In spite of the advances in prevention of dental caries in dentistry, the emergence of primary teeth with pulp involvement is still a challenge causing premature loss of teeth. Pulpectomy of the irreversibly inflamed or necrotic pulp of primary teeth remains the common treatment approach with variable prognosis. The variation in prognosis by and large is due to the anatomical root variations and the complex canalicular system; making the debridement and biomechanical preparation of canals difficult to achieve [1].

Untreated carious primary teeth or failure of endodontically treated primary teeth may cause deleterious effects as a result of periapical infection spread to the hard and soft tissues in the vicinity. It is crucial to remember that the follicular tissues of the succedaneous teeth are very close to the bifurcation and apices of primary molars and an infection may easily reach the follicle of a developing tooth causing inflammation of the follicular tissues or development of a cyst.

Dentigerous cyst (DC) is a developmental odontogenic cyst that invariably occurs between the second and third decade with low incidence in young individual. However, they may develop in association with unerupted premolars or supernumerary teeth [2,3]. Although the DCs are developmental in origin, there is a strong association between the DC development and the inflammation spreading from nonvital predecessor teeth [4].

In this report, we present a case of pulp therapy of primary tooth with no evidence of post-therapeutic follow up causing development of inflammatory DC in association with unerupted mandibular 2nd premolar and massive bone destruction in a 10-year-old child. The child was presented to the pediatric department, RAKCods clinic with severe pain and swelling on the left side of the mandible for 4 weeks before presentation. Extra-oral examination revealed a single diffuse swelling on the left side of the mandible. Intra-oral examination showed a bony hard swelling in the 74, 75 regions obliterating the buccal vestibule. Mandibular buccal cortex expansion was evident but not the lingual. The primary left 2nd mandibular molar tooth was non-vital, showing evidence of pulp therapy and composite filling. The involved tooth was slightly mobile and the adjacent soft tissues were normal with no signs of inflammation. The permanent first molar (36) was sound and the pulp vitality was not compromised. Orthopantamograph revealed an oval-shaped unilocular radiolucency around the developing second premolar with partial sclerotic border. The mesial root of 74 showed resorption with loss of bone in the bifurcation area. The cone beam computed tomography images revealed thinning of the buccal and lingual cortex [Figure 1–3]. A provisional diagnosis of dentigerous or bifurcation cyst was made. Owing to the behavior of the lesion, it was decided to refer the patient to the oral surgery department, Saqr hospital, RAK for enucleation of the cystic lesion including the associated teeth. During surgery, the cystic lining was found attached to the cervical margin of the 2nd premolar crown revealing a diagnosis of DC. Furthermore, the histopathology report of the surgical specimen confirmed the diagnosis; in addition, the cystic lining was heavily inflamed masking the classical microscopic appearance.

Soon after the full eruption of 34 crown a space maintainer (band and loop) was fitted in place until further treatment [Figure 4]. The 3-months radiographic follow up showed progressive bone regeneration filling the cavity and excellent soft tissue healing.

DC is commonly associated with mandibular 3rd mandibular molar [5]. However, in the current case, the cyst was associated with unerupted mandibular 2nd premolar. Although such cases are relatively uncommon, a few cases have been reported [6]. Shibata et al. [7] studied the occurrence of DC in association with succedaneous teeth during the transitional dentition phase and reported a prevalence of 77.1% in the premolar region. There have been several explanations for the development of inflammatory and non-inflammatory DC. Benn and Altini [8] suggested three pathways for histogenesis of DC. In the first scenario, the developmental DC arises from the dental follicle and becomes secondarily infected as a result of a non-vital tooth. The second form occurs when a permanent successor erupts into radicular cyst that forms at apex of a non-vital deciduous resulting into a DC that is extra follicular in origin. Nevertheless, a radicular cyst developing at
The apex of primary tooth is extremely rare. The third possible cause is due to spread of peri-apical inflammation from a non-vital deciduous tooth to a follicle of permanent successor.

Generally, two main surgical approaches are usually followed for management of such cystic lesions; either enucleation or marsupialization. Several factors are taken into consideration, including the size and location of the lesion, the amount of bone loss, integrity of the cystic wall, and its relation to vital structures.

Conservative approach, the marsupialization has been advocated for management of DC in children to provide a chance for the unerupted tooth to erupt [9]. Biopsy and histopathological examination of tissues remains the golden standard for any lesion. Thus, the latter approach has a disadvantage, as the cystic lining is left behind and remains without thorough microscopic examination. In the current case, because of the unusual massive bone destruction, enucleation of the cyst including the unerupted tooth and histopathological examination of the cystic tissues were deemed necessary to exclude any nasty changes.

To conclude, although the development of DC in association with an unerupted successor due to apical spread of inflammation is relatively uncommon, periodic follow-up and assessment of prognosis are extremely important to prevent or reduce the potential morbidity associated with the same.

Disclosure statement
No potential conflict of interest was reported by the authors.

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