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CASE REPORT

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Bilateral dens evaginatus and associated cystic pathology: An unusual case report

Haeseong Lee[1] | **Bach Le**[1,2] | **Parish Sedghizadeh**[1,3]

[1]Herman Ostrow School of Dentistry, University of Southern California, Los Angeles, California, USA

[2]Division of Oral and Maxillofacial Surgery, Herman Ostrow School of Dentistry, University of Southern California, Los Angeles, California, USA

[3]Division of Diagnostic Sciences, Herman Ostrow School of Dentistry, University of Southern California, Los Angeles, California, USA

Correspondence
Haeseong Lee, Herman Ostrow School of Dentistry, University of Southern California, Los Angeles, CA 90089, USA.
Email: haeseong@usc.edu

Key Clinical Message

Dens evaginatus (DE) presents a clinical conundrum due to its fragility and propensity to cause pulpal and periapical complications. Clinicians should heed the presence of DE during clinical examinations and avoid unnecessary intervention.

KEYWORDS

dens evaginatus, dental tubercle, periapical cyst, pulpal complications, tooth anomalies

1 | INTRODUCTION

Dens evaginatus (DE) describes a developmental dental anomaly that results in the projection of a tubercle with an extension of pulp tissue inside a dentinal core. Considered the antithesis of dens in dente, DE tubercles are also known as tuberculated cusp, accessory tubercle, occlusal tuberculated premolar, evaginatus odontoma and occlusal pearl.[1] The tuberculated, accessory cusp is most associated with mandibular premolars, albeit observed in incisors, canines, and molars of both arches, and forms from the abnormal folding and proliferation of inner enamel epithelium into the stellate reticulum of the enamel organ during the bell stage of dental morphological differentiation.[1–3] The result is a small accessory, protruding structure on the occlusal surface of posterior teeth and the lingual surface of anterior teeth.[4]

DE is primarily seen in people of Asian descent, including Filipinos, Chinese, Malaysians, and Indian populations, and has a marginal sexual predilection for females, with estimates of prevalence ranging from roughly 1% to 4%, depending on the specific population studied.[5] When associated with the posterior teeth, the location of the tubercle on the occlusal surface varies but is primarily seen along the central groove and subsequently on the sloped surface of the buccal cusp.[4] The DE tubercles of posterior teeth may range up to 3.5 and 2.5 mm in mesiodistal length and labiolingual width, respectively.[6,7] Additionally, DE occurs in both deciduous and permanent dentition, but is observed more frequently in permanent teeth.[2,8,9] Bilateral, symmetrical distribution of the premolars is common, involving anywhere from two to eight teeth.[9–11]

As expected, DE may cause occlusal interference as the tubercle comes into occlusion, resulting in it fracturing from the tooth. As the nodule contains a pulpal extension,

which varies in dimensional breadth, from being narrow, isolated, or constricted to being not present at all, pulpal complications are often associated with DE.[11,12] Pulp exposure can result from the natural fracture of the tubercle due to occlusal trauma or can be iatrogenic in nature, that is, "shaving off" the supplemental cusp with a dental instrument. The involved tooth may remain asymptomatic for an extended period, with the potential risk of developing periapical pathosis later.[11,12] Specifically, a periapical or radicular cyst, which is the most common inflammatory cyst of the jaw, may form because of an infection originating from pulpal exposure.[9] Depending on the stage of disease progression, treatment involves either extraction or endodontic therapy for the involved tooth.[4]

2 | CASE HISTORY/EXAMINATION

A 14-year-old Asian female with non-contributory medical history is referred to the Oral Surgery Clinic for evaluation and extraction of both mandibular second premolars. Clinical exam revealed no apparent evidence of caries or fracture on both teeth; however, there was a chronic fistula located on the buccal gingiva of both premolars (Figure 1). The patient had previously been evaluated by the Endodontics Clinic for bilateral, localized pain, and tenderness in the lower premolar region. Recurrent drainage and soft tissue swelling were also noted along with mild throbbing pain upon percussion and palpation. Mesial and distal probing depths were within normal range; however, mid-buccal probing depth showed moderate localized bone loss for both premolars. Cold test and electric pulp test were positive, indicating vital teeth. Radiographs revealed bilateral diffuse periapical radiolucencies around the second premolars and moderate loss of bone density of the buccal bone plate of the alveolar process (Figures 2 and 3). A pulpal diagnosis of symptomatic irreversible pulpitis and periapical diagnosis of symptomatic apical periodontitis were made.

3 | METHODS

Oral pathology was consulted for differential diagnosis prior to surgical extraction of the anomalous premolars, which was part of the patient's orthodontic treatment plan. Based on the patient's ethnicity, clinical presentation, and nature and site of the lesions, oral pathology suspected a history of DE and associated periapical radiolucencies with bone loss. After the extraction was performed, the gross specimen was submitted in 10% formalin solution for histologic processing (Figure 4). The periapex of both extracted teeth demonstrated cystic soft tissue that was tan brown, irregular in nature, measuring 1.2×0.8×0.7 cm and 0.9×0.7×0.5 cm in respectively (Figure 4). Sections of the sample revealed segments of soft tissue representing a cystic structure. The cyst was lined by focally hyperplastic and acutely inflamed stratified squamous epithelium, with the cyst wall composed of interwoven bundles of collagen and found to be well vascularized (Figure 5). Acute and chronic inflammation was observed throughout the specimen along with the presence of basophilic bacterial colonies. Small fragments of decalcified mineralized tissue were also seen. No evidence of dysplasia was observed in the sections studied. Additionally, the two associated teeth, which were submitted with each of the lesions described above, grossly showed evidence of a single central DE lesion.

After informing the patient's responsible person in charge and inquiring further regarding the patient's dental history, the responsible person in charge presented an image of the premolars from over a year ago revealing bilateral evaginations along the central groove of the mandibular second premolars, with a mesiodistal length of approximately 3 mm and labiolingual width of 2 mm at the base of the cusp (Figure 6). The responsible person in

FIGURE 1 Pre-extraction clinical images of mandibular second premolars showing labial fistulas.

FIGURE 2 Panoramic radiograph demonstrating bilateral localized periapical radiolucencies at the apex and extending distally at the mandibular second premolars, indicating moderate loss of bone density.

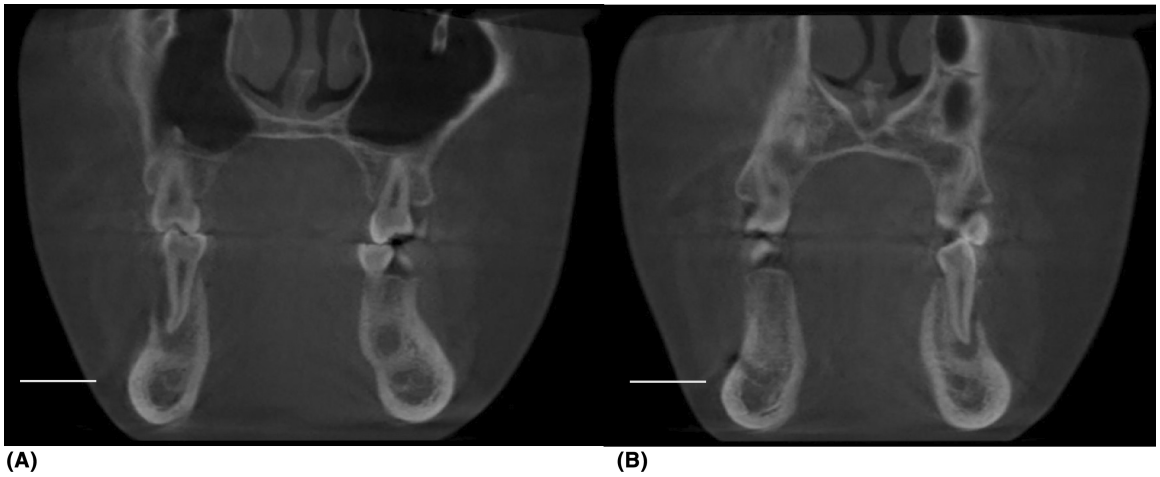

FIGURE 3 Bilateral buccal plate bone loss evident in coronal view of cone beam CT scan of (A) right and (B) left second mandibular premolars.

FIGURE 4 Gross specimen that was submitted in 10% formalin solution for histologic processing. The periapex of both extracted teeth demonstrated granulomatous-like soft tissue that was tan brown, irregular in nature and measuring 1.2 × 0.8 × 0.7 cm and 0.9 × 0.7 × 0.5 cm grossly.

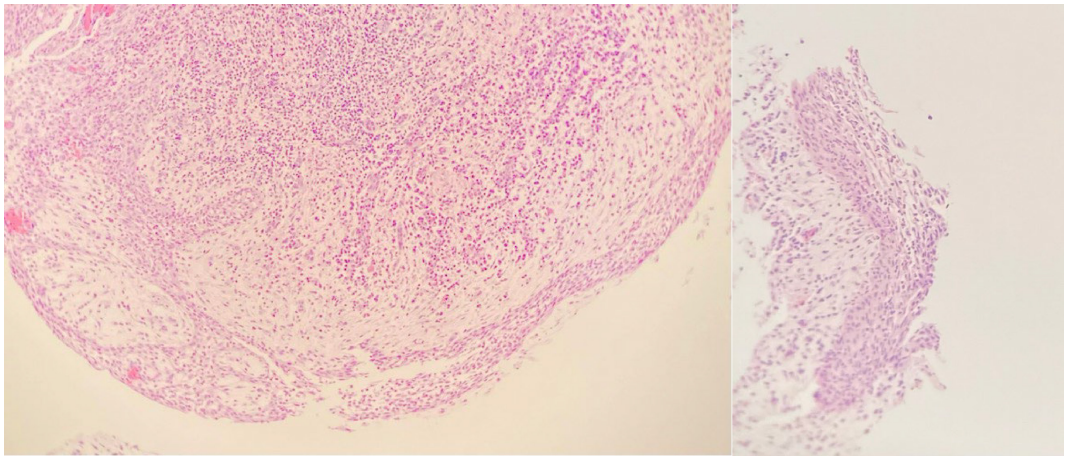

FIGURE 5 Sections of the sample revealing segments of soft tissue representing a cystic structure. Cyst is lined by focally hyperplastic and acutely inflamed stratified squamous epithelium, with the cyst wall composed of interwoven bundles of collagen and chronically inflamed.

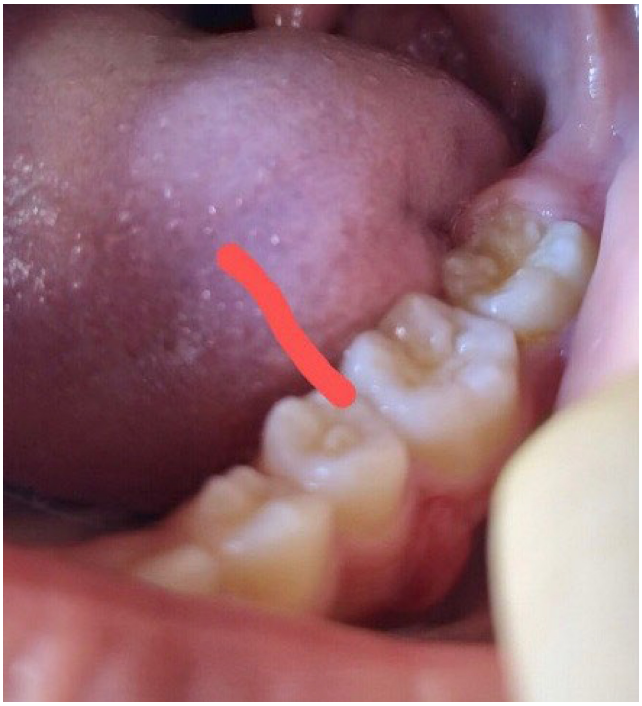

FIGURE 6 Tubercle along the central groove of the left mandibular second premolar, with a mesiodistal length of approximately 3 mm and labiolingual width of 2 mm at the base of the cusp. Image and consent courtesy of patient's responsible person in charge.

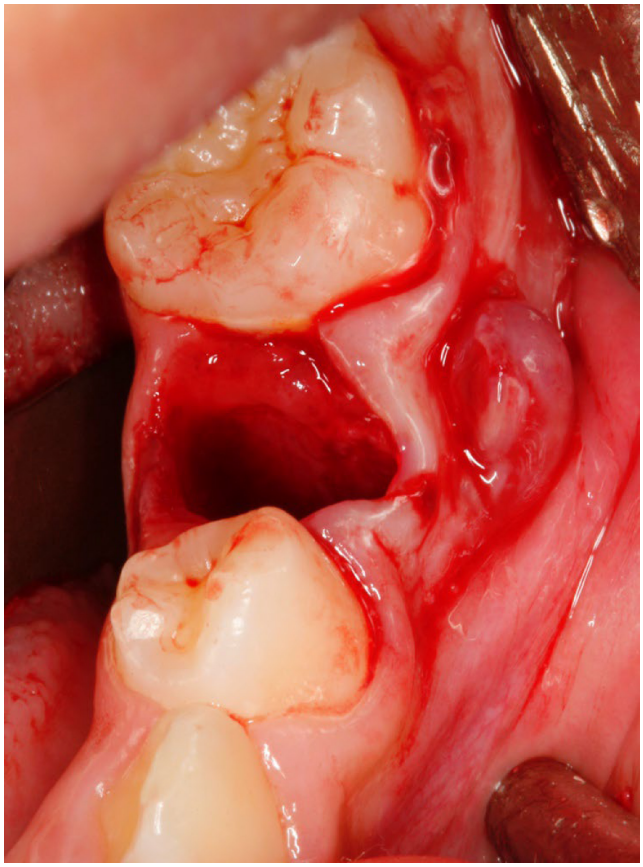

FIGURE 7 Extracted site of lower left mandibular second premolar.

charge indicated that the tubercles had been "shaven off" by their dentist the previous year.

4 | RESULTS

As the patient was anticipating orthodontic treatment, both anomalous mandibular second premolars were extracted. A mucoperiosteal elevator was used to reflect the gingival tissues surrounding the teeth. Extraction forceps were used to achieve luxation of both premolars, and the teeth were extracted without complications (Figure 7). Gauze was packed at both extraction sites following ample irrigation with saline. Postoperative instructions were provided along with a post-extraction antibiotic regimen of amoxicillin 500 mg capsule T.I.D for 7 days. Postoperative re-evaluation 4 weeks later revealed normal healing at both extraction sites. The fistulas located on the buccal gingiva of both premolars had subsided.

5 | DISCUSSION

DE is of clinical importance due to its propensity to provoke complications, as the tubercle may be easily fractured or abraded and result in pulp exposure and subsequent inflammatory infection. The pulp horn within the tubercle may extend to the dentin-enamel junction (DEJ), elevating the potential risk of pulp exposure from deliberately reducing the accessory cusp to remedy occlusal interference.[4] The risk of infection is multiplied, as the presence of the tubercle makes it especially challenging to clean resultant creviced areas on the tooth, rendering the tooth susceptible to caries. As the accessory cusp is usually fractured from the remaining tooth due to occlusal interference and concomitant trauma, observing external evidence of the anomaly clinically is rather challenging. Consequently, early detection and preventative management during routine dental examinations is paramount.[13]

With regards to treatment, the vitality of the pulp, presence of the DE tubercle on the tooth, along with the maturity of the root of the tooth may dictate the modality taken. Specifically, DE on a tooth with vital, normal pulp may warrant selective reduction of the opposing occluding tooth to prevent later fracture, while the application of acid-etched flowable light-cured resin may be indicated to seal the base of the tubercle area that has already been fractured from the tooth.[4] In instances of pulp exposure, resulting in an inflamed pulp, and an immature root, shallow mineral trioxide aggregate (MTA) pulpotomy may

be performed, whereas a mature, closed root apex would warrant conventional root canal therapy. Additionally, conventional root canal therapy and MTA root-end barrier may be indicated for pulp exposures resulting in necrotic pulp with a mature and immature apex, respectively.[4]

The root development of mandibular second premolars is generally completed at 14 years of age. As seen in the radiograph in Figure 2, the apex appears to be closed and the root seems to be mature in the presenting case. However, due to significant loss in bone density, grade II tooth mobility, and the prospect of future orthodontic treatment that would necessitate extraction of lower teeth due to crowding, the extraction of both mandibular second premolars were clinically indicated. In addition to the extraction already being included in an orthodontic treatment plan, extraction is preferred in cases where the DE tooth is a supernumerary tooth or mesiodens.[14] Extraction may also be necessary in cases of an extremely short root or open apex along with those that demonstrate funnel-shaped apical root resorption.[11] Furthermore, a recently published systematic review on the management of DE concluded that pulpal diagnosis should guide the management of the DE. Specifically, DE teeth with normal pulp or reversible pulpitis should be prophylactically treated by either the prep-and-fill technique or reinforcement.[15] DE teeth with irreversible pulpitis should be treated by either vital pulp therapy to induce apexogenesis, in the case of an immature root, or pulpectomy depending on the extent of the pulpal inflammation.[15] If the pulp of the associated tooth is necrotic, it should be treated by root canal therapy, regenerative endodontic, or apexification, contingent upon the level of root maturation.[15]

Another important aspect of the case is the presence of vital teeth associated with a histologically confirmed inflammatory apical cyst. Although periapical cysts are often considered secondary to pulpal infection, inflammation, and necrosis, both mandibular second premolars tested were symptomatic and tested vital. As the eruption of the second mandibular premolars generally occurs between 11 and 12 years of age and completes root development up to 3 years following their eruption, it is possible that ongoing root development and active re-vascularization in a young, healthy patient complicated an expected finding of necrotic pulp in both premolars.[15] The presence of vital teeth may also indicate pulp tissue that are infected incompletely or partially necrotic.[16] As a result, the diagnosis and treatment of DE-associated periapical lesions such as a periapical cyst may be overlooked or delayed, as there appears to be no apparent etiology for the radicular cyst such as a necrotic pulp due to caries.

Common diagnoses such as phoenix abscess, that is, acute exacerbation of a chronic peri-radicular disease, are also worth considering. An untreated necrotic pulp may have resulted in the superimposition of an inflammatory reaction onto an existing asymptomatic apical periodontitis. Clinical findings such as tenderness to palpation, grade II mobility, and the presence of fistulas present on the mucosa are consistent with this line of thought.[17] However, vitality testing of the pulp, using the cold test as well as electric pulp test, was positive. In the case of a phoenix abscess, we would expect a non-vital, necrosed tooth that would exacerbate an existing low-grade periapical inflammatory condition.[17] Most importantly, the histology shows focally hyperplastic and acutely inflamed stratified squamous epithelium lining a sac, which is indicative of an inflammatory cyst. Clinically, a localized collection of purulent material upon extraction was absent. However, a dense accumulation of cells of chronic inflammation are noted within the cyst wall composed of interwoven bundles of collagen, as shown in Figure 5.

6 | CONCLUSION

DE is of clinical significance to restorative dentists and surgeons alike, as the nodule often creates occlusal interference and periapical pathosis, making it a profound multidisciplinary challenge with regards to clinical diagnosis and treatment. The tubercle is easily fractured or abraded and yields the risk of pulp exposure and subsequent inflammatory infection. Hence, clinicians should heed the presence of DE during clinical examinations and avoid nonessential grinding or smoothing of the tubercle and consider prophylactic or therapeutic intervention only when clinically indicated. Consequently, clinicians may pay attention to the presence of DE during routine clinical examinations and consider a prophylactic or therapeutic intervention only when indicated. The diagnosis of DE should be considered in periapical radiolucent lesions when there is no finding of carious or pulpo-periodontal etiology.

AUTHOR CONTRIBUTIONS

Haeseong Lee: Formal analysis; investigation; writing – original draft; writing – review and editing. **Bach Le:** Formal analysis; investigation; methodology; validation; visualization; writing – review and editing. **Parish Sedghizadeh:** Conceptualization; data curation; formal analysis; investigation; project administration; resources; supervision; validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

No conflict of interests to disclose.

DATA AVAILABILITY STATEMENT

Research data are not shared.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Haeseong Lee https://orcid.org/0000-0002-1455-9521

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