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Temporary arrest of root development in a premolar of a child with hypodontia and extensive caries

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Summary. The arrested development of a permanent tooth as a sequela to a periradicular infection in a primary predecessor is a rare occurrence. A case is presented where temporary arrest of root development occurred in a premolar of a child with hypodontia and extensive caries. The aetiology, management and outcome are discussed.

Introduction

Local factors which may cause arrested root development in a permanent tooth include infection, trauma, teeth in the fracture line of the jaw and localized odontodysplasia. General factors which may lead to arrested root development include radiation to the maxilla or mandible during the period of root development [1] and regional odontodysplasia.

There are many reports in the literature on the effects of periradicular infection in a primary tooth on the permanent successor [2–8]. Early eruption, rotation, hypomineralization and hypoplasia are the most frequently reported sequelae. The arrest of tooth development as a sequela to a periradicular infection in a primary tooth is a comparatively rare occurrence [9–11]. Partial or complete arrest of root formation is a rare complication occurring in 2% of traumatized teeth [12]. It is typically associated with avulsion of a primary maxillary incisor between the age of 5 and 7 years. The complete arrest of root development has also been reported as a result of the direct involvement of a developing tooth in a jaw fracture line [13–16]. Localized odontodysplasia may also result in arrested tooth development.

Hypodontia is defined as the congenital absence or agenesis of one or more teeth. Excluding the third molar, population prevalences of hypodontia across the world vary between 3·5% and 6·5% [17]. In Caucasians, the most commonly affected teeth are third molars (25–35%), upper lateral incisors (2%) and lower second premolars (3%). Females are more commonly affected than males.

There are few reports in the literature of arrested development of the permanent tooth germ following an insult such as trauma or infection. Ultimately, the tooth erupts and exfoliates because of a lack of root support or the arrested tooth germ is sequestered [18,19]. There are no reports in the literature on the resumption of tooth development following removal of the cause of arrest. A case is presented where temporary arrest of root development occurred in a child with hypodontia and caries.

Case report

A 9-year-old female patient presented to the Department of Paediatric Dentistry at the University Dental School and Hospital, Cork, Ireland. Her mother was concerned about the appearance of her teeth. The girl had no relevant medical history. She was an irregular dental attender and a lifetime resident of a fluoridated area.

Intraorally, the subject was in the mixed dentition phase, with gross caries involving her primary and permanent dentitions, hypodontia, microdontia and very poor oral hygiene (Figs 1 & 2). The loss of occlusal vertical dimension was confirmed by the lack of intact occluding units. Clinically and radiographically, she was missing 18, 17, 15, 12, 22, 25,

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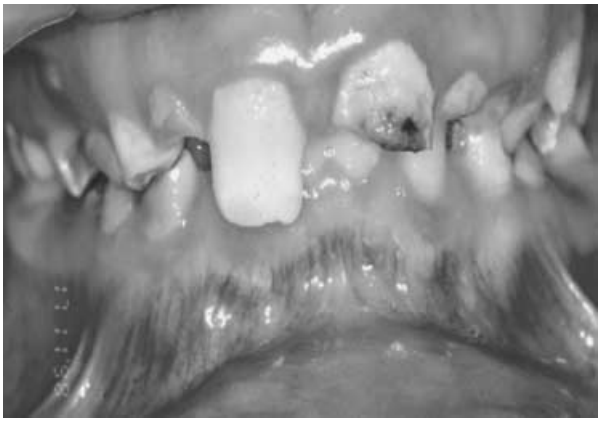

Fig. 1. Preoperative view of the subject's mouth: note the poor oral hygiene, gross caries and the sinus above 54.

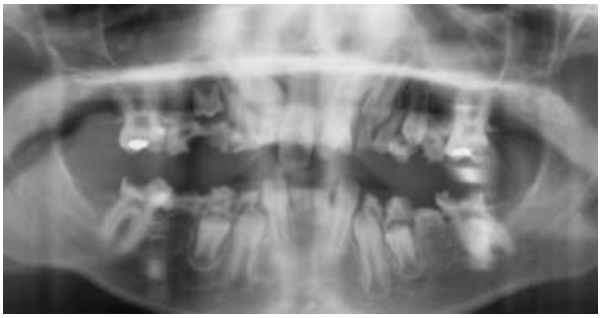

Fig. 2. Preoperative orthopantomograph taken when the subject was aged 9·9 years: note the hypodontia and the development of 14 relative to 24.

27, 28, 38, 37, 35, 31, 41, 45, 47 and 48 (Fig. 2). The tip of the buccal cusp of the partially erupted 14 was visible buccally. It appeared to be erupting through a sinus related to its primary predecessor (Fig. 1). Radiographically, the partially erupted 14 was surrounded by an ill-defined radiolucency and development lagged behind its contralateral (Fig. 2). The radiograph shown in Fig. 2 was taken at the first presentation, when the child was 9·9 years old and 14 was at the crown complete stage of development. The mean age of completion of crown development in first premolars of females is 6·3 years, with 90% of females reaching this stage by 7·5 years [20]. Therefore, the development of 14 was either delayed or arrested. The differential diagnosis included: infection of the developing tooth germ as a result of spread from an infected primary predecessor, a dentigerous cyst, trauma to the primary predecessor, irradiation to the region of the developing tooth germ, localized odontodysplasia. From the medical history,

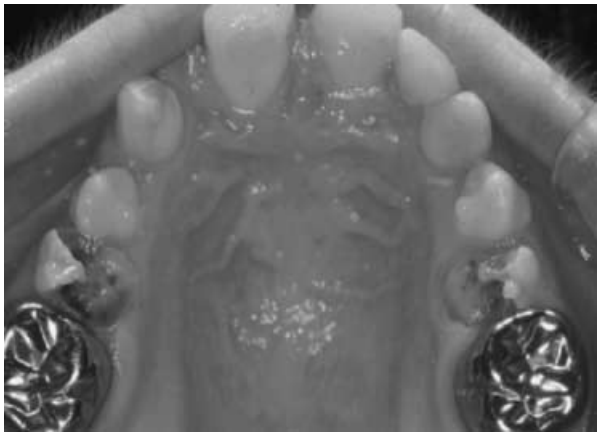

Fig. 3. Postoperative view of the maxillary arch: 14 has erupted with no evidence of hypoplasia or hypomineralization; 55 and 65 are ankylosed with no permanent successors and remain as space maintainers.

as well as the clinical and radiographic presentation, it was concluded that 14 had arrested root development associated with a chronic periradicular infection in the tooth. The tip of the buccal cusp of the partially erupted 14 was visible buccally because of the chronic nature of the infection and resultant bone resorption (Figs 1 & 2). Based on reports in the literature [11], the prognosis for the continued development of 14 was poor.

The aims of treatment were to enable the patient to maintain a healthy oral environment, maintain as many teeth as possible, improve function, improve aesthetics and maintain space for missing units.

An orthodontic opinion was sought at the start of treatment. Preventive advice was given in the form of oral hygiene and diet advice, which was reinforced at each visit. Fifty-four was extracted and the development of 14 was monitored.

The occluding vertical dimension, function and aesthetics were restored. Space for missing units was retained by maintaining the corresponding primary predecessors (Figs 3 & 4). Tooth 14 erupted and was slightly rotated, but there was no evidence of enamel hypoplasia or hypomineralization associated with it (Fig. 3). Root development of 14 continued following the extraction of 54, but lagged behind the contralateral tooth (Figs 5, 7 & 8).

Analysis of periapical radiographs taken when the child was 12·5 years old indicated that the root development of 24 (Fig. 7) was at the apex complete stage and corresponded to a mean developmental age of 12·6 years [20]. The periapical view of 14 (Fig. 8), however, indicated that root development was at the

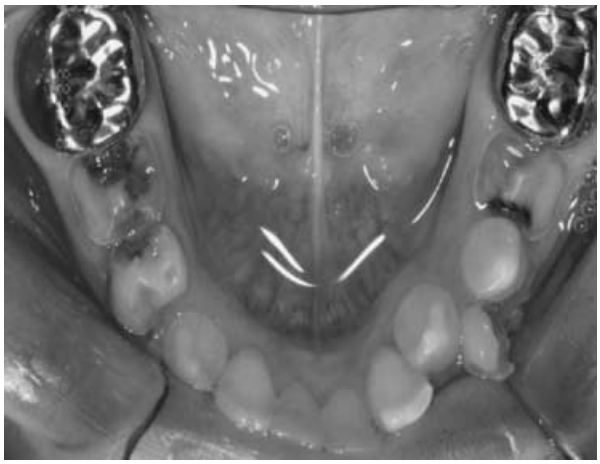

Fig. 4. Postoperative view of the mandibular arch: 75 and 85 are ankylosed with no permanent successors and remain as space maintainers; 73 and 84 are exfoliating.

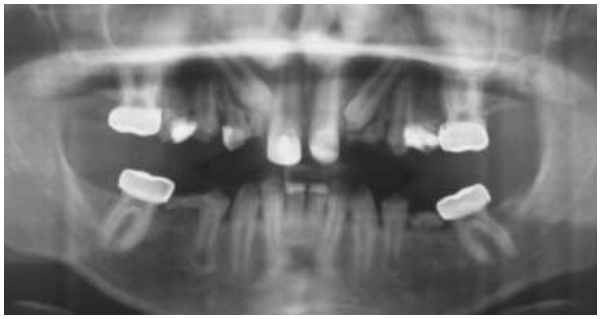

Fig. 5. Orthopantomographic radiograph taken when the subject was aged 11·5 years: note that the development of 14 has continued, but lags behind 24, which is at the apex complete stage of development.

root three-quarters stage of development and corresponded to a mean developmental age of 10·4 years [20]. The development of 24 was therefore as expected for the child's age, whereas the development of 14 was delayed. The time difference between the developmental stages of 14 and 24 was 2·2 years, according to Haavikko [20], or 3·1 years, according to Anderson *et al*. [21], representing a delay of 2·2–3·1 years in the development of 14 relative to 24 and to the expected stage of development of this tooth for this child's age [20,21]. The root development of 24 was complete radiographically by the age of 11·5 years (Fig. 5), whereas the root development of 14 was not radiographically complete until 13·6 years (Fig. 6).

Discussion

Although there are many reports of hypoplasia and hypomineralization of the permanent tooth as a consequence of infection in the primary predecessor, there are few reports of arrested root development as a sequela [10,11]. There are no reports in the literature on resumption of tooth development following removal of the cause of arrest. Therefore, the authors' main aim was to describe the temporary arrest of root development in a permanent tooth germ as a consequence of a grossly carious primary tooth that had subsequently developed an interradicular infection. The fact that this child also presented with gross caries and hypodontia emphasized the importance of a successful treatment outcome.

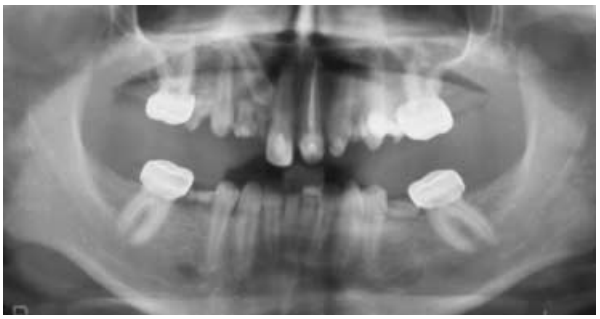

Fig. 6. Orthopantomographic radiograph taken when the subject was aged 13·6 years: 14 is now at the apex complete stage of development.

There is a known association between amelogenesis imperfecta and hypodontia. It is possible that caries was superimposed on a congenital or acquired hypoplasia, but there was insufficient sound tooth tissue to determine this at presentation. Permanent teeth which erupted subsequently, however, showed no evidence of hypoplasia, but were microdont. Although hypodontia and microdontia are often associated with a syndrome (e.g. X-linked hypohydrotic ectodermal dysplasia, autosomal dominant ectodermal dysplasia, Ellis–Van Creveld syndrome, Down syndrome, or cleft lip and palate [22,23]), there was no evidence of any associated syndrome in this case.

Analysis of periapical radiographs (Figs 7 & 8) indicated that the root development of 14 was delayed by 2·2–3·1 years compared to 24 [20,21]. According to Haavikko [20], however, there is no significant difference in the median age of the formation of teeth of the same series on the right or left side of the dental arch. It is difficult to be entirely certain that the development of 14 had been arrested or delayed because the infected primary predecessor was extracted almost immediately after presentation and tooth development appeared to resume. A series of

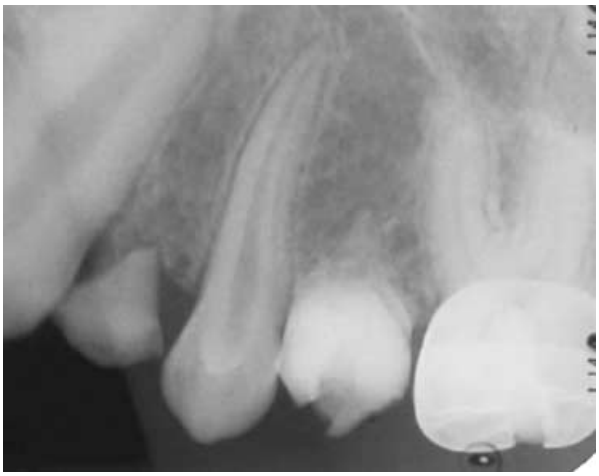

Fig. 7. Periapical view of 24 taken when the subject was 12·5 years: 24 is at the apex complete stage of development and corresponds to a mean development age of 12·6 years [20].

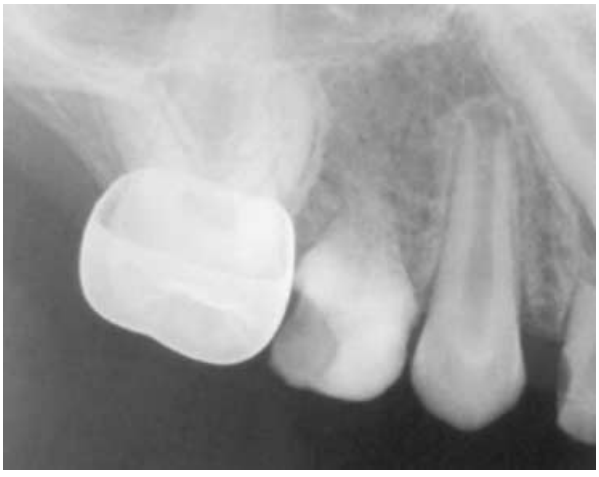

Fig. 8. Periapical view of 14 taken when the subject was 12·5 years: 14 is at the root three-quarters stage of development and corresponds to a mean developmental age of 10·4 years [20].

radiographs taken prior to the extraction of the deciduous predecessor would have been required to be certain that this was the case.

Root development of 14 continued and was complete 25 months after the contralateral (Figs 5 & 6). Therefore, it is likely that the follicular tissues were chronically inflamed, but the tissue cells remained vital. This would explain why development apparently continued following removal of the source of infection.

The pathogenesis of arrested tooth development in this case was related to the close proximity of the permanent premolar tooth germ to the interradicular area of the primary molar [2]. Pulpal infection and necrosis of the primary molar leads to periradicular inflammatory changes [24]. Acute inflammation leads to tissue destruction and bone resorption. The follicular cells, particularly the ameloblasts, are sensitive to changes in their environment, especially during the process of amelogenesis. In 20–30% of cases, inflammation will spread to the follicular tissues of the underlying permanent tooth germ, resulting in enamel hypoplasia or hypomineralization [3,6,8,24]. Occasionally, degenerative or destructive changes occur which involve all the tissues of the developing tooth germ, resulting in arrested development [10,11] and possibly sequestration [25].

Factors which may determine the spread of infection from the affected primary tooth to the underlying permanent tooth germ, and the nature and severity of changes which occur, include: the stage of development of the permanent tooth germ, the duration of the infection, the virulence of the organisms involved, and the local and general resistance of the host. Inflammatory infiltration of the follicular tissues at an early stage of development is more likely to result in serious sequelae for the permanent tooth germ, and the longer an infection in a primary tooth remains untreated, the greater the likelihood of damage to the permanent successor [8].

In this case, 14 erupted and there was no evidence of hypomineralization or hypoplasia. The chronic nature of the infection was indicated by the presence of a sinus, clinical and radiographic evidence of bone resorption, and the fact that development of 14 lagged significantly behind the contralateral. The lack of hypoplasia or hypomineralization of the crown may be because crown development was complete at the time of the onset of infection, the organisms involved were not very virulent or the resistance of the host was good.

Had root development of the permanent tooth failed to continue following removal of the primary predecessor, it would have been necessary to continue to monitor the tooth clinically and radiographically for signs of infection. Ideally, the tooth would have been removed after eruption since it had a very short root, and would not make a viable short- or long-term dental unit. If the arrested tooth germ had failed to erupt, it would have been necessary to enucleate it surgically.

It is important to distinguish the arrest of tooth development from a dentigerous cyst so that appropriate observation or intervention occurs. Dentigerous cyst formation involving the unerupted permanent tooth may also follow infection of the primary predecessor [10,25]. Presentation may be acute, with facial swelling, or chronic with bony expansion.

Radiographically, the dentigerous cyst is seen as a well-demarcated radiolucency surrounding only the crown of the permanent successor. Arrested development, however, is associated with an ill-defined radiolucency surrounding the whole tooth germ, as in this case. Unlike the tooth germ with arrested development, the dentigerous cyst is accompanied by further root growth. Marsupialization of the dentigerous cyst is required to facilitate eruption of the tooth.

Conclusions

Periradicular inflammation of a primary tooth may result in a range of damaging sequelae for the underlying developing tooth germ. This may lead to the eruption of a permanent tooth with impaired aesthetic or structural characteristics, or the arrest of the developing tooth germ. This has cost implications for further short- and long-term treatment needs. Early intervention and treatment of carious primary teeth in the form of pulp therapy or extraction is necessary to prevent these unwanted sequelae.

This case illustrates that, even when a longstanding chronic infection in a primary molar causes arrest of development of the permanent successor, it may be temporary. Therefore, it is always worth considering extracting the primary predecessor and monitoring the development of the successor before considering enucleation of the permanent tooth germ, as recommended by Brook and Winter [11]. It was particularly worthwhile in this case since it provided a vital unit in this quadrant, where the patient is already congenitally missing 17, 15 and 12.

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Résumé. Un arrêt du développement d'une dent permanente, séquelle d'une infection périradiculaire de la dent temporaire est un événement rare. Un cas est présenté où l'arrêt temporaire du développement s'est produit au niveau d'une prémolaire chez un enfant avec agénésie et caries nombreuses. L'étiologie, la prise en charge et le résultat sont discutés.

Zusammenfassung. Die Entwicklungsstörung eines bleibenden Zahnes als Folge einer periradikulären Infektion des vorangehenden Milchzahnes ist selten. Der Fall einer vorübergehenden Entwicklungsstörung an einem Prämolaren bei einem Kind mit Hypodontie und extensiver Karies wird vorgestellt. Ätiologie, Therapie und Prognose werden diskutiert.

Resumen. La detención del desarrollo de un diente permanente como una secuela de una infección perirradicular en su predecesor deciduo es un hecho raro. Se presenta un caso en que la detención temporal del desarrollo radicular ocurrió en el premolar de un niño con hipodoncia y caries extensa. Se discuten, la etiología, el tratamiento y el resultado.

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