studies that compare the relative success rates of calcified canals treated using a surgical approach.

Adherence to the principles of radiographic interpretation, recognition of the presence of CM with pathologic sequelae, and use of the techniques described in managing these cases will in many cases result in successful management of a case that may have been deemed untreatable (Figs 2 and 7 to 9).

## SURGICAL ENDODONTIC CONSIDERATIONS

Often, symptomatic teeth that exhibit complete CM radiographically or in which the canals cannot be negotiated must be treated with periradicular surgery,15,16 Subsequent to trauma, a rapid, disorganized calcification that characterizes CM can occur; pulpal remnants may become entrapped in this calcification. Ultimately, these pockets of tissue necrose but are contained within the dentin. Once a root-end resection is performed, many of the pockets of necrotic tissue may be opened to the periradicular tissues, resulting in persistent chronic inflammation with possible sinus tract involvement subsequent to surgery.35,36 Even a second root-end resection does not solve the problem, and ultimately extraction may occur. If this situation truly exists, very little can be done short of complete root-end fill of the entire root face, as suggested many years ago by Castenfeldt,35 or possibly using a surface layer of glass ionomer or a layer of composite.37-39 Presently advocated techniques of preparing a concave root face, followed by a dentin bonding agent and resin composite root-end fill, appear promising and may negate the problem of contaminated tissue debris at the resected root end. 39,40

### SUMMARY

Approximately 3.8% to 24% of traumatized teeth develop varying degrees of calcific metamorphosis. Although there are different opinions on the management of pulps exhibiting canal obliteration, studies indicate that the incidence of pulpal necrosis in these teeth is between 1% and 16%. Histologic examination of pulpal tissue from teeth demonstrating closure of the pulpal lumina revealed no significant inflammation





Fig 7 Periradicular radiograph shows working-length determination and successful negotiation of the canal space. (Case courtesy of Dr Anthony McNaught.)

Fig 8 Postoperative radiograph of case shown in Fig 7 with successful cleaning, ahaping, and obturation of the canal space. (Case courtesy of Dr Anthony McNaught.)



Fig.9 Six-month follow-up radiograph shows decrease in size of the periradioular lesions and reformation of the lamina dura compared to the preoperative radiograph (see Figs.2 and 7). (Case courtesy of Dr Anthony McNaught.)

that would warrant root canal treatment. Most of the literature does not support endodontic intervention unless periradicular pathosis is detected or the involved tooth becomes symptomatic. Because the overall failure rate of nonsurgical root canal treatment is between 10% and 19%, it may be advisable to manage cases demonstrating calcific metamorphosis through observation and periodic examination. If the pulp tissue becomes necrotic and a periradicular radiolucency develops, nonsurgical root canal treatment has been shown to be successful 80% of the time.

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