



[DOI]10.12016/j.issn.2096-1456.2020.09.008

· 防治实践 ·

## 乳牙根尖周炎致恒牙胚坏死病例报告及文献复习

卢洁<sup>1,2</sup>, 王迎菊<sup>2</sup>, 张利娟<sup>2</sup>, 李帆<sup>2</sup>, 李姗姗<sup>2</sup>, 谭凯璇<sup>1,2</sup>, 张颖<sup>2</sup>, 杨芳<sup>2</sup>

1. 大连医科大学口腔医学院,辽宁 大连(116044); 2. 青岛大学附属青岛市市立医院口腔医学中心,山东 青岛(266011)

**【摘要】目的** 探讨乳牙严重根尖周炎致牙槽骨吸收引起继承恒牙胚坏死的病因、诊断及治疗原则,为临床诊疗提供参考。**方法** 对一例罕见的恒牙胚坏死病例的临床资料及相关文献进行回顾性分析。**结果** 本例患者为5岁女孩,查体及X线片检查发现75慢性根尖周炎。X线片显示牙周骨质广泛性破坏,35牙胚周围硬骨板包绕不完整,较45发育滞后。初期诊断35牙胚因炎症停止发育,后期拔除75时发现35牙胚已坏死,术后病理检查报告为:35牙胚多处死骨形成。文献复习结果显示,根尖周病变程度及牙胚的发育阶段对继承恒牙胚有较大影响,治疗上多采取根管治疗等保守方法。乳牙根尖周炎致恒牙胚坏死在临床很罕见,应根据临床症状与影像学表现及早判断炎症浸润程度及恒牙胚受损程度,正确作出相应治疗计划。**结论** 临床对于恒牙胚病理状态的诊断还未形成客观明确的诊断和治疗标准,因此对于发育异常的恒牙胚通常采取消除病因,随访观察等手段。今后的研究方向应致力于预防及寻找更有效的诊断及治疗方法。

**【关键词】** 继承恒牙胚; 坏死; 乳牙; 根尖周炎; 牙槽骨吸收;  
病因; 诊断; 治疗



**【中图分类号】** R78 **【文献标志码】** A **【文章编号】** 2096-1456(2020)09-0590-04

开放科学(资源服务)标识码(OSID)

**【引用著录格式】** 卢洁,王迎菊,张利娟,等.乳牙根尖周炎致恒牙胚坏死病例报告及文献复习[J].口腔疾病防治,2020,28(9): 590-593.

**Permanent successor necrosis caused by periapical periodontitis of deciduous teeth: a case report and literature review** LU Jie<sup>1,2</sup>, WANG Yingju<sup>2</sup>, ZHANG Lijuan<sup>2</sup>, LI Fan<sup>2</sup>, LI Shanshan<sup>2</sup>, TAN Kaixuan<sup>1,2</sup>, ZHANG Ying<sup>2</sup>, YANG Fang<sup>2</sup>. 1. College of Stomatology, Dalian Medical University, Dalian 116044, China; 2. Center of Stomatology, Qingdao Municipal Hospital Affiliated to Qingdao University, Qingdao 266011, China

Corresponding author: YANG Fang, Email: fancy-yf@163.com, Tel: 86-15063903750

**【Abstract】 Objective** To investigate the etiology, diagnosis and treatment principles of inherited permanent tooth embryo necrosis caused by alveolar bone resorption due to severe periapical periodontitis of deciduous teeth, in order to provide a reference for clinical diagnosis and treatment. **Methods** The clinical data and related literature of a rare case of permanent tooth embryo necrosis were analyzed retrospectively. **Results** This case was a 5-year-old girl. Physical examination and X-ray examination revealed chronic periapical inflammation in 75. X-ray showed that the periodontal bone of tooth 75 was extensively destroyed; additionally, the permanent tooth germ of tooth 35 was incomplete and the development was delayed compared to that of tooth 45 because of severe periapical periodontitis in the primary teeth. The initial diagnosis was that-- the embryo of tooth 35 stopped developing due to inflammation and was necrotic after tooth 75 was extracted. The postoperative pathological examination report showed that most bone around the embryos of tooth 35 was sequestered. Through literature review and analysis, it was found that the degree of periapical lesions in the primary teeth and the developmental stage of the tooth embryo have a great impact on the formation of permanent tooth embryos. Conservative methods such as root canal therapy are usually adopted as treatment. Permanent tooth embryo necrosis caused by periapical periodontitis of deciduous teeth is rare in the clinic, so it is necessary to judge the degree of inflammatory infiltration and of tooth embryo damage as soon as possible according to the clinical symptoms and imaging manifestations; and to make a correct treatment plan. **Conclusion** There are no objective and clear diag-

**【收稿日期】** 2020-03-23; **【修回日期】** 2020-05-11

**【基金项目】** 国家自然科学基金面上科学基金项目(81670979)

**【作者简介】** 卢洁,医师,学士,Email:Nikky1224@163.com

**【通信作者】** 杨芳,副主任医师,博士,Email:fancy-yf@163.com, Tel: 86-15063903750



nostic and treatment criteria for the clinical diagnosis of the pathological state of permanent tooth embryo, thus, methods such as etiology elimination and follow-up observation are usually adopted for abnormal permanent tooth embryo development. Future research should focus on prevention and finding additional effective methods for diagnosis and treatment.

**[Key words]** permanent successor; necrosis; primary teeth; periapical periodontitis; alveolar bone resorption; etiology; diagnosis; therapy

**J Prev Treat Stomatol Dis, 2020, 28(9): 590-593.**

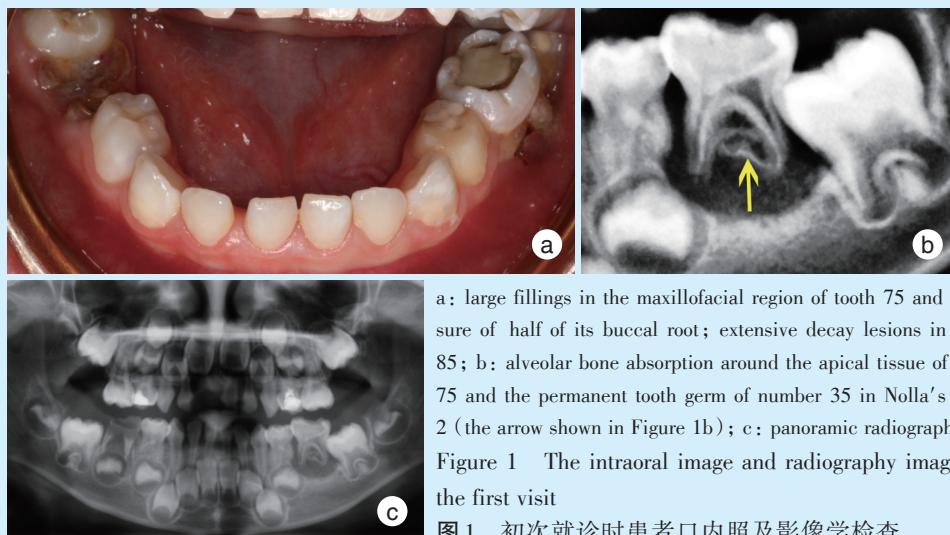
乳牙根尖周炎是常见的儿童口腔疾病,主要是指乳牙根尖周围的牙槽骨、牙骨质、牙周膜等组织的炎症。多由牙髓病发展而来,牙齿外伤、不当的牙髓治疗、物理或化学刺激等亦可引起根尖周炎<sup>[1-2]</sup>。长期反复发作的根尖周炎会破坏乳牙牙根与继承恒牙牙胚间的骨阻隔,造成继承恒牙釉质发育不全,或者恒牙萌出异常。严重者可致炎症浸润至恒牙胚,进而使其发育畸形、甚至坏死。临幊上较常見到乳牙根尖周炎引起恒牙胚发育异常,但根尖周炎致继承恒牙胚坏死并不多见,笔者所在科室收治1例因乳牙根尖周炎致继承恒牙牙胚坏死的病例,现报道如下。

## 1 病例报道

患者,女,5岁,因左下后牙肿痛3 d来诊。患

儿半年前于外院行75牙齿充填治疗,1周前自觉疼痛,呈渐进性加重。既往体健,否认系统病史及药物过敏史。检查:75殆面见大面积充填物,颊侧牙根暴露至根中1/2,叩痛(++) ,牙龈红肿,未见溢脓(图1a),Ⅱ°松动;85大面积缺损,可见髓底;36近中倾斜,口腔卫生状况不良。X线片示:75根尖周区低密度影像,35牙胚发育至Nolla 2期,密度减低,牙囊硬骨板包绕不完整(图1b);45牙胚发育至Nolla 4期(图1c);34牙胚远中、36牙胚近中骨白线不连续。诊断:①75慢性根尖周炎;②85残根;③35牙胚坏死? 治疗计划:①拔除75,85患牙,观察34、35、36恒牙胚发育及牙槽骨愈合情况,定期复查;②用扩弓簧直立36,75、85缺牙处行间隙保持。

术中拔除75时发现35恒牙胚与75牙根及周



a: large fillings in the maxillofacial region of tooth 75 and exposure of half of its buccal root; extensive decay lesions in tooth 85; b: alveolar bone absorption around the apical tissue of tooth 75 and the permanent tooth germ of number 35 in Nolla's stage 2 (the arrow shown in Figure 1b); c: panoramic radiograph

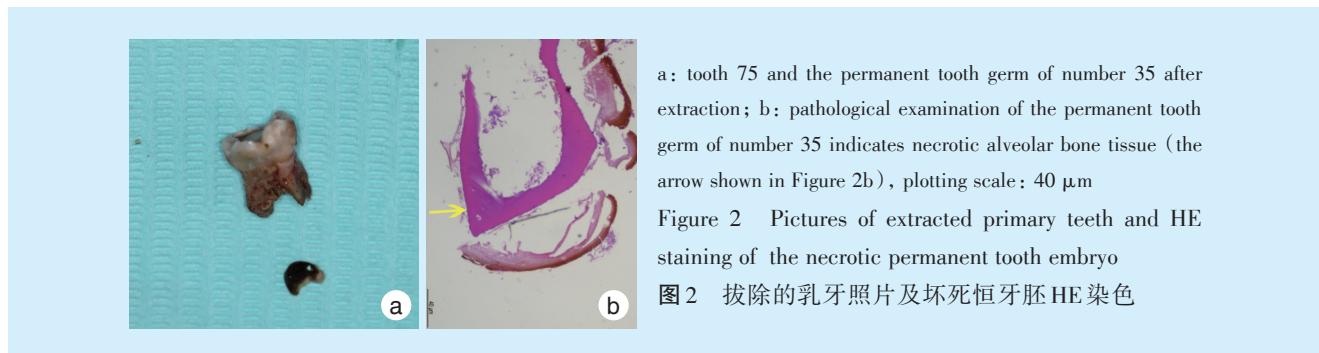
Figure 1 The intraoperative image and radiography images at the first visit

图1 初次就诊时患者口内照及影像学检查

围牙槽骨粘连,一起拔出。75根分叉处见肉芽组织,牙根吸收至根尖1/3;35恒牙胚色黑,长约5 mm,呈镰刀状(图2a);HE染色显示多处死骨形成(图2b)。

患者5个月后复诊。检查:75拔牙窝愈合良好,36、46近中倾斜;74、84Ⅱ度松动。曲面断层

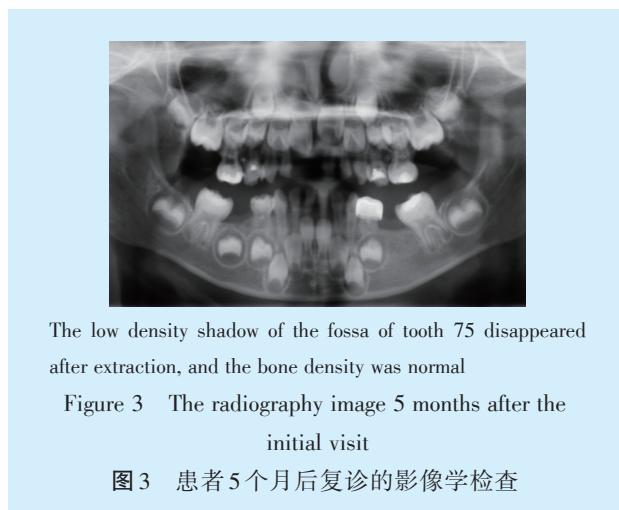
片示:75拔牙窝低密度影消失,骨密度正常。74、84牙根吸收至根中1/2,牙槽骨吸收至根尖1/3。34牙胚远中、36近中骨白线连续(图3)。治疗计划:因74、84松动Ⅱ°,扩弓簧及间隙保持器无法固位,待替换成恒牙列后应用固定矫治排齐牙列。



a: tooth 75 and the permanent tooth germ of number 35 after extraction; b: pathological examination of the permanent tooth germ of number 35 indicates necrotic alveolar bone tissue (the arrow shown in Figure 2b), plotting scale: 40 μm

Figure 2 Pictures of extracted primary teeth and HE staining of the necrotic permanent tooth embryo

图2 拔除的乳牙照片及坏死恒牙胚HE染色



The low density shadow of the fossa of tooth 75 disappeared after extraction, and the bone density was normal

Figure 3 The radiography image 5 months after the initial visit

图3 患者5个月后复诊的影像学检查

## 2 讨论

### 2.1 病因和发病机制

健康的乳牙对继承恒牙的顺利萌出有一定的诱导作用。严重的乳牙根尖周病变更会引起恒牙的发育缺陷。乳牙的根尖周病变程度及牙胚的发育阶段对继承恒牙胚的正常生长发育存在不可预估的影响。牙冠发育完成前,乳牙根尖周病变更会造成继承恒牙釉质发育不全,如特纳牙。乳牙牙髓坏死后,牙齿缺乏神经及血管的营养支持,自噬相关蛋白含量升高<sup>[3]</sup>。此外,炎症渗出物的进入有利于蛋白溶解性根管细菌的生长,并加速其生长繁殖。髓腔及根尖周主要为革兰阴性厌氧菌<sup>[4]</sup>,在释放内外毒素、酶等有害物质的同时,细胞因子IL-1α 和 IL-1β 的表达水平升高<sup>[5]</sup>,核因子κB 配体受体激活,促炎细胞因子如 TNF-α、前列腺素 E2 和 IL-17 等积累<sup>[6]</sup>,使得宿主激活的免疫系统介导的破骨细胞数量及活性增加,引起骨吸收<sup>[7]</sup>。骨组织结构长期破坏,血供不足,继而骨坏死。当根尖周牙槽骨溶解破坏,局部机械屏障消失,牙根吸收,可能会使恒牙异位萌出、替换异常,形成含牙囊肿<sup>[8]</sup>,甚至坏死(图2)等现象。

近年来国内外多见恒牙胚发育异常的病例,

但恒牙胚致死的病例鲜有报道。Cordeiro 等<sup>[9]</sup>报道了1例因75大面积缺损、髓底穿孔而拔除的病例,拔除后发现35牙胚继续发育,但牙冠形态异常,外观类似牙瘤。张帆<sup>[10]</sup>等报道了1例75根尖周病变,75根分叉至根尖区大面积暗影,35牙胚未见明显的牙囊硬骨板包绕,临床检查时发现,患牙Ⅰ度松动,牙龈红肿,75行根管治疗,11个月后随访观察时发现35恒牙胚继续发育,较45恒牙胚发育滞后。Valderhaug 等<sup>[11]</sup>通过动物实验研究发现,有87%的实验牙产生了根尖周炎,但其中仅有3%的患牙因炎症波及恒牙胚而影响了恒牙的形态或牙齿形成。作者认为,牙根较短和乳牙的病理性牙根吸收可能是阻断炎症感染恒牙胚的主要原因。这可能也从一方面揭示了为什么大多数乳牙根尖周炎没有影响继发性恒牙胚的正常生长发育。

本例患者因严重的乳牙慢性根尖周炎,导致牙周骨质广泛性破坏,35牙胚及牙囊周围骨白线包绕不完整,较45发育滞后。初期怀疑35因炎症停止发育。拔除75时,术中发现恒牙胚已坏死。究其原因,除了乳牙结构上的特点,比如多数乳牙矿化度低,易磨耗<sup>[12]</sup>,牙髓组织血运丰富,因此在口腔复杂环境中更容易因龋坏等原因而发生感染,并易向髓室底、根尖区扩散。此外患儿的生活习惯,口腔卫生护理<sup>[13]</sup>以及家长的医学知识、重视程度<sup>[14]</sup>等对患儿口腔疾病的早期预防、诊断和治疗效果产生影响。

### 2.2 诊断

判断恒牙胚是否受到影响的主要方法是临床检查结合放射学检查。若乳牙存在牙体缺损、自发性疼痛等临床症状,且放射学检查显示牙胚周围骨壁缺损、显像不清,说明恒牙胚可能受到损害。但不是所有乳牙的根尖周炎均会影响恒牙胚的发育。如果乳牙根尖周病发生在恒牙牙釉质形成和矿化完成后,并且采取了及时有效的干预措施,乳牙的根尖周炎一般不会波及到恒牙胚。

本病例在术前对35恒牙胚的状态难以做出明



確診斷，因此選擇在拔牙術中作進一步檢查。拔除時發現35恒牙胚在顏色、形態上的異常，且與75根分叉區產生粘連，因此選擇一同拔除。這提示在此類病例的臨床診斷和處理當中應當謹慎，避免產生因診斷不確或處理不當而導致的不良後果。

### 2.3 治療與預後

當確診乳牙根尖炎引起恒牙胚發育異常後，治療方法上的選擇也應慎重。通常選擇保守療法，比如炎症的引流、抗炎治療等，與此同時，積極隨訪觀察根尖病變的發展情況和繼承恒牙胚的發育情況也尤其重要。但對於因炎症造成牙槽骨破壞、引起恒牙胚發育遲緩，患牙是否拔除值得商榷。應結合患牙臨床症狀、對鄰牙的影響、繼承恒牙胚發育情況等綜合評估。若表現為局限性的慢性炎症，恒牙胚未見明顯異常，牙根完整利於充填者可考慮行根管治療<sup>[15]</sup>。術後定期複查觀察根尖周轉歸情況，炎症控制不佳則需拔除患牙<sup>[16]</sup>。本例患牙炎症浸潤廣泛，松動達Ⅱ度，頰側少有骨質包繞，牙囊周圍硬骨板包裹不完整，因此選擇盡早拔除。

### 2.4 總結

臨牀上對發育異常的恒牙胚的診斷和治療都不甚明確。如本病例，患有嚴重慢性根尖周炎的乳牙，若檢查發現其恒牙胚與對側同齡牙相比發育明顯遲緩，牙胚周圍骨白線消失，首先應積極採取合理有效的干預措施，如對引起炎症的乳牙進行根管治療，並且隨訪觀察。若發現恒牙胚影像不明顯，懷疑可能為牙胚先天缺失或壞死。向患者及家屬說明情況後，確定治療方案。建議方案如下：①方案一，若乳牙仍能保留，在控制炎症後，進一步觀察；若後期炎症無法控制，則拔除乳牙，術中進一步檢查恒牙胚狀態；②方案二，若乳牙無法保留，應在拔除乳牙過程中檢查恒牙胚狀態後再決定是否需要在拔牙過程中將恒牙胚一同拔除，以免造成殘留炎症進一步擴散，累及臨牙。

### 參考文獻

- [1] Fouad AF, Khan AA, Silva RM, et al. Genetic and epigenetic characterization of pulpal and periapical inflammation[J]. Front Physiol, 2020, 11: 21.
- [2] 馬向玉, 李建華, 彭弘達, 等. 單形束CT應用於第一乳磨牙根管形態的研究[J]. 口腔疾病防治, 2019, 27(6): 387-390.  
Ma XY, Li JH, Peng HD, et al. Study on the application of cone-beam CT in root canal morphology of first deciduous molars[J]. J Prev Treat Stomatol Dis, 2019, 27(6): 387-390.
- [3] Qi S, Qian J, Chen F, et al. Expression of autophagy associated proteins in rat dental irreversible pulpitis[J]. Mol Med Rep, 2019, 19(4): 2749-2757.
- [4] Nelson-Filho P, Ruvíere DB, de Queiroz AM, et al. Comparative molecular analysis of gram-negative bacteria in primary teeth with irreversible pulpitis or periapical pathology[J]. Pediatr Dent, 2018, 40(4): 259-264.
- [5] Yang NY, Yan Z, Zhao HY, et al. Increased interleukin 1 $\alpha$  and interleukin 1 $\beta$  expression is involved in the progression of periapical lesions in primary teeth[J]. BMC Oral Health, 2018, 18(1): 124.
- [6] Rechenberg DK, Bostancı N, Zehnder M, et al. Periapical fluid RANKL and IL-8 are differentially regulated in pulpitis and apical periodontitis[J]. Cytokine, 2014, 69(1): 116-119.
- [7] Souza JAC, Magalhães FAC, Oliveira GJPL, et al. Pam2CSK4 (TLR2 agonist) induces periodontal destruction in mice[J]. Braz Oral Res, 2020, 34: e012.
- [8] Dhupar A, Yadav S, Dhupar V, et al. Bi-maxillary dentigerous cyst in a non-syndromic child--review of literature with a case presentation[J]. J Stomatol Oral Maxillofac Surg, 2017, 118(1): 45-48.
- [9] Cordeiro MM, Rocha MJ. The effects of periradicular inflammation and infection on a primary tooth and permanent successor[J]. J Clin Pediatr Dent, 2005, 29(3): 193-200.
- [10] 張帆, 任偉偉, 曲幸輝, 等. 乳磨牙根尖周病影響繼承恒牙胚發育1例[J]. 臨床口腔醫學雜誌, 2018, 34(9): 513.  
Zhang F, Ren WW, Qu XH, et al. Periapical disease of primary molars affected the development of permanent successor[J]. J Clin Stomatol, 2018, 34(9): 513.
- [11] Valderhaug J. Periapical inflammation in primary teeth and its effect on the permanent successors[J]. Int J Oral Surg, 1974, 3(4): 171-182.
- [12] Pineda-Higuita S, Saldaña-Bolívar V, González-Penagos C, et al. Characteristics and severity of tooth wear in 2 to 5-year-old kindergarten children in Medellín[J]. Acta Odontol Latinoam, 2019, 32(2): 75-78.
- [13] Pierce A, Singh S, Lee J, et al. The burden of early childhood caries in Canadian children and associated risk factors[J]. Front Public Health, 2019, 7: 328.
- [14] Al-Batayneh OB, Al-Khateeb HO, Ibrahim WM, et al. Parental knowledge and acceptance of different treatment options for primary teeth provided by dental practitioners[J]. Front Public Health, 2019, 7: 322.
- [15] Siqueira Junior JF, Rôças IDN, Marceliano-Alves MF, et al. Unprepared root canal surface areas: causes, clinical implications, and therapeutic strategies[J]. Braz Oral Res, 2018, 32(Suppl1): e65.
- [16] Lee J, Kang S, Jung HI, et al. Dentists' clinical decision-making about teeth with apical periodontitis using a variable-controlled survey model in South Korea[J]. BMC Oral Health, 2020, 20(1): 23.

(編輯 張琳, 錢虹)



官网

公众号