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Comparative evaluation of Ca(OH)₂ plus points and Ca(OH)₂ paste in apexification

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Immature permanent incisors with open apices may lose their vitality as a result of trauma. Apexification is the process that allows the formation of a calcific barrier across the open apex, after pulp death, by creating a suitable environment within the root canal and periapical tissues. Calcium hydroxide $[Ca(OH)_2]$ has been the material of choice for apexification since 1964 when Kaiser first reported its capacity to induce physiological closure of immature pulpless teeth (1, 2). Published case reports and several long-term studies have stated success rates of 74–100% for Ca(OH)₂ apexification (3–14).

An alternative treatment to $Ca(OH)_2$ apexification is using an artificial apical barrier with mineral trioxide aggregate (MTA) that allows immediate obturation of the canal. Although at present no long-term follow-up study is available on MTA apexification, this procedure has steadily gained popularity among clinicians because of its short treatment time, and it has had reported success rates of 77–100% (15, 16). Despite its popularity, the outcomes of MTA apexification do not vary greatly from those of Ca(OH)₂ apexification. Thin dentinal walls still present a clinical problem. Moreover, associated high costs and difficulties in handling the material may restrict it widespread use (15, 17).

Biologically based treatment approaches could be of particular value in the treatment of necrotic immature permanent teeth in terms of restoring root development and reinforcing dentinal walls. Although the precise mechanisms and outcomes of such approaches are unknown, an increasing number of case reports have shown the treatment to result in regeneration of nonvital immature permanent teeth (18, 19), and the procedure has had a reported success rate of 78% (20).

Regenerative endodontic procedures or MTA apexification should be more preferable approaches for treatment of nonvital immature teeth but in cases where no signs of success have been observed after 3 months of treatment, more traditional methods are recommended (17). In such cases, $Ca(OH)_2$ apexification is indicated. However, $Ca(OH)_2$ paste has certain inherent disadvantages, including causing very brittle dentinal walls and high risk of root fractures, eventual conversion to calcium carbonate, retention of paste in the root canals before final obturation and interaction with zinc oxide eugenol-based sealers (21–24).

Calcium hydroxide plus points (CHPPs) (Roeko, Langenau, Germany) designed to release calcium hydroxide from a gutta-percha matrix have recently been developed as an alternative to $Ca(OH)_2$ paste for use as an intracanal dressing between appointments (25) and to treat root resorption (26, 27). However, there are no published reports available on the effectiveness of CHPPs in apexification treatment.

CHPPs are 28 mm in length, light brown in color and are available in ISO sizes 15–140. They contain 58% calcium hydroxide, 35–37% gutta-percha, and coloring agents and have handling characteristics similar to those of conventional gutta-percha points (28).

Densitometric measurement has been used in implantology as well as in assessing the relationship between oral bone loss and osteoporosis, measuring bone density after periodontal treatment and measuring the optical density of various dental materials (29, 30). However, there are no published reports available that densitometric measurement has been used to quantify the newly formed apical barrier following apexification treatment.

The aims of this study were as follows:

- 1 To compare clinically and radiographically the effectiveness of calcium hydroxide paste (CHP) and guttapercha points containing calcium hydroxide in the apexification of immature upper central incisors;
- **2** To identify any clinical variables and inter-appointment symptoms that correlate with closure time and overall success rates; and
- **3** To identify densitometric changes in the apical regions of treated teeth on radiographs taken using paralleling cone technique, step-wedge, and digital radiography system.

Material and methods

A total of 16 children (nine girls, seven boys) were selected from among patients attending the clinic of the Pediatric Dentistry Department of Ankara University in Ankara, Turkey. Each child had at least one upper central incisor requiring apexification, for a total of 22 teeth. None of the teeth had received previous treatment for necrosis. Children's ages ranged from 7 to 11 years, with a mean age of 9.06 years. All children were healthy and cooperative. All apexification treatments were performed by the same pediatric dentist following receipt of ethical approval and informed parental consent. This study has been approved by the Ankara University, Faculty of Dentistry, Institutional Review Board.

Inclusion criteria for teeth included the following: (i) clinical and radiographic evidence of irreversible pulpitis or pulp necrosis as a result of previous trauma; (ii) no pathological external or internal root resorption, periapical bone destruction, root fracture, pulp calcification, ankylosis, or severe luxation injury; (iii) possibility of resin composite restoration; and (iv) root formation at Stage 7, according to Moorrees (31). Pulpal necrosis was diagnosed by dental history and clinical examination also included electric pulp testing (Digitest; Parkell, Farmingdale, NY, USA) and cold stimulation testing (Chloraethyl, Wehr, Baden, Germany). Clinical symptoms of irreversible pulpitis such as spontaneous pain or pain persisting after the disappearance of existing stimulus and sensitivity to pressure were also considered.

The study examined the effects of the variables age, sex, trauma type, time lapse between trauma and initiation of treatment, preoperative and inter-appointment symptoms (abscess, fistula, swelling, spontaneous pain, sensitivity to pressure), and total treatment time from initial instrumentation to completion of apexification on treatment success and closure time.

Treatment procedures

Teeth were randomly distributed between the treatment groups (10 CHP, 12 CHPP). For both groups, teeth

were isolated with a rubber dam (OptraDam; Ivoclar Vivadent AG, Schaan, Liechtenstein), and a large access was made using a diamond round bur (801H-016; Hager&Meisinger GmBH, Heisinger, Germany) to allow removal of all necrotic tissue. After removing all necrotic pulp tissue using a large K-file, teeth were instrumented to 1 mm short of the radiographic apex using a gentle, circumferential filing motion and copious irrigation with 2.5% sodium hypochlorite (NaOCI) to maximize cleansing and to minimize dentin removal. Final irrigation was performed using a sterile saline solution, and the canal was dried using large, sterile paper points arranged to the appropriate working length.

Calcium hydroxide was applied upon completion of the initial instrumentation appointment. For the CHP group, paste (Metapaste; Meta Biomed, Chungbuk, Korea) was injected using material's own syringe with a disposable tip placed to the working length and withdrawn on placement and then slightly condensed using a sterile cotton pellet. For the CHPP group, in line with the manufacturer's recommendation that moist air be allowed to circulate freely around the point, a point of appropriate size was selected, moistened with sterile saline solution, and inserted into the canal to the working length, followed by one or two smaller points, and the extending lengths were cut off using a sterile scissors. All canals were sealed with cotton pellets and reinforced zinc oxide eugenol cement (IRM, Dentsply, Milford, DE, USA).

Clinical and radiographic examinations

All cases were reviewed at 1 week following initial placement of the apexification medicament and further reviewed every 3 weeks to replace the materials until apexification occurred. In the control appointment, after removal of the temporary restorative material, sterile saline irrigation was used to wash out the CHP from the root canal. In the CHP group, canals were irrigated with sterile saline and dried with paper points, and paste was applied again as described above. In the CHPP group, points were removed with tweezers or a Hedstrom file, canals were irrigated with sterile saline and dried with paper points, and new points were inserted as described above.

Standardized periapical radiographs were taken preoperatively and once every 3 months during follow up. The radiographs were obtained with an intra-oral X-ray system operating at 60 kVp, 7 mA by Heliodent DS (Sirona Dental System GmbH, Bensheim, Germany) and a phosphor plate digital system (Digora Soredex; Soredex Medical Systems, Helsinki, Finland). Exposure time was 0.1 s. These were taken using parallel technique with a XCP system (Rinn, Dentsply, Elgin, Moray, UK) device with a 12 inch cone attached. Standardization was achieved by providing each subject with silicone bite blocks that were used in all radiographic examinations.

Apexification was recorded as either 'successful' or 'unsuccessful' based on the following clinical and radiographic criteria: 'Successful':

- 1 Absence of signs or symptoms of periradicular pathosis
- **2** Presence of a calcific barrier across the apex, as demonstrated by radiographs and tactile examination. 'Unsuccessful':
- 1 Symptoms of pain, tenderness to percussion, swelling, fistulization, or pathological mobility,
- **2** Growth of granulomatous tissue into the canal, manifested by bleeding when files were placed short of the apex,
- **3** Evidence of periradicular pathosis or internal/external root resorption,
- **4** No evidence of root-end closure or resorption of a previously formed apical barrier (13).

The presence of a barrier was assessed clinically every 3 weeks after 6 months of first placement of $Ca(OH)_2$ medicaments by a tactile method and every 3 months using radiographic methods. A barrier was considered present if both findings were positive. The tactile method was performed by introducing a size 15 gutta-percha cone into the canal and slowly tapping it with a finger toward the apex. If an obstruction was met without eliciting pain, it was presumed to be caused by a calcified bridge.

To detect the apical barrier radiographically, image enhancement features with the ability to adjust contrast, brightness, gamma curve, and magnification were used. For this instance, images were magnified by a factor of 25 so that the root anatomy could be displayed entirely on the monitor. If image resolution exceeded that of the monitor, the image was displayed in 1:1 (full size) and a scroll bar was used to view separate parts of the image. Also, contrast, brightness and gamma curve features, histogram equalization, and shadow were used to adjust the gray scale to visualize and examine the apical barrier constitution, which can be manipulated by altering the gradient of gray levels of the image. The barrier was classified as being apically placed if it was within 1 mm from the apex and coronal to the apex in other cases.

Densitometric analysis

All radiographic images were digitally stored and the root apex density values established by an expert radiologist using the Digora device software. The density value of the apical barrier was automatically calculated by measuring a standardized $(2 \times 1 \text{ mm})$ rectangular region of interest after magnifying the images to the maximum scale of 1/25 (with no resolution loss). Apical barrier density values were converted to mmAl by measuring the thickness of the step-wedge that had been prepared earlier and mounted on the sensor while taking each radiographic image. Measurements were recorded for each patient preoperatively, at 3 weeks following initial placement of the apexification medicament and further every 3-month intervals until completion of apexification treatment.

Statistical analysis

Statistical analysis was performed using the program SPSS 12.0 (SPSS 12.0 for Windows; SPSS Inc., Chicago, IL,

USA). Clinical and radiographic data were assessed using Mann–Whitney U and Fisher's exact tests, with a P-value of < 0.05 considered to be statistically significant.

Results

Clinical findings

Of the 22 teeth in 16 patients in this study, 19 teeth had pulp necrosis caused by complicated and noncomplicated crown fractures, one tooth had minimal extrusion, one tooth had subluxation, and one tooth had minimal intrusion. All 22 teeth were available for follow-up evaluations.

Time lapse between trauma and initiation of treatment ranged from 1 to 48 months (mean: 9.9 months). Preoperative acute symptoms including night pain, spontaneous pain, and extreme sensitivity to percussion were observed in seven patients, and preoperative apical abscesses were recorded in two patients. Moreover, seven patients (three in the CHP group and four in the CHPP group) developed painful symptoms during treatment that required an unplanned office visit, four of which were because of the loss of a temporary restoration.

Clinical and radiographic success rates

Apexification treatment was assessed as successful both clinically and radiographically in all 10 teeth (100%) in the CHP group and in 11 of 12 teeth (92%) in the CHPP group; however, one tooth in the CHPP group was clinically and radiographically assessed as unsuccessful owing to persisting pain, tenderness to percussion, swelling, and resorption of a previously formed apical barrier after 8 months of treatment. Difference in success rates between the groups was not statistically significant (P > 0.05). Clinical and radiographic findings related with follow-up times were given in Table 1. Figures 1a–e and 2a–e show radiographs of teeth successfully treated with CHP and CHPP.

The study showed no significant relationship of the success of apexification treatment to type of trauma, post-trauma period, preoperative abscess and acute symptoms and inter-appointment symptoms (P > 0.05).

Apical barrier formation - time

The patients were followed between 6–15 months until apical barrier formation occurred in every individual case. The mean duration of apexification treatment was 9.6 months (SD \pm 2.4) in the CHP group and 9.54 months (SD \pm 2.5) in the CHPP group. The difference in treatment time between the two groups was not statistically significant (P > 0.05).

The mean speed of barrier formation for patients with preoperative apical abscess was 13 months as opposed to 9.2 months for patients without abscess. However, this difference was statistically insignificant (P > 0.05).

Barrier formation was observed in 9 months $(SD \pm 2.1)$ in patients without inter-appointment symptoms and in 11 months $(SD \pm 2.6)$ in patients with

Table 1. Clinical and radiographic findings for each case related with follow-up periods

Cases	3 months		6 months		9 months		12 months		15 months		
	C	R	С	R	C	R	C	R	С	R	Total success
CHP											
1	Ν	Ν	IAS	Ν	Ν	Ν	Ν	Ν	ABF	ABF	100%
2	Ν	Ν	N	Ν	ABF	ABF	-	-	-	-	
3	Ν	Ν	IAS	Ν	Ν	N	Ν	N	ABF	ABF	
4	Ν	Ν	ABF	ABF	-	-	-	-	-	-	
5	Ν	Ν	Ν	Ν	ABF	Ν	ABF	ABF	-	-	
6	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
7	Ν	Ν	N	Ν	ABF	ABF	-	-	-	-	
8	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
9	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
10	IAS	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
CHPP											
11	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	92%
12	Ν	Ν	Ν	Ν	IAS	Ν	Ν	Ν	ABF	ABF	
13	Ν	Ν	N	Ν	Ν	N	ABF	N	ABF	ABF	
14	Ν	Ν	N	Ν	Ν	N	ABF	N	ABF	ABF	
15	IAS	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
16	Ν	Ν	N	Ν	ABF	ABF	-	-	-	-	
17	Ν	Ν	IAS	Ν	F	F	-	-	-	-	
18	Ν	Ν	N	N	Ν	N	ABF	ABF	-	-	
19	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
20	Ν	Ν	IAS	N	ABF	ABF	-	-	-	-	
21	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	
22	Ν	Ν	Ν	Ν	ABF	ABF	-	-	-	-	

inter-appointment symptoms; however, this difference was not statistically significant (P > 0.05).

Apexification time was not affected by patient age, sex, trauma type, post-trauma period, preoperative abscess, or inter-appointment symptoms (P > 0.05); however, treatment time was significantly longer (P < 0.05) in patients with preoperative acute symptoms such as night pain, spontaneous pain, and extreme sensitivity to percussion. Whereas the mean barrier formation time was 8.8 months (SD \pm 2.0) in patients without signs or symptoms, it increased to 11 months (SD \pm 2.5) if signs or symptoms were present.

Apical barrier formation – location

Barriers were located apically in all of the teeth successfully treated with CHPP; however, in the CHP group, the barrier of one tooth was located 2 mm coronally, possibly as a result of a loss of tooth vitality because of an intrusive injury. Barrier location did not differ significantly (P > 0.05) between the two groups, and there was no significant correlation between apical barrier location and type of trauma, time lapse between trauma and treatment, or pretreatment or inter-appointment symptoms (P > 0.05).

Densitometric analysis' results

According to densitometric analysis, the mean optical density of the apical regions of successfully treated teeth was 7.0 mmAl preoperatively and 4.98 mmAl postoper-

atively. Differences in the 3-month optical density values were statistically significant (P < 0.05) (Fig. 3). However, the differences in mean optical density values of the apical barriers between the two groups were not statistically significant (P > 0.05) (Fig. 4), and there were no correlation between optic density values and apexification time or any other clinical variables evaluated (P > 0.05).

Discussion

This study found CHPP to be effective when used in apexification treatment, with apical barriers formed in 11 of 12 teeth treated with CHPP.

MTA apexification and regenerative endodontic treatment are the current approaches highly recommended for treatment of nonvital permanent teeth. $Ca(OH)_2$ apexification is only recommended if the tooth or the patient is not suitable for regeneration procedures or MTA apexification, or if these treatment methods have already been attempted and produced a poor prognosis (17).

The use of Ca(OH)₂ to promote apical closure in nonvital immature teeth is a well-established procedure. The mechanism of action of calcium hydroxide is directly related to its pH, which is influenced by the concentration and rate of release of hydroxyl ions. When used in endodontic therapy, Ca(OH)₂ is activated by the moisture in the canal, potentially raising the pH in the canal to >12. Both the basic pH of CHP and its physical presence within the canal space are thought to possess a

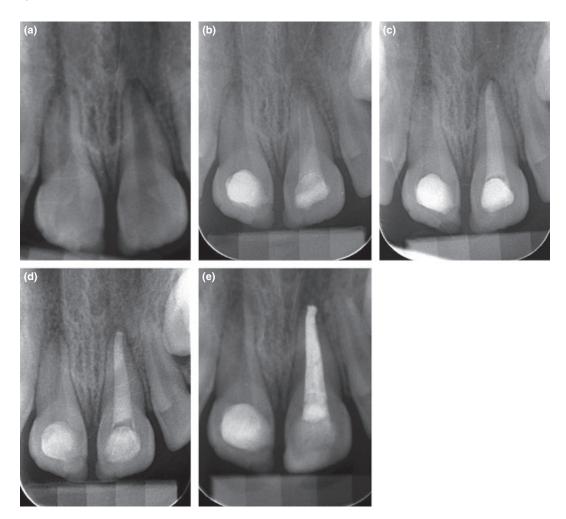


Fig. 1. (a) Preoperative radiograph of tooth 21 treated with calcium hydroxide paste. (b) Three-month follow up. (c) Six-month follow up. (d) Nine-month follow up and apical barrier formation. (e) Final root canal treatment of tooth 21.

potent antibacterial effect, prevent ingress of granulation tissue, and inhibit osteoclastic activity. These characteristics encourage the formation of hard tissue at the root apex (1, 11, 32).

Despite these positive characteristics, CHP has inherent disadvantages. An in vivo study found that 6 weeks after placement of CHP in the apical part of the root canal, 11% of the Ca(OH)₂ had been converted to calcium carbonate, which has very low solubility, a pH of 8, and possesses neither the biological nor the antibacterial properties of Ca(OH)₂ (22). CHP can also make the tooth brittle because of its hygroscopic and proteolytic properties (23). Moreover, complete removal of the paste represents another challenge. Although CHP is routinely removed before final obturation using EDTA + NaOCl + saline and/or instrumentation, these methods are unable to remove all dressing from the canal walls and residual CHP can inhibit the effects of endodontic sealers or prevent their distribution into lateral canals. The residual calcium hydroxide left in the root canals has been shown to interact with zinc oxidebased sealers to form calcium eugenolate that exhibited poor cohesion and granular appearance (21, 24).

The introduction of calcium hydroxide-releasing gutta-percha points that contain calcium hydroxide instead of zinc oxide represents an innovate solution to these problems. When used as short-term medication, Calcium Hydroxide Points (Roeko) exhibited the same clinical success as CHP (25); however, it has been shown that their capacity to provide active ions is limited and that they are unable to alkalize dentine or maintain an alkaline pH within the root canal for more than approximately 1 week (33). Recently, a new type of Ca(OH)₂ gutta-percha points were introduced that contain sodium chloride and tensides in addition to Ca(OH)₂ into the market (CHPP; Roeko). The additional components in CHPP are dissolved upon contact with aqueous solutions, allowing water to penetrate deeper into the gutta-percha points. This enables maintenance of a high pH over a longer period of time and enhances the wettability of the adjacent canal surfaces. Lohbauer et al. (28) reported the release of Ca ions from CHPP to be three times that of calcium hydroxide points. The new points also showed a slightly higher pH value. The pH of CHPP and calcium hydroxide solution was found to be in the range of pH 12. One clinical study

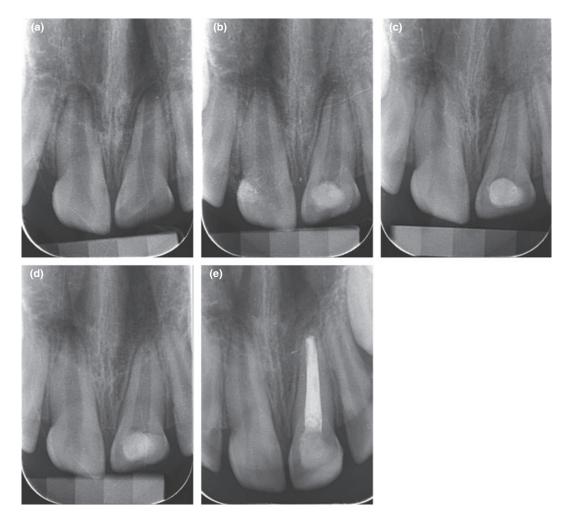


Fig. 2. (a) Preoperative radiograph of tooth 21 treated with calcium hydroxide plus points. (b) Three-month follow up. (c) Six-month follow up. (d) Nine-month follow up and apical barrier formation. (e) Final root canal treatment.

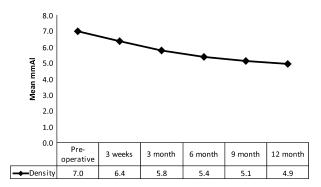


Fig. 3. Mean optical density results (mmAl equivalent) in the apical region for all successfully treated teeth.

found CHPP to be very successful in treating teeth with persistent periapical inflammation (26).

Hedge and Niaz (26) reported $Ca(OH)_2$ points to have a number of advantages over $Ca(OH)_2$ paste, namely, the points are ready to use with no mixing required, and they can be easily inserted and removed from the pulp space with the help of a tweezers, thereby ensuring that there is

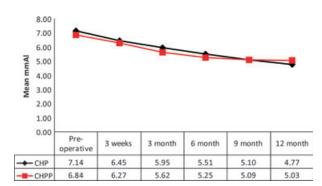


Fig. 4. Mean optical density results (mmAl equivalent) in the apical region for calcium hydroxide paste and calcium hydroxide plus points groups.

no smearing around the access cavity during insertion and no residue left upon removal. Moreover, the points are firm yet flexible enough to follow the natural canal curvature and can easily reach the apex, ensuring that calcium hydroxide is released throughout the canal, which in turn provides enhanced alkalization of outer dentin (34). Despite these advantages, the action of $Ca(OH)_2$ points is short-lived, they lack sustained release, and they are radiolucent.

In line with the manufacturer's recommendations, the CHPP dressings in this study were replaced every 3 weeks, because ion release after this time is insufficient. To provide standardization between the two study groups, the CHP dressings were also changed every 3 weeks. There is no consensus in the literature regarding the frequency of debriding and repacking of CHP in teeth with open apices. Recommendations vary from repacking every 3–6 months (13, 14), repacking only in the presence of symptoms and loss of radiographic density of the paste (3, 10, 11), or repacking only to monitor formation of a hard-tissue barrier (6). Although some authors claim that any reopening and recleaning of the root canal disturbs the process of apexification (5, 12), more recent studies have recommended frequent repacking (22, 35, 36). Finucane and Kinirons (35) reported that the strongest predictor of rapid barrier formation was the rate of change of calcium hydroxide dressings. Those that use several regular dressings claim that regular application of fresh calcium hydroxide promotes a faster healing response and prevents formation of calcium carbonate (22, 35, 36). However, if the points produced a more sustained ionic release, the frequency of repacking the medicament could be decreased from every 3 weeks.

Studies have reported the length of time required for apical barrier formation in CHP apexification to range from as early as 5 months to as late as 39 months (3–14, 35, 36), with more recent studies citing shorter times. The different results reported may be due to differences in study methodology. In the present study, the mean barrier formation time was 9.5 months, with no difference between treatment groups. This result is similar to those of other recent studies. The rapid treatment time in the present study may be related to frequent repacking of $Ca(OH)_2$ materials.

Few apexification studies have examined the effect of time lapse between trauma and treatment on the success of apexification treatment. Thater and Marechaux (8) found a relationship between treatment delay and the time of barrier detection, with a greater delay in seeking treatment correlating with a greater delay in barrier formation. In the present study, the mean time elapsed between trauma and treatment was 9.9 months; however, apexification time was found to be independent of time lapse between trauma and treatment (P > 0.05).

It is possible that age may be inversely related to the time required for apical barrier formation. Mackie et al. (13) reported significantly shorter treatment times among patients older than 11 years of age, although other studies found no correlation between age and apexification time (9, 10). The present study found no relationship between patient age and barrier formation time (P > 0.05). This may be due to the fact that teeth in this study had apical openings of similar sizes.

Heithersay (4) and Cvek (5) reported that the presence of periapical abscess at the initiation of treatment increases the time required for barrier formation; however, other studies found no relationship between pretreatment abscess and barrier formation time (7, 9, 35). In the present study, although patients with pretreatment abscesses required longer treatment times than those without abscesses, the difference was not statistically significant (P > 0.05).

In one study, acute symptoms such as spontaneous pain and night pain were reported to correlate with delayed root closure time (4), whereas another study reported no correlation between pretreatment symptoms and barrier formation time (14). However, in the latter study, no differentiation was made between acute and chronic symptoms. In the present study, the mean apical barrier formation time for teeth with pretreatment acute symptoms was statistically longer (11 months, SD \pm 2.5) than for symptom-free teeth (8.8 months, SD \pm 2.0) (P < 0.05).

Inter-appointment symptoms have also been reported to be associated with an increase in the time required for root-end closure (7, 10, 12). Kleier and Barr (10) reported a delay of approximately 5 months because of bacterial invasion if painful inter-appointment symptoms were present. In the present study, although barrier formation was delayed in teeth with inter-appointment symptoms, the length of delay was not statistically significant (P > 0.05). Moreover, continued formation of the apical barrier was facilitated by re-instrumentation, replacement of CHP or CHPP and appropriate antibiotics except one tooth recorded as failure in the CHPP group.

In the present study, the apical barrier was located at the radiographic apex in most cases. This may be a result of the frequent repacking of apexification material. Previous studies have reported that if calcium hydroxide is not replaced often enough, the barrier is more likely to form coronal to the apex because of an in-growth of tissue from the apex (9, 35).

Radiographic density and gray-level values have been described as important visual characteristics that allow researchers to identify the relationship between dental tissues, distinguish unusual formations, and monitor recovery as well as spread of disease (37). Our study found significant changes in density values during barrier formation that were related to the recovery process. However, no earlier studies exist in this area with which to compare our findings.

Conclusions

Within the limitations of this study, CHPP provided acceptable results in apexification treatment and may be used successfully as an apexification agent in cases where $Ca(OH)_2$ apexification is indicated; however, improvements may be required to produce a more sustained ionic release. Further studies of apexification using CHPP with larger sample sizes and longer follow-up periods would be beneficial and are recommended.

Optical density values of the newly formed apical barrier increased significantly throughout the course of treatment, reaching a final average value of 4.98 mmAl. These values can be used as important criteria in more advanced studies of barrier formation.

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