Dens Evaginatus: Literature Review, Pathophysiology, and Comprehensive Treatment Regimen

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Abstract

Dens evaginatus (DE) is an uncommon dental anomaly, having been well documented since 1925. It occurs primarily in people of Asian descent and is exhibited by protrusion of a tubercle from occlusal surfaces of posterior teeth, and lingual surfaces of anterior teeth. Tubercles have an enamel layer covering a dentin core containing a thin extension of pulp. These cusp-like protrusions are susceptible to pulp exposure from wear or fracture because of malocclusion, leading to pulpal complications soon after eruption. Endodontic intervention of permanent teeth with immature roots is unpredic for inflamed pulps, and leaves a tooth with compromised root structure when treating necrotic pulps. Efforts to ensure root maturity have involved preventive or prophylactic treatment with varying degrees of pulp invasiveness. Treatment options have changed as technology and materials have improved. The goal is to review the literature and pathophysiology regarding DE, and present a new comprehensive treatment regimen, including a truly prophylactic approach without pulpal invasiveness. A case study of a mestizo with DE is documented. Treatment of four affected mandibular premolars exhibiting three distinct diagnostic categories will illustrate various aspects of the treatment protocol presented, and tooth morphology of the anomaly is shown to aid clinical recognition. *(J Endod 2006;32:1–9)*

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Dens evaginatus (DE) is a developmental aberration of a tooth resulting in formation of an accessory cusp whose morphology has been variously described as an abnormal tubercle, elevation, protuberance, excrescence, extrusion, or bulge. This uncommon anomaly projects above the adjacent tooth surface, exhibiting enamel covering a dentinal core that usually contains pulp tissue that on occasion may have a slender pulp horn which extends various distances up to the full length of the tubercle's dentin core (1). The presence of pulp within the cusp-like tubercle has great clinical significance and distinguishes the anomaly from supplemental cusps, such as the cusp of Carabelli (2), which contain no pulp. The cusp of Carabelli has been reported in 17.4 to 90% of the white population, occurring most often on the palatal aspect of the mesiolingual cusp of maxillary first molars, but is a rare occurrence in Asians (1). DE arises most frequently from the occlusal surface of involved posterior teeth and primarily from the lingual surface of associated anterior teeth. Though DE was first reported in 1892 (3), and has been well documented since 1925 (4), the etiology remains undetermined.

DE predominantly occurs in people of Asian descent (including Chinese, Malay, Thai, Japanese, Filipino, and Indian populations) with varying estimates reported at 0.5 to 4.3%, depending upon the population group studied (5). A higher incidence has been observed in specific Eskimo (Alaskan Natives) (up to 15%) and North American Indian (Amerindians) populations (6–8). These patterns suggest an inherited component to the trait, which can be supported by the association DE has with other developmental anomalies (5, 9, 10), such as shovel-shaped incisors that also occur with some frequency in Asian populations (1), DE, mesiodens (11, 12), and three-rooted mandibular molars (13). Both autosomal dominant and X-linked dominant inheritance patterns have been proposed (8, 14, 15).

However, reports tracing a familial history or evidence demonstrating DE occurring in siblings have been few (14). As well, there has been documentation of the unusual occurrence in whites (9, 14) and the rare occurrence in African-Americans (two cases) (16) suggesting the anomaly could be a localized developmental peculiarity, possibly from pressure exerted upon the developing tooth bud from trauma (15). Lastly, a multifactorial etiology involving both genetic and environmental factors has been suggested (5, 15, 17).

A review of the literature reveals an abundance of terms for this anomalous dental structure, the most frequently encountered being: odontome, odontoma (odontome) of the axial core type, evaginatus odontoma (evaginated odontome), occlusal enamel pearl, occlusal tubercle, tuberculum anomalous, accessory cusp, supernumerary cusp, interstitial cusp, tuberculated cusp, tuberculated premolar, Leong's premolar, and talon cusp (specifically for anterior teeth) (5–22). While one article attempts to distinguish talon cusp from DE (23), most authors agree both are the result of an exacerbation of the same phenomena during the morphodifferentiation stage of tooth development (1, 5, 20). Talon cusp originated as a descriptive term for DE when observed on the lingual surface of anterior teeth because of a resemblance to an eagle's talon (5, 21). Currently, DE is the preferred terminology utilized to describe this developmental abnormality, first recommended by Oehlers in 1967 (19).

Pathophysiology

DE is thought to develop from an abnormal proliferation and folding of a portion of the inner enamel epithelium and subjacent ectomesenchymal cells of the dental papilla into the stellate reticulum of the enamel organ during the bell stage of tooth

formation (24–26). The resultant formation is defined as a tubercle, or supplemental solid elevation on some portion of the crown surface.

To establish a proposed embryologic etiology for this unusual entity, it is important to review the most recent work regarding tooth cytodifferentiation and morphogenesis during tooth development. Current embryologic evidence indicates that tooth morphogenesis is characterized by transient signaling centers in the epithelium, consisting of epithelial cell clusters that correspond to the initiation of individual cusps (27, 28). These signaling centers of nonproliferative transitory epithelial cells, the primary and secondary enamel knots, serve a regulatory function and are surrounded by strongly proliferative epithelium and underlying mesenchyme (29).

The primary enamel knot appears at the late bud stage, grows in size until the cap stage of tooth development is reached, and is responsible for the induction of the dental papilla (30). The primary enamel knot regulates the advancing cuspal morphogenesis of the crown through expression of up to 20 molecules, such as fibroblast growth factors (FGF-4 and 9), transforming growth factor β (TGF-β) and bone morphogenetic proteins (BMP-2, 4, and 7) (31). It has been suggested that mesenchymal BMP-4 induces expression of p21, a cyclin-dependent kinase inhibitor associated with terminal differentiation and possibly linked to the programmed disappearance of the primary knot cells (32). One theory is that these cells are induced to undergo a process of apoptosis, and by the early bell stage are no longer visible (33). The accumulation of molecules expressed by the primary enamel knot is thought to induce the initiation of the secondary enamel knots at the sites of epithelial foldings that mark cusp formation during the early bell stage of tooth development (32). The phenomenon of embryonic induction within and between different cell types is considered to be an important factor in the orderly formation of various parts of the tooth.

Activators from the primary enamel knot regulate the expression of the secondary enamel knots. The resultant cusp morphogenesis and positions appear to be determined sequentially, and cusps that form late in development, after the main cusps, are typically small (32). The secondary enamel knots disperse after formation of the cusp tips, indicating the termination of crown morphogenesis. Furthermore, the actual number of cusps realized in each tooth is also determined by the initiation of root formation. Thus, specific signaling molecules diffusing from the mesenchymal cells may act as inhibitors for the cusp tips while simultaneous production of other molecular signals may induce differentiation of Hertwig's epithelial root sheath at the cervical loop.

DE can arise on any tooth but is most commonly associated with premolars (19, 34). There is typically a bilateral, symmetric distribution, with a slight sexual predilection for females (6). Studies report various prevalence rates for a particular dental arch, and for percentages in specific teeth and populations reviewed. DE occurs in both deciduous and permanent teeth, but more frequently in the permanent dentition (1, 18, 22, 35). When the developmental anomaly appears in the anterior dentition, the tubercle most often forms on the lingual surface and is referred to as a talon cusp (36). When associated with the posterior dentition, the tubercle is variously located on the occlusal surface, primarily from the central groove followed next in frequency by developing on the inclined plane of the buccal cusp.

Morphology

The DE tubercles of posterior teeth average 2.0 mm in width (37) and up to 3.5 mm in length (9), and up to 3.5 mm in width and 6.0 mm in length for anterior teeth (17). Other than the cusp-like variable sized and shaped tubercle of teeth with DE, the remaining portion of the crown has a normal anatomy (6). This is an additional distinguishing characteristic from the accessory cusp of Carabelli, that when present,

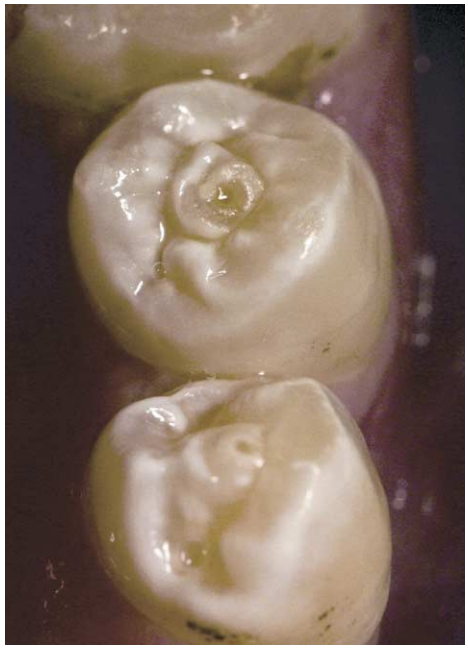

Figure 1. Teeth #20 and #21. Clinical pretreatment photograph (September 9, 2003).

the associated teeth are often larger than normal mesiodistally (1). However, abnormal root patterns are very often linked with DE involved teeth (9, 13, 18). Schulge (1987) distinguishes the following five types of DE for posterior teeth by the location of the tubercle (5).

1. A cone-like enlargement of the lingual cusp.
2. A tubercle on the inclined plane of the lingual cusp.
3. A cone-like enlargement of the buccal cusp.
4. A tubercle on the inclined plane of the buccal cusp.
5. A tubercle arising from the occlusal surface obliterating the central groove (Figs. 1 and 5).

Accordingly, Lau further classified each type of tubercle on the basis of four anatomical shapes of smooth, grooved, terraced (Figs. 1 and 5), and ridged (26).

Finally, Oehlers identified the evagination according to the pulp contents within the tubercle by examining the histological appearance of the pulp using decalcified serial sections of extracted teeth with DE (19). These categories are listed as follows along with their percentage of occurrence:

1. Wide pulp horns (34%)
2. Narrow pulp horns (22%)
3. Constricted pulp horns (14%)
4. Isolated pulp horn remnants (20%)
5. No pulp horn (10%)

Clinical Issues

Because the DE tubercle may extend above the occlusal surface up to 3.5 mm (6.0 mm for anterior teeth), malocclusion with the opposing tooth upon the cusp-like elevation occurs as involved teeth erupt into the dental arches. The resultant occlusal traumatic force causes abnormal wear or fracture of the tubercle, and is the usual manner of pulp exposure for this anomaly. Caries has historically not been a factor for consideration regarding pulpal involvement for this entity. A pathology report of one case extracted because of cellulitis was submitted for histologic analysis (18). The pathology report noted a necrotic pulp and

periapical abscess. No etiology was noted, but trauma or "anachoretic pulpitis" were suggested. Via data collected by Oehlers, since the pulp extends into 70% of the tubercles, an exposure of the pulp horn will likely occur following fracture, or when the layer of dentin is worn through, as a result of the concomitant malocclusion. Subsequent pulpal inflammation or infection will most likely ensue. It is important for the clinician to be able to recognize and treat the entity soon after affected teeth have erupted into the oral cavity to avoid pathological conditions. The authors of this current paper suggest the following six categories to determine treatment of teeth with DE.

Type I: Normal pulp, mature apex (Fig. 2, tooth #21 and Fig. 6, tooth #28)
Type II: Normal pulp, immature apex
Type III: Inflamed pulp, mature apex (Fig. 6, tooth #29)
Type IV: Inflamed pulp, immature apex
Type V: Necrotic pulp, mature apex
Type VI: Necrotic pulp, immature apex (Fig. 2, tooth #20)

Treatment of DE

Teeth presenting with a normal pulp and a mature apex (type I) should have the opposing occluding surface reduced to eliminate traumatic occlusion with the tubercle, followed by an application of topical fluoride to increase the enamel's hydroxyapatite resistance to acid breakdown. Then, incremental layering of an acid-etched flowable light-cured resin (AEFLCR) is applied to the tubercle and surrounding surface (Fig. 2, tooth #21 and Fig. 6, tooth #28). Reevaluation at 6-month intervals is advised to check the occlusion and perform any necessary adjustments, and assess the need for additional resin. Yearly radiographic reevaluation is suggested to assess the progress of pulp recession. When pulpal recession is considered adequate, the tubercle should be reduced to the level of the normal occlusal plane of the tooth. Any exposed dentin should be protected with an application of acid-etched microhybrid light-cured resin (Table 1).

When a tooth has a normal pulp and an immature apex (type II), treatment of the tooth should proceed as described in type I except that the reevaluation intervals should be every 3 to 4 months. It is necessary to more frequently monitor the occlusion of the erupting teeth, as well as ensure that root development is progressing normally. This should continue until the tooth has a mature apex.

If trauma to the tubercle results in a pulp exposure of a tooth with a mature apex, bacterial invasion will result in an inflamed pulp (type III) and usually develop symptoms of irreversible pulpitis (Fig. 6, tooth #29). Once diagnostically confirmed, conventional root canal therapy should be performed followed by placement of an appropriate final restoration (Fig. 7, tooth #29).

The complexity of treatment is increased when pulp inflammation develops because of contamination from the oral cavity when the tooth has an immature apex (type IV). In these cases, a shallow pulpotomy using a layer of mineral trioxide aggregate (MTA) (ProRoot, Dentsply Tulsa Dental, Tulsa, OK) applied to the exposed pulpal surface should be performed (modified Cvek technique) (38).

Cvek originally proposed the use of calcium hydroxide ($CaOH_2$) to be applied directly to the pulpal surface to create an environment that stimulates the formation of a dentin bridge (38). Studies have demonstrated that the healing is more predictable when there is no bacterial contamination of the pulp (39). Some investigators have asserted that $CaOH_2$ starts to soften over time resulting in leakage through the original seal (40). Because of the caustic actions (pH approximately 11.0 to 12.5) of $CaOH_2$, a localized area of coagulation necrosis occurs in the tissues immediately subjacent to the medicament (41). As a result, pulpal healing and hard tissue formation are delayed; and a potential for

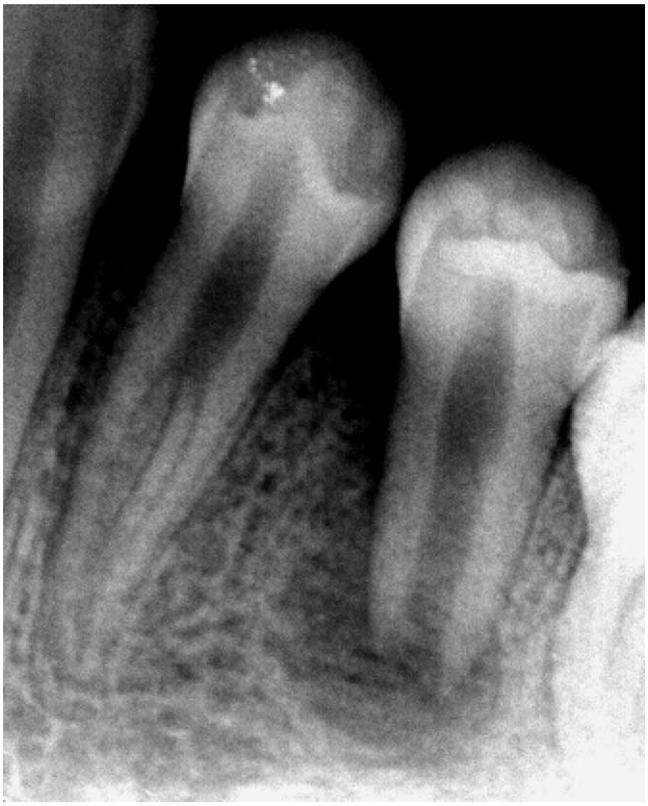

Figure 2. Teeth #20 and #21. Pretreatment radiograph (September 9, 2003).

patient symptoms exists. Adjacent to the zone of necrosis of up to 0.7 mm, cells in the pulp differentiate into odontoblasts that elaborate the matrix for the dentin bridge (42). The resultant dentin bridge formed is most often porous (43), and subsequent bacterial leakage through the porosities may result in pulpal inflammation and necrosis (44).

MTA has been shown to induce hard-tissue formation more predictably than $CaOH_2$ in shallow pulpotomy procedures (45), even though it also is highly alkaline when initially mixed with water (pH 12.0 to 13.0 per manufacturer's material safety data sheet). There is less pulpal inflammation (46), and the lack of any localized tissue necrosis following application of MTA to pulpal tissue may be a result of the more rapid set of the hydrophilic material, as compared to $CaOH_2$ that maintains a local state of alkalinity for a longer period.

MTA has excellent sealing properties, actively promotes hard tissue formation, is biocompatible, and upon setting has higher mechanical strength and better adhesion to dentin and restorative materials compared to $CaOH_2$ (46). Histologic examination of the dentin bridge formed following MTA application to the pulp reveals the bridge to form immediately adjacent to the MTA. The MTA induced dentin bridge begins to form sooner, becomes thicker and has less porosity than the hard tissue $CaOH_2$ induces the pulp to form (46).

When performing a shallow pulpotomy procedure, the superficial layers of the pulp are gently removed to a depth of 2.0 mm below the level of the exposure (47). Placement of MTA, as with $CaOH_2$, onto the remaining pulp tissue results in retaining vitality and function of the pulp-dentin complex, allowing odontoblasts to complete root development for the tooth (i.e., the process of apexogenesis leading to normal thickness of dentinal walls, and increased root length along with apical closure). An etch technique and other medicaments have also been proposed to be placed onto the pulp (48); but any agent may produce a permanent tissue necrosis, canal obliteration or pulpal resorption

TABLE 1. Treatment Regimen for Dens Evaginatus

Prophylaxis Tubercle Intact or Without Enamel		Intervention Tubercle with Pulp Exposure			
Normal Pulp		Inflamed Pulp		Necrotic Pulp	
Type I Mature Apex	Type II Immature Apex	Type III Mature Apex	Type IV Immature Apex	Type V Mature Apex	Type VI Immature Apex
Reduce opposing occluding tooth	Same as Type I except:	Conventional root canal therapy	Shallow MTA pulpotomy	Conventional root canal therapy	MTA root-end barrier
Apply acid-etched flowable light-cured resin to tubercle	Reevaluation every 3–4 months until development of mature apex	Restoration	Glass ionomer layer	Restoration	Glass ionomer layer
Yearly reevaluation to assess occlusion, resin, pulp and periapex			Acid-etched light-cured resin		Acid-etched light-cured resin
When reevaluation demonstrates adequate pulp recession, remove tubercle and apply resin					

(44). The key to pulpal healing is providing a seal that will prevent future bacterial microleakage (40).

Following set of the MTA, a glass ionomer layer (e.g. Fuji Triage or Fuji II LC [GC Corporation, Tokyo, Japan] or Ketac-Silver [3M ESPE AG, Seefeld, Germany]) should be placed directly onto the coronal surface of the MTA creating a protective barrier from potential breakdown by acid etching agents (W. de Rijk, 2004, personal communication). Per communication with the GC America Corporation, the pH of acid etching agents is low enough to etch the surface but not cause any breakdown of a glass ionomer matrix. Most acid etch products contain phosphoric acid in concentrations ranging from 10% to 40%. After the glass ionomer has set, an AEFLCR restoration should be placed into the remaining portion of the canal to a depth of a least 4.0 mm to minimize coronal leakage while obtaining a seal of the access (41, 45). With periodic reevaluation that could take up to 3 yr, there should be no indication of pulpal or periapical symptoms. If radiographic evidence demonstrates mature root development with apical closure, and without pulpal or periapical pathology, then an appropriate final restoration is indicated.

Once the pulp of a tooth with a mature root becomes necrotic (type V), with or without a periapical component, conventional root canal therapy followed by a final restoration is indicated.

If a tooth with a blunderbuss root apex becomes necrotic (type VI), with or without a periapical component, endodontic therapy is required; and the creation of a root-end barrier should be considered (Fig. 2, tooth #20). During mechanical instrumentation of the large canal, it is suggested to use a piezoelectric ultrasonic device to enhance the effectiveness of the intracanal irrigating solutions used to minimize bacterial levels (e.g. 2.5% NaOCl, 2% chlorhexidine, 0.9% sterile saline, ozonated water), resulting in better dentinal tubule penetration of the antimicrobial agents (49). The substantivity of chlorhexidine gluconate results in an extended period of antimicrobial activity for up to 72 hr (50). After drying the canal, a final rinse with 2% chlorhexidine combined with subsequent placement of a $CaOH_2$ paste as an interappointment medicament is advised for an enhanced spectrum of antimicrobial activity (51). Recently, intracanal medicaments containing antibiotic have had resurgence. MTAD (BioPure, Dentsply Tulsa Dental), containing the tetracycline isomer doxycycline, has been introduced into the market as a disinfectant irrigating solution (52, 53). One published case indicated the use of a triantibiotic paste (ciprofloxacin, metronidazole, and minocycline per Hoshino et al.) as an intracanal disinfectant (41). Ultimately, longitudinal in vivo studies regarding intracanal antibiotic use are necessary to provide data to demonstrate any enhanced or undesirable outcomes.

The canal is then sealed with a cotton pellet and placement of a hard setting, noneugenol containing, temporary restoration resistant to leakage. The MSDS inserts of eugenol-containing products (IRM, Dentsply Caulk, Tulsa, OK) mention interference with subsequent placement of resin restorations. Current data also support that eugenol-containing materials reduce the effectiveness of bonding an AEFLCR to dentin, resulting in a decreased resistance to fracture (54). When it has been assessed the tooth is free of infection and symptoms, a root-end seal can be effected.

It has been demonstrated that MTA used as an apical barrier for apexification induces hard tissue formation (55). The most predictable technique for obtaining the desired results of a densely obturated root-end seal is by ultrasonically compacting MTA into the apical portion of the open canal (56, 57). Manufacturer's directions for MTA indicate the need to create a 3 to 5 mm barrier for an apexification root-end seal. Recent studies have demonstrated that a 5.0 mm barrier is best to resist displacement as well as provide a superior apical barrier to bacterial leakage (58, 59). One study demonstrated that gray MTA was superior

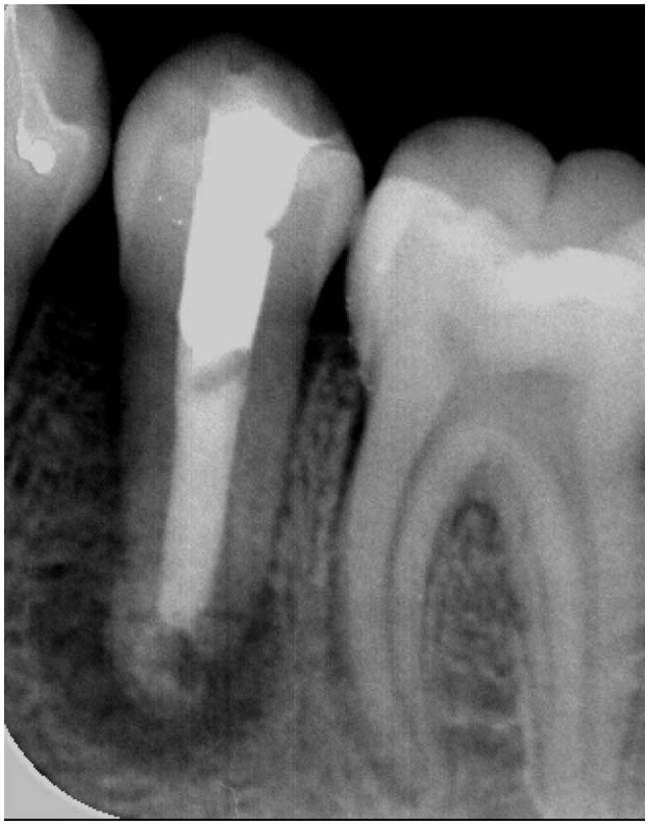

Figure 3. Tooth #20. Immediate posttreatment radiograph (November 17, 2003).

to white MTA for use as an apical plug (58). Some of these same studies suggest a one-step placement of the MTA root-end barrier (57, 59).

Apexification traditionally has involved placing $CaOH_2$ paste into the canal of a necrotic immature permanent tooth to induce cells of the periapex to create a calcific barrier (60, 61). However, $CaOH_2$ has some considerable drawbacks, and several studies have advocated placement of various artificial barriers as alternative medicaments (62–64). One experimental study demonstrated that the fracture strength of immature teeth filled with $CaOH_2$ was 50% less in 1 year, most probably a result of the weakening of the dentin structure by the medicament (65).

A study of trauma cases at a dental teaching hospital assessing teeth treated by $CaOH_2$ apexification reported the technique required from 3 to 17 appointments (66). Frequent replacement of the canal medicament was required; and treatment was lengthy, taking from 6 months to 2 years to obtain a barrier before obturation. Barriers sometimes were not induced; and when they were, the calcific tissue formed was often irregular or had inconsistent porosity, and was frequently unpredictably located within the canal (66). As a consequence, one author has concluded that long-term $CaOH_2$ therapy is no longer the treatment of choice for apexification because of the variability of its treatment outcomes and adverse effects on dentin (67).

Until recently, following the formation of a root-end barrier, obturation of the canal was performed with sealer and gutta-percha. This technique has been shown to significantly decrease the fracture resistance of the immature permanent tooth (56). This is an extremely important consideration, for in a retrospective longitudinal study, 32% of completed apexification cases ended up with root fracture because gutta-percha obturation does not enhance the strength of the thin and fragile dentin walls of the root (66). Others have indicated up to a 50% reduction in strength, with increased susceptibility to fracture in the cervical region of these teeth (68, 69). Because of the significant problematic issues of $CaOH_2$ apexification with later sealer and gutta-percha obturation, some suggest not only to supplant $CaOH_2$ with MTA for these cases, but also to place an AEFLCR into the canal as an alternative to conventional obturation treatment. This approach has been shown to significantly increase the fracture resistance of the tooth (62).

Following the compaction and set of the MTA barrier, a layer of glass ionomer is placed. After the glass ionomer set, a microhybrid AEFLCR should be inserted to fill the remaining canal and seal the access, thus resisting coronal leakage (Fig. 5, tooth #20). This newer format has the possibility of being completed at the second treatment session.

Parenthetically, because of the uncertain outcome for type VI involved teeth, extraction may be one treatment choice for patients unable to comply with a possible multiappointment regimen; or for those young patients possessing crowded dental arches, and when the loss of the type VI tooth can be included in an orthodontic treatment plan (70). This alternative is offered as only one consideration within a comprehensive treatment plan. In the not too distant past, for immature affected permanent teeth with either inflamed or necrotic pulps, extraction was the only treatment option (8, 19, 70, 71). The difficulty for treating these involved teeth is more recently exampled by a case reported in 2002 (18). A 9-year-old female patient presented with tooth #13 for six emergency visits over a 5-week period. The tooth was finally removed after the patient developed a left facial cellulitis.

Prophylaxis

Prevention of pulpal involvement in cases of DE is preferred over more invasive techniques. Because pulp exposure of the tubercle resulting from occlusal forces occurs soon after eruption, prophylactic intervention will prevent the need for treatment of teeth with immature apices and thin, weak roots. Even when pulp exposure is avoided past the period of complete root formation, the tubercles are still susceptible to fracture. Past treatment modalities suggested have had inconsistent results. Spot grinding of the tubercles to stimulate reparative dentin formation has not proven to be predictable (18). One article reported the use of an unfilled resin sealant over the tubercle to provide pulpal protection, but this material did not resist occlusal wear (72). Others have performed indirect and direct pulp capping in an attempt to accelerate the rate of reparative dentin formation, but results have been irregular, sometimes resulting in canal obliteration (34, 66, 73, 74).

Recently, an approach utilizing a modified Cvek technique pulpotomy with MTA was described as being prophylactic (45). Invading a normal pulp to avoid the potential development of pathology is not prophylactic, whether it is direct pulp capping or a shallow pulpotomy procedure. Following this logic would be to suggest that all teeth should have prophylactic endodontic intervention to avoid the potential development of necrosis from caries. Per Oehlers, 30% of tubercles do not possess a pulp horn; and more recent literature presents histological evidence that supports his findings (75). The authors believe that a truly prophylactic treatment modality is one that prevents development of a pathological or compromised pulpal status without violation or invasion of the pulp. The protocol described above in the type I and II categories and one was developed as a prophylactic technique without damage to the pulp of affected teeth.

While not always the case (76, 77), usually there is some traumatic occlusal interference from the opposing tooth with the tubercle. When present, the opposing cusp should be slightly reduced by about 0.5 mm, leaving approximately 1.5 mm of enamel. This reduction will permit a protective resin buildup over the tubercle of about 0.25 mm, and still allow for occlusal clearance. The reduced cusp should have an appli-

cation of topical fluoride to enhance the caries resistance of the enamel hydroxyapatite.

One proposal for eliminating the malocclusion was to remove enamel from the tubercle until barely revealing the underlying dentin, and then treat the newly exposed dentin with 8% stannous fluoride to stimulate formation of reparative dentin (10, 77). Because the pulp horn within the tubercle may extend to the dentino-enamel junction, the potential for pulp exposure exists with this grinding technique (18). To eliminate the risk of exposing the pulp, an AEFLCR should be applied to the surface of the tubercle, coating the enamel and any dentin that has been exposed as a result of occlusal wear. Then, incremental layers of an AEFLCR are applied until a sufficient thickness is obtained that will resist abrasion and re-exposing the tubercle's surface, at least until the next scheduled reevaluation. The suggestion in 1983 (72) and reported use in 1996 (76) of acid-etched resins exists in the literature. Since 1996, microhybrid and microfill flowable light-cured resins have been developed and are currently available. These newer, smaller (0.04–0.8 μm) particle-filled resins are much more resistant to abrasion, and the flowable format facilitates ease of application. A confidence now exists that these newer materials will be able to resist the occlusal wear between reevaluation sessions if placed onto the tubercle. At the patient reevaluations, as necessary, incremental additions of the flowable resin can easily be made to the surfaces where abrasion is evident.

Pulpal recession is a normal consequence of the aging process from odontoblast deposition of secondary dentin. Additionally, the placement of an acid-etched material to the tubercular dentin can stimulate a more rapid recession of the pulp horn extension by the stimulation of tertiary dentin. It is anticipated that at a time when radiographic evidence indicates a sufficient amount of pulp recession subjacent to the tubercle, the protuberance can be gradually reduced to the occlusal surface of the affected tooth. The newly exposed dentin can be sealed with a microhybrid AEFLCR.

Case Study

The authors believe this to be a rare documented case of DE in a patient of Mestizo heritage. The earliest reported DE case in an individual with this familial heritage is by Priddy et al. in 1976 (37). However, early articles are devoid of the phraseology currently used to depict individuals of this heritage (14, 37, 78). In September 2003, a 13-year-old Mestizo female presented to the University of Tennessee College of Dentistry patient clinic. She presented with DE involving all four mandibular premolars. Bilateral symmetry and affected premolars are commonly associated with this anomaly, involving from two to eight teeth (1, 6, 18, 34). No other anomalies were observed or reported. During the initial diagnostic examination, radiographs, and digital photographs were taken of all involved teeth.

Tooth #29 had acute pulpal symptoms. A radiograph revealed a mature apex that appeared normal (Fig. 6), and sensitivity to percussion testing was within normal limits. The endodontic cold testing with Endo-Ice (Hygenic, Akron, OH) demonstrated an exaggerated and lingering response compared to other adjacent teeth, indicative of irreversible pulpitis and the necessity for endodontic therapy, a category type III case. Following administration of local anesthesia and placement of rubber dam, the case was accessed and instrumented with hand and rotary NiTi files to a size #120 and 0.04 taper at a working length of 16.0 mm concomitant with 2.6% NaOCl and 17% EDTA (RC-Prep, Premier, King of Prussia, PA), followed by a final flush with 0.9% sterile saline solution. The canal was dried with paper points and sealed with cotton and Triage. One week later the tooth was asymptomatic, and the case was completed with obturation of gutta-percha and a resin sealer (ThermaSeal Plus, Dentsply Tulsa Dental) via a cold lateral condensation technique. The access was sealed with an AEFLCR (Venus flow, Heraeus Kulzer, Armonk, NY) (Fig. 7).

Tooth #20 was asymptomatic. Radiographic examination revealed an immature apex with a large radiolucency (Fig. 2). Response to percussion was normal and pulp tests yielded no response when compared to other adjacent teeth, yielding a diagnosis of a necrotic pulp and chronic apical periodontitis, a category type VI case. Following administration of local anesthesia and placement of rubber dam, the tooth was instrumented with hand and rotary NiTi files to a size #140 and 0.04 taper at a working length

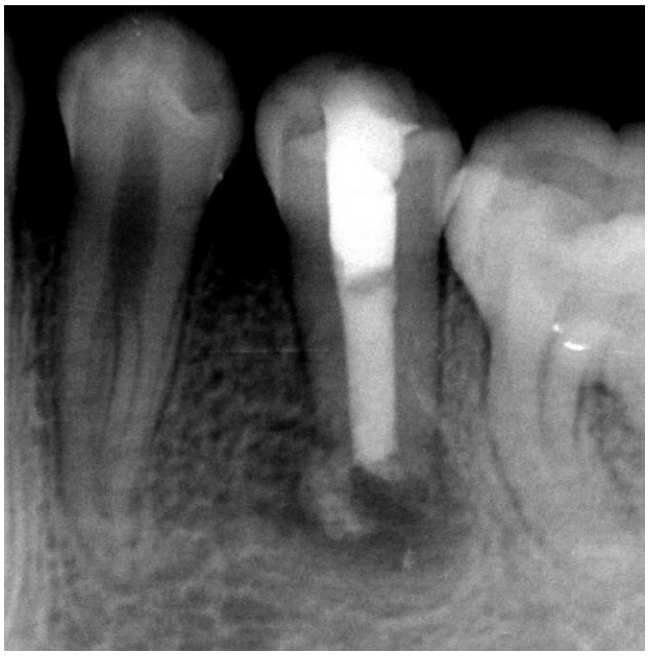

Figure 4. Teeth #20 and #21. Five month reevaluation radiograph (April 14, 2004).

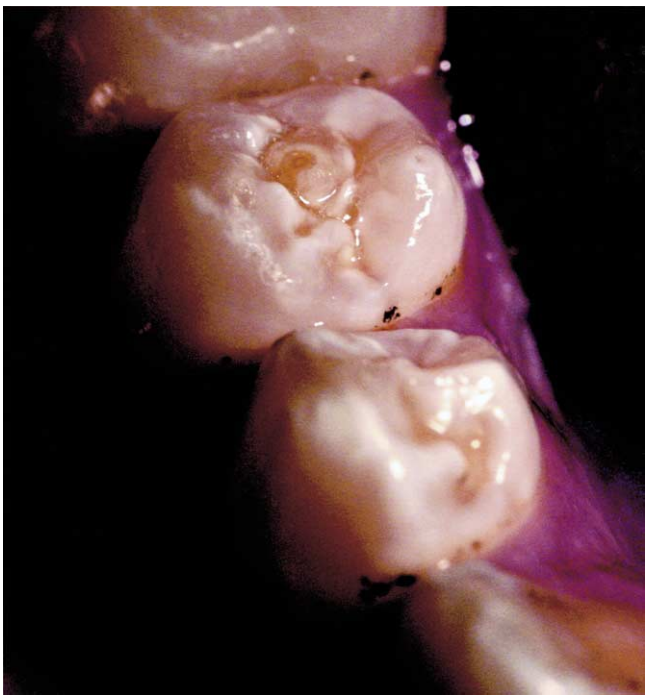

Figure 5. Teeth #28 and #29. Clinical pretreatment photograph (September 9, 2003).

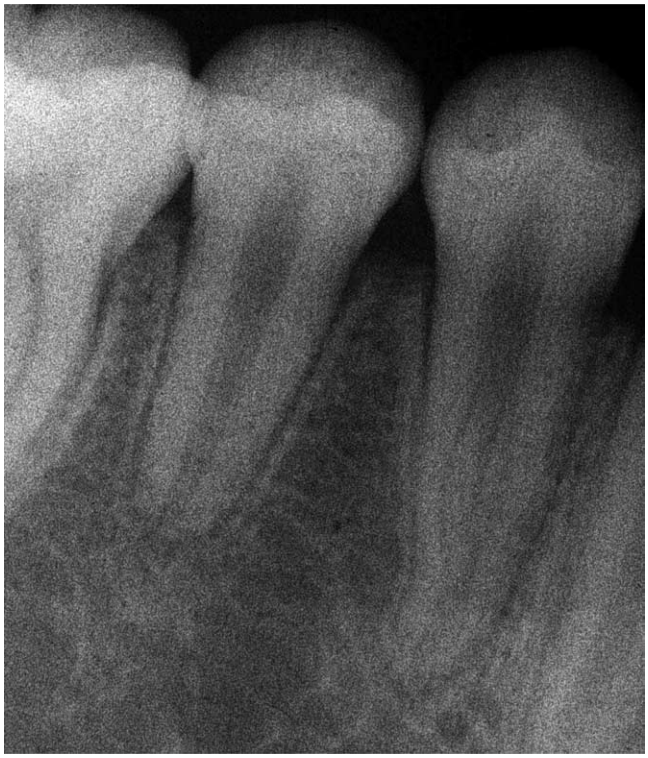

Figure 6. Teeth #28 and #29. Pretreatment radiograph (September 9, 2003).

of 16.0 mm. Because of the necrotic canal content, minimizing bacterial levels was aided by irrigation with 2.6% NaOCl and 17% EDTA during instrumentation, and a final flush with 0.9% sterile saline solution. After drying with paper points, the canal had a rinse with 2% chlorhexidine solution (Chlorhexi-Prep, Premier, Plymouth Meeting, PA) followed by placement of $CaOH_2$ paste (Pulpdent Paste, Pulpdent, Watertown, MA). The canal was sealed with cotton and Triage.

The patient returned 1 week later and presented with no symptoms. Removal of the intracanal paste from the canal was manifested with sterile saline irrigation and use of the master apical file to working length. After drying the canal with paper points, gray MTA was mixed and ultrasonically compacted into the apical portion of the canal to create a barrier of at least 5.0 mm. A moist cotton pellet was placed into the canal to cover the MTA and enhance the set of the hydrophilic material. Following a one hour period to allow the MTA to reasonably set, the cotton pellet was removed, and a layer of filled glass ionomer (Ketac-Silver) was applied to cover the surface of the MTA. Manufacturer's directions for MTA indicate a final set occurs after 4 hours, but at least one study determined the set to be a much faster 2 hours and 45 minutes (79). It has been suggested that acid might break down the MTA, so before placing the final coronal seal, Ketac-Silver was placed. This type of protective covering is also indicated in the manufacturer's directions for use. Two and a half minutes were allowed for the glass ionomer material to set (per manufacturer's directions). The remaining coronal portion of the canal was sealed with an AEFLCR (Fig. 3).

Both of the first premolars (teeth #21 and #28) were asymptomatic. Diagnostic pulpal and periapical testing of the teeth were within normal parameters, and radiographs demonstrated mature apices, a category type I for these teeth. However, radiographic images did reveal similar abnormal root formation for both teeth, with each exhibiting a main canal with trifurcation at mid-root into three slender roots (Figs. 2 and 6). If these teeth were someday to become pulpally involved, conventional root canal therapy would present a challenge.

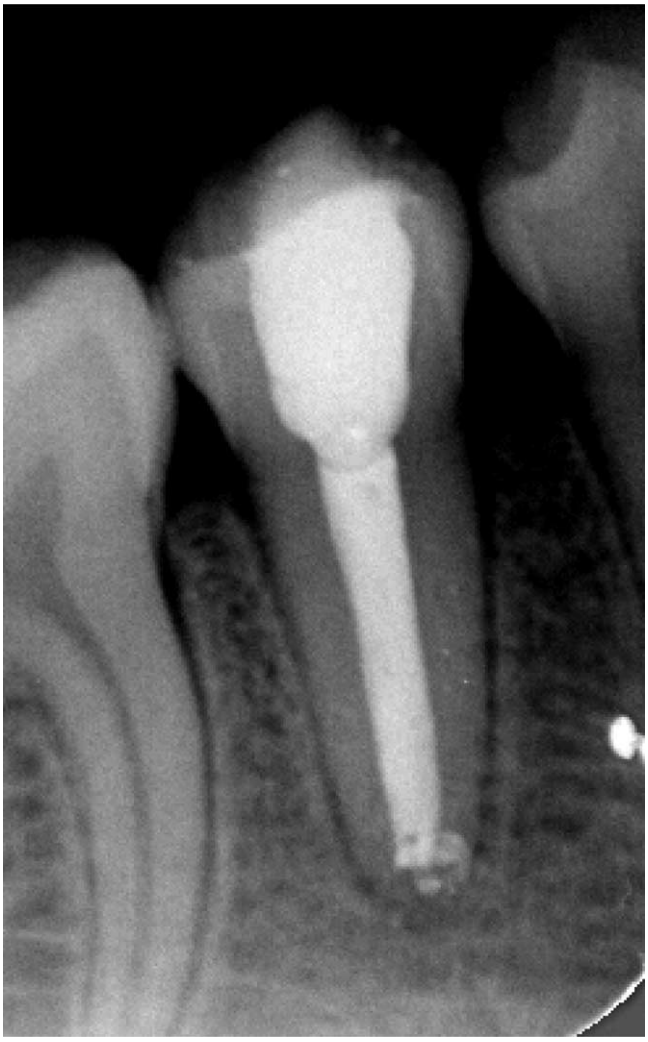

Figure 7. Tooth #29. Immediate posttreatment radiograph (October 3, 2003).

A prophylactic regimen for the mandibular first premolars involved a 0.5 mm reduction of the palatal cusp of the opposing maxillary first premolars. Sodium fluoride 2.0% gel (Neutra-Foam, Oral-B, Mississauga, Ontario, Canada) was placed in a tray, and then seated onto the maxillary teeth for 4 minutes (per manufacturer's instructions). Venus flow was then applied incrementally to the tubercle and adjacent surrounding area for teeth #21 and #28.

At the 6-month reevaluation, clinical examination and radiographs were taken for all involved teeth. Tooth #29 was asymptomatic and radiograph evaluation assessed the periapex as normal (Fig. 8). Tooth #20 also was reported as being asymptomatic. The radiographic image demonstrated an approximate 30% reduction in the size of the periapical radiolucency (Fig. 4). Teeth #21 and #28 were both asymptomatic, and pulpal testing for both teeth compared to other adjacent nonendodontically involved teeth. Reapplication of resin to the tubercles was not required. The occlusal adjustment of the opposing teeth did not seem to have affected the pulps of these teeth as they tested within normal limits.

Discussion

The treatment regimen for cases of type III through type VI can also be applied to traumatized teeth presenting with fractured crowns and pulp exposure. All of the considerations for evaluating the pulpal status

and extent of root maturity when diagnosing these traumatized teeth are similar to patients who present with fractured tubercles as a complication of DE.

America's largest minority, the Hispanic/Latino/mestizo people, constitutes 13.7% (40 million) of the US population. Since 1980, their growth rate has been five times greater than the general population. By 2025 they are expected to comprise 22% of the US total, over 80 million people, with 14 million of Mestizo descent. This rapid growth will result in an increase of the DE anomaly observed in US dental practices as Mestizos integrate into health care facilities throughout the country. Reports from other regions of the world have indicated similar concerns about an increased prevalence of this anomaly, but in these cases the influx in immigration involves Asians (73).

For type VI cases, any treatment suggested still has a result that must contend with a structurally deficient root system. With this limitation in mind, a technique has been reported that claims to have induced a clot within the canal that acted as a matrix for new tissue growth. The tissue was subsequently treated with MTA as one would when performing a shallow pulpotomy. The possibilities of transforming an immature infected permanent tooth requiring the creation of an apical barrier into an apexogenesis case is significant. However, only one such case was reviewed (41). It is impressive to observe the application of newly developed materials innovatively utilized to treat difficult clinical problems more easily and predictably. Additional favorable results from a longitudinal study are eagerly awaited.

The patterning cascade mode of tooth cusp development is not unlike the process of blood coagulation, where a progression of events in a particular sequence is dependent on several interacting factors. It is well known that genetic deficiencies for any of the required blood coagulation factors can variously alter the process, depending upon the factor involved. Likewise, gene mutations, interference with production of transcription factors, or inability to induce gene expression could cause induction of an uncharacteristic enamel knot late in the differential growth process, resulting in an abnormally located small cusp as observed with DE. The belated enamel knot may develop from excess accumulation of accelerator signaling molecules from the secondary enamel knots. The accelerator signal may persist because of faulty upregulation of certain molecules, thus delaying the necessary inhibitor feedback mechanism between cusp tip termination and induction of root formation. This could explain a few of the observed associations of DE cusp-like tubercles. Cusp tip termination indicates the end of enamel deposition, normally occurring during the developmental stage when apposition of dentin is induced within the pulp. As well, differentiation of odontoblasts producing dentin begins in the area of Hertwig's root sheath proliferation. If pulpal dentin formation is delayed this could explain why the late forming DE cusp-like tubercles have an uncharacteristic pulp extension at time of eruption not present in the normal cusp. Also, any delay in dentin formation could explain why DE is so commonly associated with abnormal root morphology.

With an eye to the future, the best treatment modalities available should be implemented as soon as valid research can support a successful long-term outcome. Keeping this in mind, innovative and better outcome techniques are only as far away as the imagination of the researcher. We will be encountering a population presenting with a new gene pool that will challenge the US health care delivery system. Still the most intriguing prospects reside within the realm of molecular biology. Gradually revealing the very essence of human genetic expression and diversity is leading to an understanding of how genes control our life processes. Some day soon stem cells will be implanted into the site of a missing tooth to create a perfect replacement.

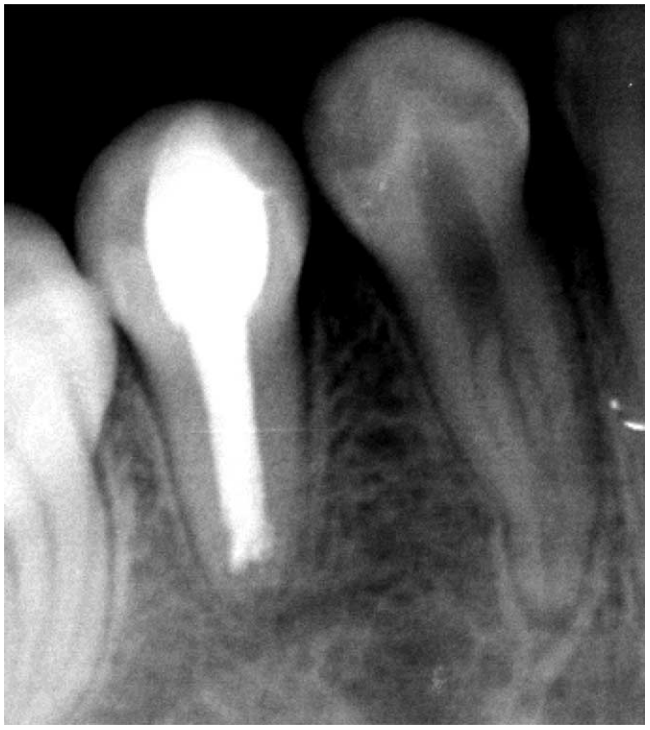

Figure 8. Teeth #28 and #29. Six month reevaluation radiograph (April 14, 2004).

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