LESIONS ASSOCIATED WITH UNERUPTED OR ERUPTING TEETH IN CHILD AND ADOLESCENT PATIENTS

Editor
BURCU GÜÇYETMEZ TOPAL

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İÇİNDEKİLER

TOOTH ERUPTION PROCESS

MELİKE TIRAŞ[1]

Introduction

Tooth eruption is the physiologic process of the movement of the teeth through the jaw to their position in the oral cavity in functional closure. This process starts at an average age of 6 months and may cause local inflammatory symptoms as well as signs and symptoms in the general health of infants and children. (Canto et al. 2022) Carlson (1944) presented 5 opinions about tooth eruption as a result of radiographic examinations. According to this

1. Tooth eruption starts after the formation of the crown of the tooth.

2. Root formation of teeth begins before movement occurs in the basal bone and crown.

3. A significant part of the root growth of teeth occurs in the eruption phase before bite.

4. Root development is completed within the basal bone.

[1] Special Pediatric Dentist, Republic of Türkiye Ministry of Health ,Sivas Oral and Dental Health Hospital, Orcid: 0000-0001-7723-1155

5. Tooth eruption is a lifelong process that develops slowly and depends on the growth of the dentoalveolar structure.

Tooth eruption is examined in 5 stages. These stages are pre-eruption movements, intra-osseous eruption, mucosal penetration, pre-occlusion eruption and post-occlusion eruption. (Wang,2013)

Pre-eruption movement

The teeth are in the same position in the bone during crown formation, but minor movement of the teeth has been observed prior to active eruption. (Carlson 1944) It is thought that these movements may be due to both follicular events required for tooth eruption and regional differences due to the growth and development of the jaws. (Björk, 1977)

Intra-osseous eruption

The eruption movement in teeth starts with root formation and continues with the movement of the tooth from its fixed position to the position where the tooth will bite. (Proffit and Frazier-Bowers 2009) The eruption movement depends on the force to move the tooth along the eruption path and the removal of the bone in the eruption path by the deciduous germ. Although it is thought that the increase of cells at the root tip of the deciduous tooth germ creates a force for the eruption of the teeth, it has been observed that tooth eruption continues even when the root tip is cut. (Marks and Schroeder 1996) The eruption path of teeth is formed as a result of osteoclastic movements, but the actual mechanism that causes osteoclastic movement and forms the eruption path has not been fully determined. (Cahill,1969)

When the tubercles or incisals of the teeth reach their position in the arch, the formation of the eruption path is completed and tooth eruption gains speed after this stage. When the erupting tooth reaches the gingival epithelial surface, the enamel epithelium thickens and

changes and the enamel epithelium and gingival epithelium merge. Enamel matrix proteins released prior to mucosal penetration are also thought to cause the symptoms of tooth eruption. (Pierce, Lindskog, and Hammarström 1986)

Pre-occlusion eruption

After mucosal penetration, root development continues during the eruption phase before occlusion. As the dentoalveolar structure continues to grow and develop, the tooth must grow more than the normal amount of growth in order to continue erupting. (Proffit 2016)

Post-occlusion eruption

When the teeth reach the plane of occlusion, the eruption rate decreases significantly compared to the other stages; however, tooth eruption is a lifelong process. (Carlson, 1944) Different factors are thought to cause the continuation of tooth eruption throughout life. These factors include the tendency to maintain occlusion, the effort to maintain the plane of occlusion and vertical dimension, the occurrence of functional bone deformities and direct occlusal forces. (Katona and Qian 2001)

Theories of Tooth Eruption

Root Development Theory

The root development theory suggests that the elongation and growth of tooth roots during the eruption period generate the force needed for tooth eruption. (Kalk, Batenburg, and Vissink 1998) Contrary to this theory, different studies examining human, canine, monkey and rodent teeth have found that teeth without roots also erupt. (Brin et al. 1985; Carl and Wood 1980) It has also been observed that children with impaired root formation due to genetic

and environmental factors continue to wear crowns without root development. (Kalk, Batenburg, and Vissink 1998)

In a study by Cahill and Marks (1980) in which the roots of developing premolars were cut in dogs, the teeth showed normal eruption functions. In another study, the tooth germ structures were removed and the crown structures were replaced with acryl. In this case, tooth eruption did not occur and inflammatory changes were observed in the tooth follicle. (Marks and Cahill 1984) When these studies are evaluated in general, it is concluded that root development is not the only factor required for tooth eruption. (Wang 2013)

Periodontal Ligament Theory

According to the periodontal ligament theory, fibroblasts provide contraction of periodontal ligaments and create the force required for the eruption of teeth. In studies conducted in rabbits and mice, it was observed that the distal parts of the teeth continued to erupt when the tooth roots were cut transversely. (Berkovitz and Thomas 1969; Moxham and Berkovitz 1974) However, the fact that the studies were conducted on rodents that continuously erupt teeth is not compatible with human physiology. (Cahill and Marks 1982) In osteopetrotic mutations of teeth, it has been observed that the teeth do not erupt despite the presence of periodontal ligament. (Marks 1989) On the other hand, it has been reported that eruption continues in Type I dentin dysplasia in which periodontal ligament is absent. (Witkop 1975) In general, when the studies in the literature are examined, tooth eruption cannot be clearly explained by the periodontal ligament theory. (Cahill and Marks 1980)

Hormonal Control Mechanism of Tooth Eruption

Tooth eruption is thought to be affected by hormonal mechanisms. There are studies evaluating the effects of growth

hormone secreted from the pituitary gland and hormones secreted from the thyroid gland on tooth eruption. (Baume, Becks, and Evans 1954b, 1954a)

In studies on thyroidectomized mice, thyroid gland deficiency was found to cause delays in tooth eruption and deformities in dental structures. (Baume et al. 1954a, 1954b) There are studies reporting that children with growth retardation due to growth hormone deficiency and genetically short stature have delays in tooth eruption. (Cohen and Wagner 1948; Garn, Lewis, and Blizzard 1965) However, normal tooth development has been observed in children with growth delays due to other reasons. (Björk 1968) Growth hormone and thyroid hormones are thought to have a synergistic effect on tooth eruption. While growth hormone provides control of the growth process, thyroid hormone has been found to be effective on differentiation and development. (Baume et al. 1954a)

Dental Follicle Theory

The dental follicle, which originates from the cranial neural crest mesenchyme, is a connective tissue surrounding the tooth germs. (Wise, Frazier-Bowers, and D'Souza 2002) In a study by Cahill and Marks (1980) in which the dental follicle was removed from premolar teeth of dogs, it was observed that eruption was prevented. In another study, it was found that tooth eruption continued even though the dental follicle was left and the tooth germ was replaced with an artificial tooth. (Marks and Cahill 1984)

It is thought that the dental follicle initiates and regulates bone resorption and apposition during the intraosseous eruption phase of teeth . The dental follicle also acts as a necessary factor that allows mononuclear cells to migrate to the region during tooth eruption and these cells transform into osteoclasts in the region. Since the dental follicle is located between the alveolar bone and the tooth germ, it is also involved in the cellular events of tooth eruption.

In addition to its effect on osteoclastic activity, the dental follicle can also transmit the signals received from dental struc tures to the relevant points. (Wise et al. 2002)

Studies have shown that the dental follicle is necessary for tooth eruption, and the fact that eruption continues even in the absence of tooth pulp or root supports this theory. (Cahill and Marks 1980; Marks and Cahill 1984)

Hydrostatic Pressure Theory

The hydrostatic pressure theory proposes that the force needed for tooth eruption is created by the difference in hydrostatic pressure between the tissues surrounding the erupting tooth crown and its root apex. (Craddock and Youngson 2004) van Hassel and McMinn (1972) thought that the tissue pressure in the apical part of the erupting teeth of dogs was greater than the occlusal pressure and this created the force required for tooth eruption; however, no relationship between the magnitude of the eruption force and the eruption rate was shown. In contrast to the study supporting this theory, it was also found that there were changes in tooth eruption rate when tissue pressures were changed pharmacologically. (Moxham 1979)

Theory of Bone Remodeling

The theory of bone remodeling suggests that bone resorption takes place coronally to the tooth germ, while bone formation occurs apically to it. The source of osteoclasts and osteoblasts required for resorption and apposition is the dental follicle. (Bhaskar 2012) The observation that molar eruption was delayed in mice injected with bisphosphonate and pamidronate, which slow down bone resorption, revealed that bone resorption is necessary for tooth eruption. (Grier IV and Wise 2016) Likewise, it has been observed that eruption of

teeth is interrupted in the absence of bone formation. (Beertsen et al. 2002)

It is also thought that teeth form the eruption pathway by applying direct and uninterrupted pressure on the bone during eruption. However, it has been observed that the eruption pathway is also formed when the teeth are immobilized in the bone and pressure on the bone is prevented. (Cahill 1969) This has shown that the resorption and apposition of bone in tooth eruption is genetically controlled and not mechanically regulated. (Wise et al. 2002)

Molecular Mechanism of Tooth Eruption

Tooth eruption is a localized and independent event that occurs in a certain time interval. The molecules that initiate tooth eruption, the localization of molecules and the cellular events of eruption are independent for each tooth. (Wise et al. 2002) The molecules required for tooth eruption began to be discovered in 1962 by Cohen (1962) when epidermal growth factor (EGF) was isolated and injected into rodents, accelerating eruption in the incisors of rodents. The eruption of incisors in mice was also found to be accelerated by another growth factor that influences Transforming growth factor-α (TGF-α). (Tam 1985) Considering that TGF-α and EGF bind to the same receptor, it was thought that they have similar effects on tooth eruption. (Todaro, Fryling, and de Larco 1980) However, the fact that tooth eruption continued in mice lacking the TGF-α gene suggests that EGF is a molecule that can initiate tooth eruption alone. (Mann et al. 1993)

Osteopetrotic mice with unerupted teeth have functional Colony stimulating factor-1 (CSF-1) deficiency. When CSF-1 molecule was injected into these mice, it was observed that the teeth started to erupt. (Kodama et al. 1991) In another study evaluating the direct effects of EGF and CSF-1 molecules on tooth eruption, EGF accelerated the eruption of incisors, but EGF had no effect on the

eruption of molars. In contrast to EGF molecule, CSF-1 molecule affected the eruption of molar teeth. The mononuclear and osteoclast increase effect of CSF-1 molecules on the tooth follicle was not shown by the EGF molecule. (Cielinski et al. 2009)

In recent years, experiments on knockout mice have provided more information about the molecules required for tooth eruption. In these studies, it has been observed that teeth do not erupt in the absence of genes that act on osteoclasts. In mice lacking the nuclear factor kappa B1 (NF-κB1) and nuclear factor kappa B2 (NF-κB2) transcription factor genes or the c-fos transcription factor gene, osteoclasts are absent and consequently, teeth cannot erupt. (Franzoso et al. 1997) Mice lacking the osteoclast differentiation factor (ODF) gene, a gene required for osteoclast formation and activation, were also found to lack tooth eruption. (Kong et al. 1999)

It was also reported that injection of Interleukin-1α (IL-1α), another molecule involved in tooth eruption, accelerated tooth eruption by two days in mice with delayed eruption of incisor and molar teeth. (Huang and Wise 2000) Inhibition and activation of osteoclasts are shown in Figure 1.

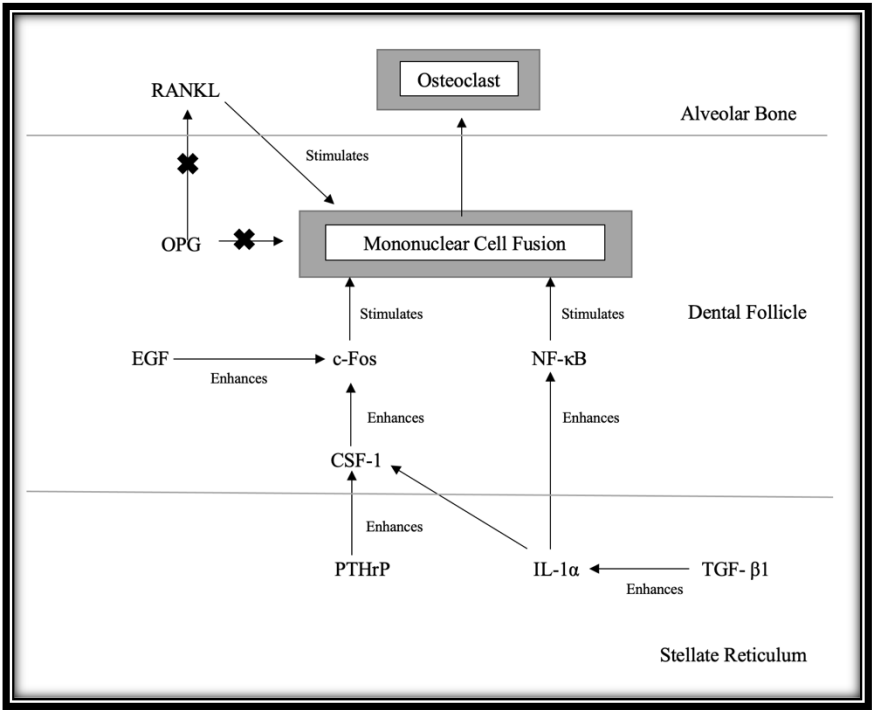

Figure 1: Possible molecular signaling cascade that may promote fusion of mononuclear cells recruited to the dental follicle

**Crossed arrows reflect inhibition of gene expression, other arrows reflect increased gene expression or stimulation of cell fusion*

Current Approaches in Tooth Eruption

From past to present, studies on tooth eruption have been conducted experimentally on animals. (Cahill and Marks 1980, 1982; Carlson 1944; Marks et al. 1996; Marks and Cahill 1984) Tooth eruption in humans cannot be fully explained because the anatomy and physiology of animals are different from humans and because experimental studies on tooth eruption in humans are limited due to ethical values. (Avery and Chiego 2007)

Current approaches to tooth eruption are based on a combination of information obtained from prenatal and postnatal

histologic examination of human teeth and jaws. This information was obtained by examining both healthy and pathologic fetuses with genetic differences. (Avery and Chiego 2007) In the current hypothesis developed in the light of new information, there are three mechanisms;

1. Gap in the tooth eruption pathway: The eruption pathway required for tooth eruption is formed when the dental follicle resorbs the bone on it. The molecular process in this process is carried out by the ectoderm. (Avery and Chiego 2007)

2. Pressure under the dental follicle: Due to the function of the tooth root membrane as a glandular membrane and the innervation of the membrane, pressure can occur on the root surface, periodontal membrane and pulp tissue. (Wise and King 2008) This pressure creates the force required for the tooth to erupt. (Fujiyama et al. 2016)

3.Adaptive capacity of the periodontal membrane: The adaptive and reorganizing capacity of the periodontal membrane is a necessary factor for tooth eruption. It is thought that the mechanism of cell necrosis and apoptosis occurring in the inner layer of the periodontium close to the root occurs thanks to the ability of the periodontal membrane to reorganize. It has been reported that apoptosis mechanisms occur in the eruption pathway of both deciduous and permanent teeth. (Bille, Thomsen, and Kjær 2011)

Theoretically, the pressure that develops in the apical region of the tooth drives the periodontal membrane into continuous adaptation, resulting in the eruption of the tooth. The active movement of the dental follicle resorbs the bone above the follicle. In this context, the crown follicle, periodontal membrane and the apical region of the tooth are structures that interact during tooth eruption. It is thought that the pressure in the apical region may cause

changes in the periodontal membrane and the pressure may affect the crown follicle and lead to bone resorption. (Wise et al. 2001)

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ERUPTION DISTURBANCES

AYÇA HANDE SARİ[1]

Introduction

Tooth eruption is defined as the process by which a tooth moves from its developmental site in the alveolar bone to its functional position in the oral cavity (Massler & Schour, 1941). Following the completion of the crown formation of the teeth in the alveolar bone, an active eruption process commences with the initiation of root formation. The eruption of the teeth is characterised by its progression along the axial direction, which persists until the point of contact with the opposing tooth is reached (Marks Jr & Schroeder, 1996).

The eruption of deciduous and permanent teeth is a multifaceted process influenced by a variety of systemic, local and genetic factors. This process occurs over a broad chronological age range, spanning from infancy to adulthood (Cohen-Lévy & Cohen, 2015). Disruptions in these factors can result in the onset of eruption disorders.

[1] Assistant professor, Afyonkarahisar Health Sciences University, pediatric dentistry, Orcid: 0000-0003-3725-3696

Early Tooth Eruption

There are several etiological factors associated with early tooth eruption (Maheswari, Kumar, & Kumaran, 2012);

- Genetic factors
- Endocrine disorders
- Infectious causes
- Environmental factors
- Malnutrition

Natal and Neonatal Teeth

Teeth that are present at birth are referred to as natal teeth, while those that erupt within one month after birth are known as neonatal teeth (Markou, Kana, & Arhakis, 2012). The prevalence of natal and neonatal teeth in newborns is low, with an incidence of 1 in 2,000 to 3,500 births. These teeth are part of the permanent dentition and only a very small percentage of them are present as supernumerary teeth. These teeth are highly mobile because they erupt before adequate root development is complete (Kana, Markou, Arhakis, & Kotsanos, 2013).

The morphology, colour and structure of natal and neonatal teeth may be different compared to normally erupting primary teeth. The crowns of these teeth are more conical and smaller in size compared to other primary teeth and can be seen in yellow-brown colour with hypoplastic enamel structure (Cunha, Boer, Torriani, & Frossard, 2001).

The most significant complications observed in relation to natal and neonatal teeth pertain to maternal nipple injury during sucking, Riga-Fede syndrome, and the potential risk of aspiration

due to excessive mobility (Ramos-Jorge, Pordeus, Ramos-Jorge, & Paiva, 2011).

Delayed Tooth Eruption

Delayed eruption of teeth is defined as the eruption of a tooth into the oral cavity at a time that deviates significantly from the established norm for different races, ethnicities and genders (Suri, Gagari, & Vastardis, 2004). The delayed eruption of teeth may be the primary or sole symptom of both local and systemic pathologies (Pulse, Moses, Greenman, Rosenberg, & Zegarelli, 2001).

Local Factors

One of the most significant factors that contribute to the delayed eruption of teeth is the presence of a physical barrier. The most common factors in the formation of the physical barrier include supernumerary teeth, odontogenic-nonodontogenic tumours, cysts, ectopic eruption, eruption sequester, scar tissue after trauma-surgery, ankylosis, early loss of primary dentition, and gingival fibromatosis (Suri et al., 2004).

Supernumerary Teeth

Dental supernumerary teeth have been shown to be associated with a range of malocclusal problems, including crowding, dislocation, rotation and delayed eruption of the associated teeth. The presence of supernumerary teeth is a frequent occurrence in the permanent dentition, with a prevalence that is twice as high in males as in females. The most prevalent supernumerary tooth is the mesiodens (Nevil, Damm, Allen, & Bouquot, 2002).

Odontogenic-Nonodontogenic Tumours

Odontomas and some other tumours have been reported to cause delayed eruption of both primary and permanent teeth (Flaitz & Hicks, 2001).

Eruption Cysts

Eruption cysts are benign mucosal lesions that manifest a few weeks before the eruption of teeth. Such lesions are also known as eruption hematomas and exhibit a bluish-purple appearance (Dhawan, Kochhar, Chachra, & Advani, 2012)

Eruption cysts are most commonly associated with primary second molars and permanent first molars. Although the hematoma is generally self-limiting following the eruption of the tooth, surgical intervention may be necessary in rare cases (Aguilo, Cibrian, Bagan, & Gandia, 1998).

Fibrotic Mucosa

The failure of the erupting tooth follicle to fuse with the mucosa is known to result in delayed tooth eruption. Histologic studies show that there are differences in the submucosa between normal tissues and tissues that have undergone trauma or surgery (Di Biase, 1971).

The presence of dense connective tissue and an acellular collagen structure, indicative of hyperplasia in the gums, has been observed to result from various factors, including genetic predispositions, hormonal influences, and medications. These factors have been shown to contribute to delayed eruption of teeth (Katz, Guelmann, & Barak, 2002).

Trauma

The delayed eruption of permanent teeth has been demonstrated to be influenced by traumas sustained on the deciduous teeth. Research has demonstrated that traumatic injuries to the primary dentition can result in ectopic eruption, dilacerations and physical displacement of the tooth germ in the permanent dentition (ANDREASEN, Sundström, & Ravn, 1971; Brin, Ben-Bassat, Zilberman, & Fuks, 1988).

Ankylosis

Ankylosis is defined as the fusion of cementum with the surrounding alveolar bone. Although ankylosis has been observed in both primary and permanent teeth, it occurs with a frequency that is 7-14% higher in primary molars (Silva, Edo, Llorente, & Leache, 2014).

Despite the unknown etiology of ankylosis in primary teeth, genetic factors have been identified as a contributing element in several studies. Moreover, despite the existence of studies indicating a correlation between permanent tooth deficiency and ankylosis in primary teeth (Darling & Levers, 1973), this perspective is not endorsed upon thorough review of the extant literatüre (Dean, 2021).

The diagnosis of ankylosis can be confirmed by clinical and radiographic findings. Ankylotic primary teeth remain inferior to other teeth. This is because the alveolar bone around them continues to develop. Other indicators of ankylosis are a lack of physiological tooth mobility and the presence of a dull sound on percussion. Although limited, two-dimensional radiographic imaging can show loss of the periodontal ligament, external resorption and alveolar replacement (Ducommun, Bornstein, Bosshardt, Katsaros, & Dula, 2018).

In ankylosed primary teeth, a delay of a few months in the underlying permanent teeth erupting is considered normal and follow-up is recommended. If prolonged, the tooth needs to be extracted to prevent space being lost (Kurol, 2002).

Tooth Size/Arch Length Discrepancy and Crowding

Early loss of primary teeth due to caries, infection, trauma, ectopic eruption, etc. can lead to reduced arch length and delayed eruption of permanent teeth (Brothwell, 1997).

In a study examining the relationship between the formation and eruption of maxillary teeth and the skeletal structure of the maxilla, it was observed that insufficient arch length delayed the eruption of the maxillary second molar, but did not cause any delay in the formation of the teeth (Suda, Hiyama, & Kuroda, 2002).

Although insufficient arch length causes delayed eruption of teeth, it often causes ectopic eruption of teeth (Suda et al., 2002)

Ectopic Eruption

Ectopic eruption of permanent first molars is caused by an abnormal mesioangular eruption pathway. This is associated with reduced maxillary transverse and sagittal space (Barberia-Leache, Suarez-Clúa, & Saavedra-Ontiveros, 2005; Yaseen, Naik, & Uloopi, 2011). Ectopic eruption is rarely seen in permanent second molars (Hwang, Choi, Lee, Chung, & Kim, 2017).

Ectopic eruptions of teeth can be diagnosed by radiographs in early mixed dentition. Studies have shown that 71% of these teeth straighten on their own by the age of nine (Dabbagh, Sigal, Tompson, Titley, & Andrews, 2017). In some cases, however, early intervention is needed to prevent the premature loss of primary second molars and the space restriction that can occur (Barberia-Leache et al., 2005; Yaseen et al., 2011).

Maxillary permanent canines remain impacted in 1-3% of the population. This is due to the fact that canines are the last teeth to erupt in the mouth, and the impaction caused by the horizontal eruption path (Ericson & Kurol, 1987).

Ectopic eruption of the maxillary permanent incisor usually occurs palatally and often causes an anterior crossbite. The mandibular permanent incisor is more likely to erupt from the lingual side (Kotsanos, Sarnat, & Park, 2022).

X-rays

In addition to abnormalities in tooth size, number, crown-root structure and relationship, and hard tissue mineralisation, children exposed to radiation also had delayed tooth eruption (Aguiar, Jham, Magalhães, Sensi, & Freire, 2009). Periodontal cell damage and inadequate mandibular growth at the site of x-ray exposure also appear to be associated with x-ray induced tooth eruption disorders (Piloni & Ubios, 1996).

Primary Eruption Disorder

Primary eruption disorder is a condition characterised by the failure of teeth to erupt in the absence of any physical obstacle or syndromic condition that would prevent the teeth from erupting (Proffit & Vig, 1981).

Although primary eruption disorder is most commonly seen in permanent teeth, cases have been reported in the primary dentition. In primary eruption disorder, at least one permanent molar is affected and the teeth distal to the affected tooth cannot erupt (Ahmad, Bister, & Cobourne, 2006; Hanisch, Hanisch, Kleinheinz, & Jung, 2018).

Primary eruption disorder is a condition often confused with ankylosis or infraocclusion (Pilz et al., 2014). The characteristic features of this condition are an open bite in the posterior teeth with normal vertical growth and an ankylosis when the teeth are moved orthodontically (Frazier-Bowers, Long, & Tucker, 2016).

Systemic Factors

Endocrine Disorders

Problems in the endocrine system have an effect on the development of the tooth structure as well as on the development of the whole body.

Hypothyroidism, hypopituitarism, hypoparathyroidism and pseudohypoparathyroidism are the most common endocrine disorders associated with delayed tooth eruption. Reduced levels of these hormones inhibit the formation of osteoclasts and delay bone resorption (Choukroune, 2017).

Hypothyroidism leads to cretinism (congenital hypothyroidism) in children. Dentofacial changes in cretinism vary according to the degree of thyroid deficiency (Shaw & Foster, 1989).

Hypopituitarism is a condition in which there is a delay in the eruption of teeth and in the growth of the body in general. It has been reported that in patients with hypopituitarism, the dental arch is smaller than normal and the roots of the teeth are shorter than normal (Kjellberg, Beiring, & Wikland, 2000; Shaw & Foster, 1989).

Preterm Birth/Low Birth Weight

Studies have shown that dental growth and development is delayed and teeth erupt later in preterm and low birth weight babies (Seow, 1997).

Genetic Disorders

Delayed eruption of teeth can be seen as a finding associated with many diseases and syndromes (Suri et al., 2004). Some of these include;

- Amelogenesis imperfecta
- Apert syndrome
- Cherubism
- Cleidocranial dysplasia,
- Dentin dysplasia
- Down syndrome

- Ectodermal dysplasia
- Gardner syndrome

Medicines

Delayed eruption can be caused by some long-term medicines (aspirin, paracetamol, ibuprofen, bisphosphonates). Non-steroidal anti-inflammatory drugs reduce bone resorption by blocking prostaglandin E2 synthesis. This causes a delay in the eruption of teeth (Charan et al., 2022).

HIV Infections

Studies have suggested a link between human immunodeficiency virus (HIV) infection and delayed tooth eruption. In a study by Hauk et al. of 70 perinatally HIV-infected children, delayed tooth eruption was directly related to clinical symptoms (Hauk, Moss, Weinberg, & Berkowitz, 2001).

Cerebral Palsy

It has been observed that unerupted primary and permanent teeth are common in children with cerebral palsy, especially permanent first molars, which erupt significantly later (Pope & Curzon, 1991).

Chronic Kidney Failure

Several cross-sectional studies in patients with chronic renal failure have found a delay in tooth eruption of up to 9 months compared with normal tooth growth curves (Velan & Sheller, 2021).

Conclusion

Eruption of teeth is a physiological process. Many systemic, local and genetic processes are involved. The dysfunction that can develop in relation to these factors causes eruption disorders. Eruption disorders affect the development of both the dentition and

the craniofacial structures. Therefore, early recognition and correct intervention are very important.

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ETIOLOGICAL FACTORS OF UNERUPTED TEETH

MUHAMMET YASİN PEKTAŞ[1]

Introduction

Health-related quality of life refers to how a person views their illness and the outcomes of treatment, along with their personal concerns(Niemann, Ingleshwar, & Paulson, 2024). Oral and dental health-related quality of life specifically looks at how pain or discomfort in the mouth and face impacts psychological, social, functional, and overall well-being. Key factors that enhance oral and dental health-related quality of life include the proper eruption and alignment of teeth in the mouth. Any form of tooth impaction can negatively influence a person's quality of life(Kaczor-Urbanowicz, Zadurska, & Czochrowska, 2016a; Niemann et al., 2024).

Normal biological eruption time is defined as the eruption that occurs after 2/3 of the tooth root has formed, and if the tooth has not erupted even though 2/3 or more of the root has formed, it is considered as late eruption(Marks & Schroeder, 1996). The factors affecting the impaction of deciduous teeth and permanent teeth are

[1] Asst. Prof., Kütahya Health Science University Faculty of Dentistry, Department of Oral and Maxillofacial Surgery, Orcid: 0000-0003-4508-946X

divided into two groups: local and general factors(Becker & Chaushu, 2015).

Local factors affecting impaction of teeth

Local factors include space limitations, abnormalities in the direction and position of the tooth germ, premature loss of primary teeth, traumatic injuries and dental ankylosis, supernumerary teeth, fibrotic mucosa and eruption cysts, odontogenic cysts and tumors, chronic inflammation of the surrounding mucosa- bone and neoplastic formations(Bello, Adeyemo, Bamgbose, Obi, & Adeyinka, 2011; Sajnani, 2015).

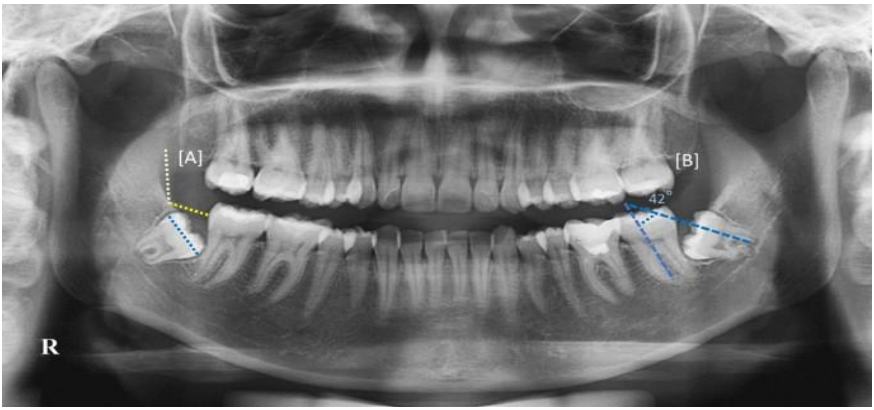
Space limitations

Teeth often become impacted when the alveolar bone arch length is too small to accommodate the total dental arch size, leading to insufficient space(Seehra et al., 2023). The most frequently impacted teeth are the lower and upper third molars, followed by the upper canines and lower premolars. Third molars are the most commonly impacted because they are the last to erupt, often lacking sufficient space(Idris et al., 2021).

In the anterior maxilla, canines tend to remain impacted due to crowding by adjacent teeth. Canines typically erupt after the maxillary lateral incisor and the first premolar; if space is insufficient, they fail to erupt properly. A similar situation occurs with mandibular premolars, as they erupt after the first molar and canine. If space is inadequate, the second premolar is most commonly affected(Seehra et al., 2023).

Physical obstruction is a frequent local cause of delayed eruption. Factors such as mucosal barriers, scar tissue, cysts, and tumors can contribute to the issue. Additionally, gingival hyperplasia from various causes may create a mucosal barrier that prevents proper tooth eruption(Idris et al., 2021; Seehra et al., 2023).

Figure 1: Third molar teeth that cannot erupt due to lack of space



Ref:(Alsaegh, Abushweme, Ahmed, & Ahmed, 2022)

Abnormalities in the direction and position of the tooth germ

Direction and position anomalies of tooth germs are among the main reasons why teeth remain impacted. Abnormalities in the direction and position of the tooth germis assessed in three anatomical planes(Haddaji Mastouri et al., 2016; Kaczor-Urbanowicz, Zadurska, & Czochrowska, 2016b):

- **Sagittal plane**: Includes mesial or distal displacement of molars, vestibular or oral displacement of frontal teeth, and transposition.
- **Vertical plane**: Involves supraposition or infraposition, as well as tooth rotation around its longitudinal axis.
- **Horizontal plane**: Refers to the medial and lateral displacement of anterior teeth, as well as vestibular and oral lateral deviations.

Etiological factors that cause teeth to be placed in abnormal positions include supernumerary teeth, pathology of follicle formation, mouth breathing, macrodentia, bad habits, swallowing

pathology, jaw sizes are not proportional, pathology of follicle formation, early extraction of baby teeth, hyperdontia, mesial-distal location of chewing teeth, pathology of speech and swallowing(Allareddy, Caplin, Markiewicz, & Meara, 2020; Esposito, 2005).

Premature loss of primary teeth

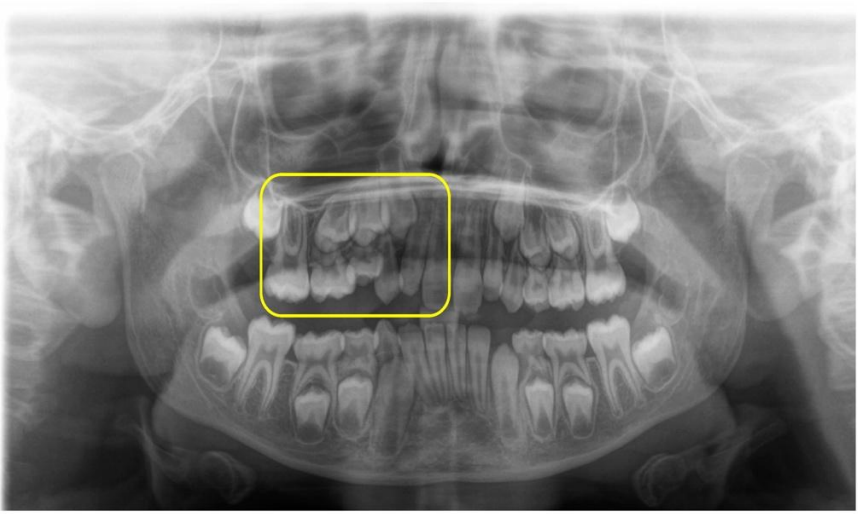
Early tooth loss or premature exfoliation, refers to the loss of teeth before their expected time of shedding. This condition can result from both local and systemic factors(Spodzieja & Olczak-Kowalczyk, 2022). Common local causes include dental trauma, neonatal tooth extraction, early childhood caries, and periodontal issues, while systemic diseases can also contribute. Although dental caries and trauma are the most frequent causes, diagnosing other underlying factors can be challenging due to limited clinical experience(Nadelman, Magno, Pıthon, Castro, & Maıa, 2021). Premature loss of primary teeth can lead to orthodontic complications such as crowding, ectopic eruption, or tooth impaction, ultimately contributing to malocclusion(Nadelman et al., 2021; Spodzieja & Olczak-Kowalczyk, 2022).

Traumatic injuries and dental ankylosis

Traumatic injuries and dental ankylosis is a fusion anomaly resulting from damage to the periodontal ligament, leading to the direct attachment of the tooth root's cementum to the alveolar bone(Zaleckiene, Peciuliene, Brukiene, & Drukteinis, 2014). It typically presents with mild to moderate progressive infraocclusion and occlusal plane inclination. While its exact cause remains unclear, factors such as dental trauma, genetic predisposition, metabolic disorders, or local deficiencies in vertical bone growth are believed to contribute to its development(Moccelini et al., 2022). Ankylosis in primary molars can significantly affect occlusion, leading to abnormal proximal contacts, food impaction, and an increased risk

of dental caries. Additionally, it can compromise both dental and periodontal health. In cases of severe infraocclusion, ankylosed primary molars may cause tilting of adjacent teeth, supraeruption of opposing teeth, occlusal disturbances, arch length reduction, and even impaction or delayed eruption of the succeeding permanent tooth(Moccelini et al., 2022).

Figure 2: Premolars that cannot erupt due to ankylosing of deciduous teeth



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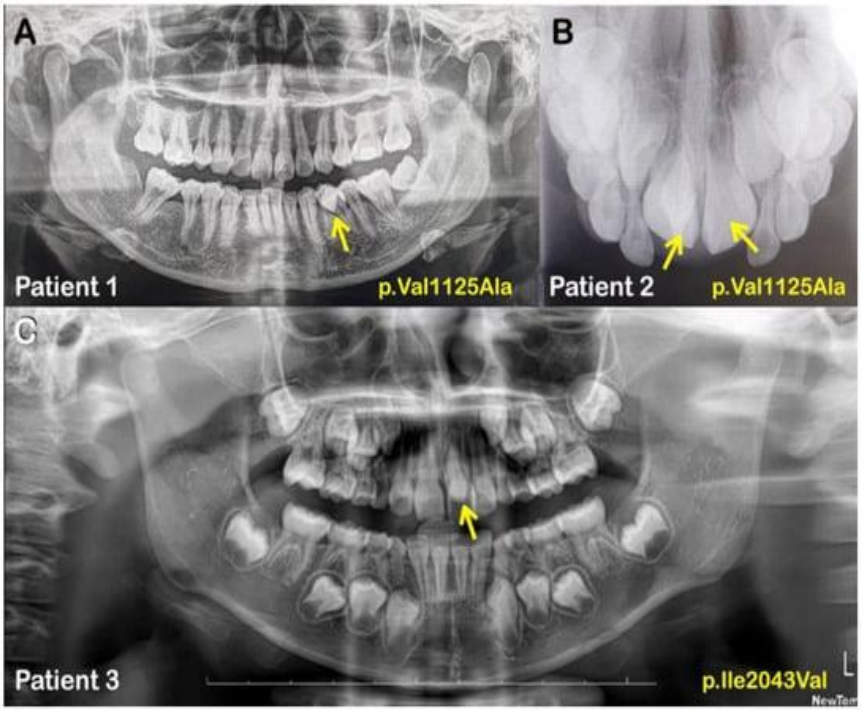
Supernumerary teeth

Extra teeth are often found in permanent dentition and are twice as prevalent in males. The most common type is mesiodens(‘Main genetic entities associated with supernumerary teeth’, 2018). These additional teeth can lead to impaction, displacement, and delayed eruption of nearby teeth. Research indicates that they contribute to space limitations, crowding in the dental arch, gaps between teeth, tooth rotation, delayed eruption of permanent teeth, and forward thrusting(Garvey, Barry, & Blake,

1999; ‘Main genetic entities associated with supernumerary teeth’, 2018).

Figure 3: Supernumerary teeth that prevent permanent teeth from erupting



Ref:(Panyarat et al., 2023)

Fibrotic mucosa and eruption cysts

A common local cause of delayed eruption is physical obstruction. Factors such as mucosal barriers, scar tissue, cysts, and tumors have been identified as contributing to this issue. Additionally, gingival hyperplasia from various causes can form a mucosal barrier that hinders tooth eruption(Nahajowski et al., 2021). Changes in the mucosa over an erupting tooth can also contribute to delayed eruption. The most common findings in young children are

fibrotic mucosa and eruption cysts(Sen-Tunc, Acikel, Saroglu-Sonmez, Bayrak, & Tuloglu, 2017).

Since these typically delay eruption by only a few weeks, treatment is generally unnecessary unless they cause significant discomfort or become pathological(Nahajowski et al., 2021; Sen-Tunc et al., 2017).

Eruption cysts are benign lesions that develop in the oral mucosa just before a tooth emerges. Clinically, they present as fluctuant, well-defined, and usually transparent swellings over the erupting tooth on the alveolar crest. When they contain blood, they appear purple or blue, a condition known as an eruption hematoma. Under transillumination, eruption cysts exhibit brightness, whereas eruption hematomas do not(Nahajowski et al., 2021; Sen-Tunc et al., 2017).

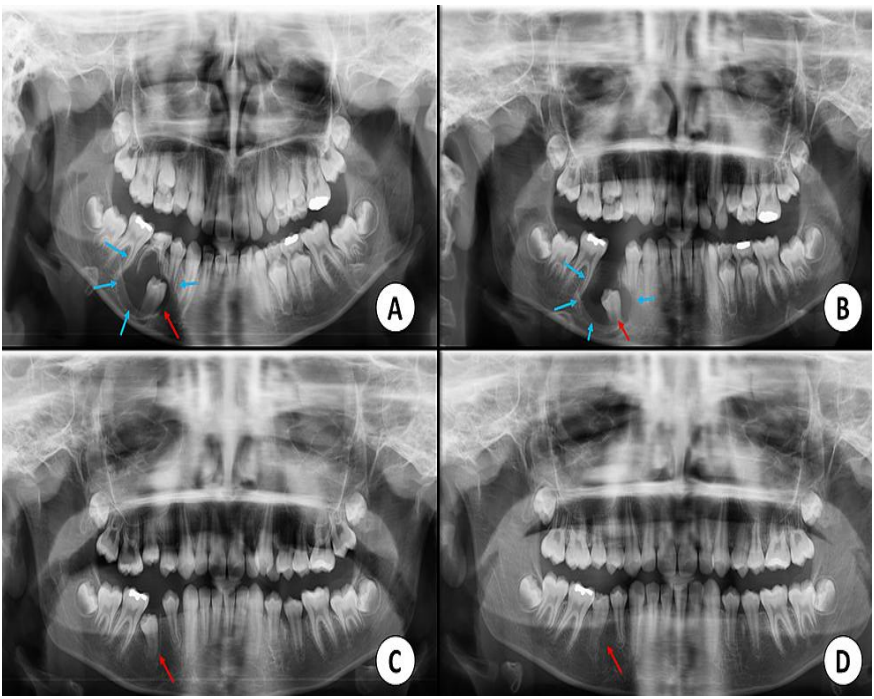
Radiographically, eruption cysts appear as a crescent-shaped radiolucency over the crown of an unerupted tooth. Although their exact cause is not fully understood, factors such as trauma, early caries, infection, and insufficient space have been linked to their development(Nahajowski et al., 2021; Sen-Tunc et al., 2017).

Odontogenic cysts and tumors

The literature generally estimates the prevalence of erupted and impacted third molars in individuals of varying ages to be between 6% and 14%(Rajae & Karima, 2021; Rajendra Santosh & Ogle, 2020). However, there is a low reported correlation between impacted teeth and associated cysts or tumors. This discrepancy may stem from the fact that enucleated tissues are often discarded after surgical removal rather than being sent for histopathological examination, leading to undiagnosed pathologies. Among cystic lesions and tumors linked to impacted teeth, dentigerous cysts have

the highest reported incidence, accounting for approximately 70% to 100% of cases.

Figure 4: Radiograph view demonstrating the dentigerous cyst (blue arrows) related to the unerupted right second premolar (red arrow)



Ref:(Alnofaie, Alomran, Ababtain, & Alomar, 2019)

In contrast, the occurrence of periodontal pathogens contributing to second molar caries, as well as conditions such as ameloblastoma, odontoma, odontogenic keratocyst (OKC), paradental cyst, and fibrosarcoma, is significantly lower(Mello, Melo, Kammer, Speight, & Rivero, 2019; Rajae & Karima, 2021; Rajendra Santosh & Ogle, 2020). The most common odontogenic cysts and tumors are summarized in Table 1.

Table 1. List of common odontogenic cysts and tumors

Calcifying Odontogenic Cyst
Adenomatoid Odontogenic Tumor
Dentigerous Cyst
Odontogenic Keratocyst
Ameloblastoma& Variants
Calcifying Epithelial Odontogenic Tumor
Odontogenic Fibroma &Mixoma
Lateral Periodontal Cyst
Odontoma

Ref: (Mello et al., 2019)

Chronic inflammation of the surrounding mucosa- bone

Teeth are unique in that they provide a direct route for infection to spread to the surrounding bone and soft tissue structures(Hajishengallis & Chavakis, 2021). For this reason, infections of teeth and surrounding tissues (Periodontal diseases, inflammations due to dental caries, periapical diseases, perio-endo lesions, pericoronitis, infections originating from the maxillary sinus) can spread rapidly and become chronic in the area they spread. As a result of this chronicity, the teeth surrounding the inflammation may remain impacted(Hajishengallis & Chavakis, 2021).

General factors affecting impaction of teeth

General factors encompass genetics, vitamin D deficiency, congenital factors and congenital syphilis, down syndrome, anemia,

endocrine disorders, HIV infections(Çakıroğlu Erbay & Öztürk, 2013).

Genetics

Several genetic diseases are associated with delayed tooth eruption. Syndromes characterized by multiple tumors and cysts, such as Gorlin syndrome, Cherubism, and Gardner syndrome, have been linked to delayed eruption(Çakıroğlu Erbay & Öztürk, 2013). Additionally, conditions like Apert syndrome, Cleidocranial dysostosis, and Gardner syndrome, which are often accompanied by supernumerary teeth, can contribute to delayed eruption. Increased bone density in disorders such as osteopetrosis, sclerosteosis, Carpenter syndrome, Apert syndrome, Cleidocranial dysplasia, and pyknodysostosis can hinder bone resorption, further delaying tooth eruption(Jensen & Kreiborg, 1990; Kaloust, Ishii, & Vargervik, 1997; Stephen, Hamersma, Gardner, & Beighton, 2001).

Vitamin D deficiency

Vitamin D is an essential nutrient that plays a crucial role in maintaining dental and gum health. Sufficient vitamin D intake is vital for the protection and development of healthy teeth and gums(Çakıroğlu Erbay & Öztürk, 2013; Fulton, Amlani, & Parekh, 2020). Deficiencies in vitamin D metabolites can lead to eruption path disorders, resulting in impacted teeth. Additionally, jawbone demineralization, loss of trabeculae and lamina dura, giant cell lesions, and metastatic calcifications may also occur(Fulton et al., 2020).

Congenital factors and congenital syphilis

These are non-genetic factors that occur before or during birth. Not all congenital disorders are hereditary. Congenital factors include certain maternal diseases during pregnancy and medications

taken by the mother, which can impact the fetus's dental development(Thean, Moore, & Nourse, 2022).

Syphilis is an infectious disease caused by *Treponema pallidum* and can become highly chronic if left untreated(Fang, Partridge, Bautista, & Sankaran, 2022). It can affect nearly all skin and internal organs. The primary mode of transmission is through sexual contact, known as direct transmission. Indirect transmission can occur from an infected mother to her child during pregnancy, through medical examinations by doctors and nurses, via blood transfusions, and through contaminated environments such as poorly maintained baths, pools, and saunas, as well as shared items like towels(Pascoal et al., 2023). If a mother with syphilis does not receive treatment, the infection can be transmitted to the baby, leading to congenital syphilis. Late congenital syphilis symptoms emerge after the age of two and can include Hutchinson teeth, hearing impairment, intellectual disability, and a saddle-shaped nose. These patients also frequently experience tooth eruption disorders and impacted teeth(Fang et al., 2022; Pascoal et al., 2023; Thean et al., 2022).

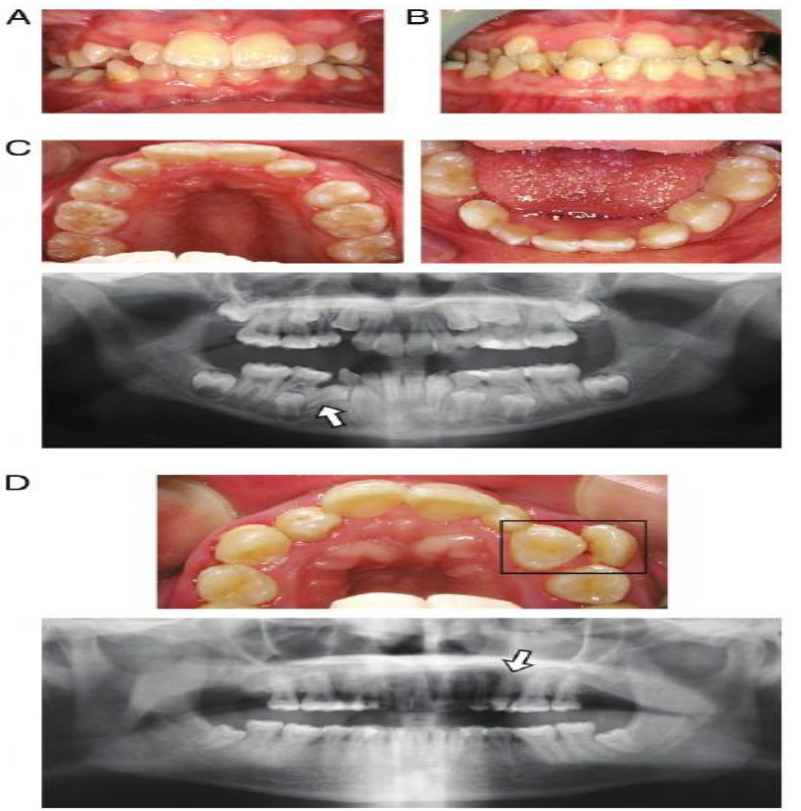
Down syndrome

Although the genetic factor is emphasized in the development of this disease, first described by Langdon-Down in 1866, determining its exact cause remains challenging. While heredity plays a role, environmental factors are considered the primary contributors(AlJameel, Watt, Tsakos, & Daly, 2020).

Common jaw findings include spacing between the incisors, maxillary endognathia, and lower proalveoli due to tongue positioning. Delayed tooth eruption is a notable feature. The emergence of primary teeth is significantly delayed, with the first tooth appearing anywhere between 8 months and 4 years. The eruption sequence may also differ, with primary molars sometimes

emerging before incisors(Mayoral-Trias, Llopis-Perez, & Puigdollers Pérez, 2016). A similar pattern is observed in permanent teeth, where the first molar, typically a 6-year-old tooth, may not erupt until 9 years of age, and the 12-year-old molar may not appear until 16-20 years of age(AlJameel et al., 2020; Mayoral-Trias et al., 2016).

Figure 5: Malalignment and tooth impaction in Down Syndrome subjects. A. Malaligned mixed dentition; B. Malaligned permanent dentition; C. Impacted lower right first premolar (arrow). Subject also has missing lower left lateral incisor; D. Transpositioned upper left canine and first premolar (box and arrow)



Ref:(H.W., K.Y., & Keung, 2011)

Anemia

Anemia is a condition in which erythrocyte mass and serum hemoglobin (Hb) levels fall below the age- and gender-specific values established by the World Health Organization. In patients with anemia, dental and skeletal manifestations may include delayed tooth eruption, enamel hypoplasia, asymptomatic pulp necrosis, pulp stones, trabecular changes in the bone, and osteomyelitis(Piccin et al., 2019).

Studies have reported that tooth eruption in individuals with sickle cell anemia occurs later than in healthy individuals. In sickle cell anemia, the lifespan of erythroblasts is significantly reduced from 120 days to 10-30 days, leading to increased erythroblast production and bone marrow hyperplasia(Rumeli Atıcı, 2020). As a result, trabecular changes, jawbone expansion, and an increase in bone marrow space can be observed. Alterations in craniofacial bone structure have been associated with malocclusion. Additionally, delayed tooth eruption and advanced periodontitis are common in patients with sickle cell anemia, both of which may contribute to malocclusion. Consequently, a potential relationship between sickle cell anemia and malocclusion has been suggested in the literature(Piccin et al., 2019; Rumeli Atıcı, 2020).

Endocrine disorders

Endocrine disorders can lead to delayed eruption of both primary and permanent teeth. The most common endocrine conditions associated with delayed tooth eruption include hypothyroidism, hypopituitarism, hypoparathyroidism, and pseudohypoparathyroidism(Loevy, Aduss, & Rosenthal, 1987).

HIV infections

A study investigating the relationship between HIV infection and delayed tooth eruption found that 70 perinatally infected

children exhibited delayed eruption. Other studies have suggested that this delay is associated with the low socioeconomic status of HIV-infected children, as well as their nutritional and overall health challenges(Alsaegh et al., 2022).

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HYPERPLASTIC DENTAL FOLLICLE

ÖZGÜR DOĞAN[1]
NİLAY YILMAZ[2]
HATİCE HATİPOĞLU[3]

Introduction

Dental follicle is radiographically characterized by a radiolucent area surrounding the crown of an unerupted tooth, which plays a key role in the development and eruption of teeth.(Honda et al., 2010) Hyperplastic dental follicle (HDF) has been described as odontogenic hamartomatous lesion that occurs in pericoronal tissues of the unerupted tooth, tooth eruption failure in young patients. (Gardner, 1980; Philipsen et al., 1992) The occurrence of this pericoronal dental lesion seems to be more frequent than 3 the literature has reported. It involves mostly permanent first and second molars.(Rajabi-moghaddam et al., 2022) Although its occurrence

[1] Doç. Dr. Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı, ORCID: 0000-0002-5793-175X
[2] Arş. Gör. Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı, ORCID: 0009-0001-3389-2223
[3] Arş. Gör. Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı, ORCID: 0009-0000-9419-0455

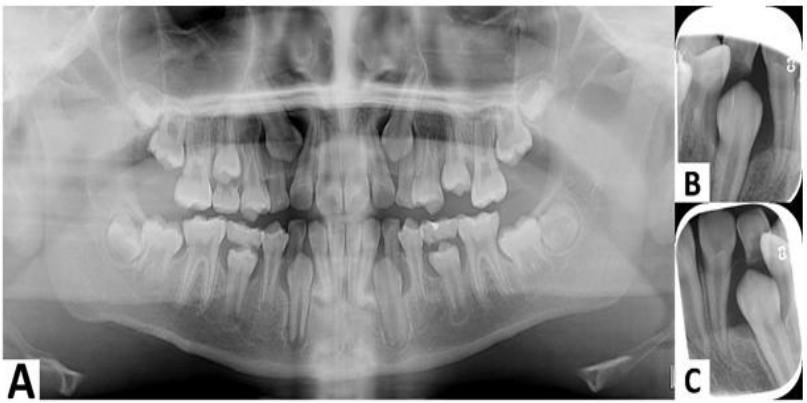
can be found in any age, most cases in the literature affect young individuals.(O'Connell et al., 2014)

Since 1980, when Gardner(Gardner, 1980) first described the hyperplastic dental follicle, there are difficulties regarding the pathological differentiation of this hamartomatous lesion with other odontogenic tumors, particularly with central odontogenic fibroma. Both lesions present similar clinical and histopathological characteristics and the distinction may be challenging (Gardner, 1980; L. Barnes, J. W. Everson, P. Reichart, 2005; Sandler et al., 1988) It has been considered that hamartomatous lesion such as hyperplastic dental follicle presents a less aggressive behavior than tumors as odontogenic fibroma and that the clinical outcome may be an important tool for diagnosis(L. Barnes, J. W. Everson, P. Reichart, 2005; Nikitakis et al., 2006).

The radiographic appearance of hyperplastic dental follicle is characterized by well-circumscribed radiolucent area with sclerotic borders surrounding the crown of an unerupted tooth, frequently mimicking dentigerous cyst. (Figure 1A-C, Figure 2 A&B) (van Heerden et al., 1990). Delayed or tooth eruption failure has been associated with this hamartomatous lesion. Microscopically, the hyperplastic dental follicle consists of fibrous connective tissue containing odontogenic epithelium, multinucleated giant cells, and calcification foci.(Nikitakis et al., 2006; Yonemochi et al., 1998) Recently, some authors described the occurrence of multiple calcifying hyperplastic dental follicles associated with multiple unerupted teeth affecting young male patients and they suggested that this condition should be considered a distinct pathology.(Cho et al., 2011; Jamshidi et al., 2013) The occurrence of hamartomas from odontogenic origin seems to be more frequent than that which has been reported in the literature.(L. Barnes, J. W. Everson, P. Reichart, 2005) Furthermore, the diagnosis of hyperplastic dental follicle is important in order to distinguish this condition from other

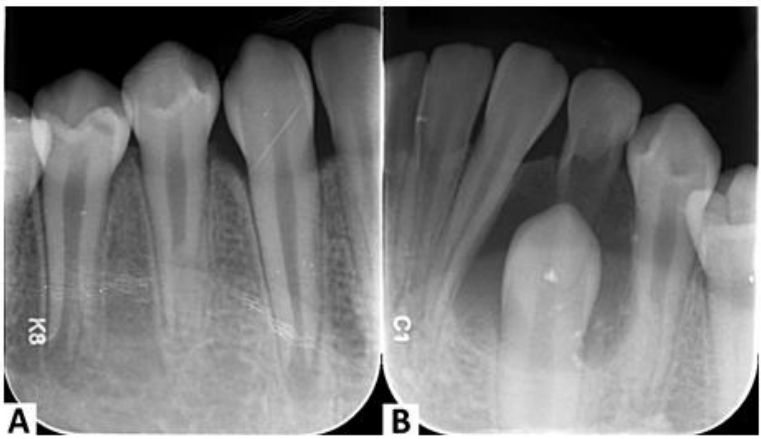
odontogenic tumors that present different pathogenesis and recurrence potential.(Nikitakis et al., 2006)

Figure 1A-C: Routine panoramic radiograph (A) and periapical radiograph of right and left mandibular canine region taken at age 8 demonstrating unilocular pericoronal radiolucent lesions surrounding all permanent canines and lower left second premolar indicating multiple hyperplastic dental follicles affecting canines and one premolar.



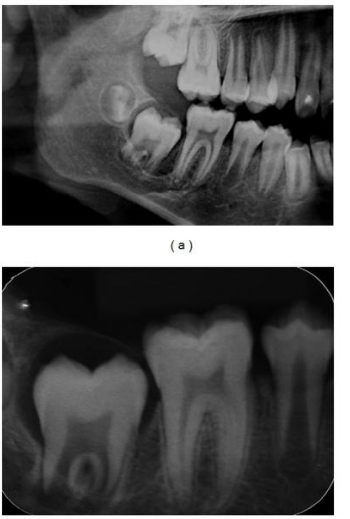
Reference: (Ghods, 2022)

Figure 2A & B: Periapical radiograph of right (A) and left (B) canine-premolar region demonstrating resolution of right canine and left second premolar while lower left canine demonstrated unilocular radiolucency with sclerotic borders and evidence of resorption of lower left primary canine and mild displacement of roots of lower left lateral incisor and first premolar.



Reference: (Ghods, 2022)

Radiographic aspect of the unerupted teeth. (a) Sectioned panoramic radiography: radiolucent well-defined area surrounding the crown of tooth 47, extending to apical region in the anterior area. (b) Periapical radiography: delicate sclerotic border, normal enamel and radicular formation, and absence of visible calcifications in pericoronal space.



Reference:(Schmitd et al., 2014)

Etiology

It was reported that HDF may have a familial tendency thereby suggesting a genetic predilection.(Sun et al., 2010) While the HDF is associated with an unerupted tooth, the causes of which are uncertain, but have been attributed to endocrinal disturbances, metabolic disease, muscle pressure, febrile disease, inflammation, and vitamin D deficiency.(Bishara et al., 1976)

HDF is often associated with syndromes such as Gardner syndrome(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020), Lowe syndrome(Roberts et al., 1994), amelogenesis imperfecta(O'Connell et al., 2014; Roquebert et al.,

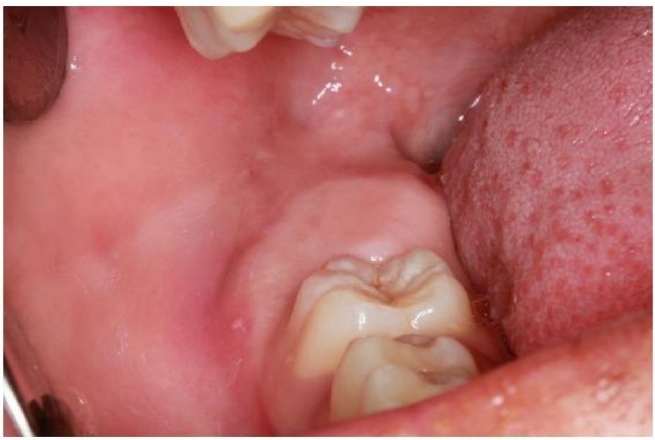
2008; van Heerden et al., 1990) enamel renal syndrome(de la Dure-Molla et al., 2014), enamel dysplasia(Feller et al., 2006), cleidocranial dysplasia(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020), Noonan syndrome(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020), and mucopolysacaridosis.(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020) Non-syndromic HDF is rarely reported so the true incidence is unknown. Except for third molar involvement, multiple non-syndromic HDFs appear to be very rare in a young child.(Sun et al., 2010) HDF most frequently affects posterior teeth (molars or premolars) while canines and incisors are less frequently involved.(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020) Therefore, non-syndromic HDF affecting multiple canines in a pediatric patient is rarely found in the literature. The occurrence of HDF in maxilla and mandible is comparable.(Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de Los Santos, et al., 2020)

Kim et al reported that the expression of several matrix metalloproteinases is down-regulated in hyperplastic dental follicles; moreover, several collagen genes are up-regulated. It was demonstrated that several collagen genes (Col I, IV, VIII, and XI and TIMP 1,2, and 4) were two-fold upregulated in the HDF, while the expression of matrix metalloproteinases (1, 2, 10, and 16) and interleukin 8 were two-fold downregulated Hence, the defective regulation of matrix metalloproteinases mediating connective tissue remodeling may be the underlying cause for fibrous hyperplasia leading to the abnormal tooth eruption, and resultant HDF.(Kim et al., 2008)

Clinical Features

Hyperplastic dental follicle (HDF) is typically asymptomatic and may present with slight enlargement in the affected area. (Figure 3) (Fukuta, Totsuka, Takeda, & Yamamoto, 1991).

Figure 3. Enlargement of gingival area associated with unerupted right mandibular second molar.



Reference: (Schmitd et al., 2014)

Radiographic Features

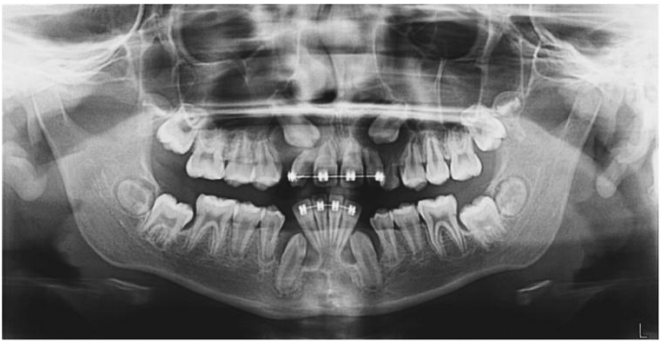
On radiographic evaluation, the typical pericoronal follicular space measures between 2 to 3 mm in width. Mesgarzadeh, Esmailzadeh, Abdolrahimi, & Shahamfar, 2008) Radiographically, a hyperplastic dental follicle presents as a well-defined radiolucent area with sclerotic margins encircling the crown of an unerupted tooth, often resembling a dentigerous cyst. This hamartomatous lesion has been linked to delayed tooth eruption or eruption failure. (van Heerden et al., 1990; Walker et al., 2004)

While a radiolucent area of up to 5 mm around the crown of an unerupted tooth is highly indicative of a dentigerous cyst or other

odontogenic tumors, hyperplastic dental follicle should also be considered in the differential diagnosis. (Gardner, 1980a; Gomez et al., 1998; Hirschberg et al., 1996)

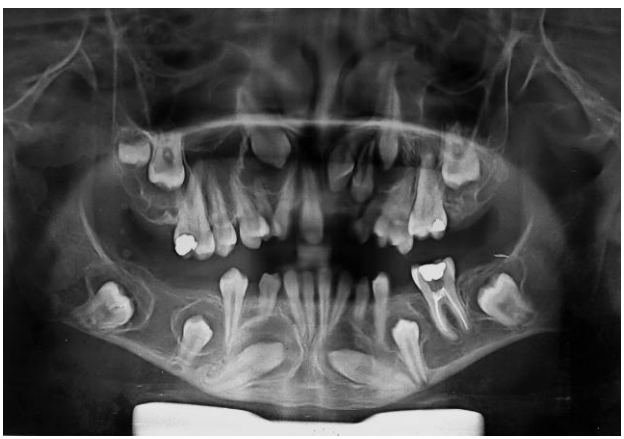
A rare variant of hyperplastic dental follicle, known as multiple calcifying hyperplastic dental follicle (MCHDF), was first described by Sandler et al. It is characterized by extensive calcifications and remnants of odontogenic epithelium.(Figure 4, Figure 5)(Jamshidi et al., 2013; Sandler et al., 1988) In differential diagnosis, this lesion may be confused with central odontogenic fibroma due to the shared histological features, including connective tissue, odontogenic epithelial remnants, and calcifications.(Jamshidi et al., 2013) Given these overlapping features, an accurate diagnosis should rely on a combination of clinical, radiographic, and histological assessments. Interestingly, reports indicate that multiple calcifying hyperplastic dental follicle exhibits a male predominance and is most commonly observed in the mandible. (Jamshidi et al., 2013; Lukinmaa et al., 1990)

Figure 4. Panoramic radiograph showing pericoronal radiolucencies affecting the four canines. Distal inclination of mandibular incisors and radicular resorption of the maxillary lateral incisors is also present. Several teeth show apparently short roots



Reference: (Guardado-Luevanos, Haro, Godínez-Rubí, Puente-de los Santos, et al., 2020)

Figure 5. Panoramic radiograph showing large pericoronal radiolucencies associated with multiple unerupted teeth.

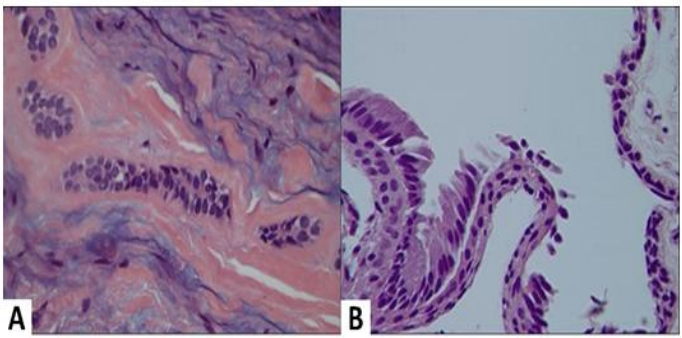


Reference: (Gardner & Radden, 1995)

Histopathology

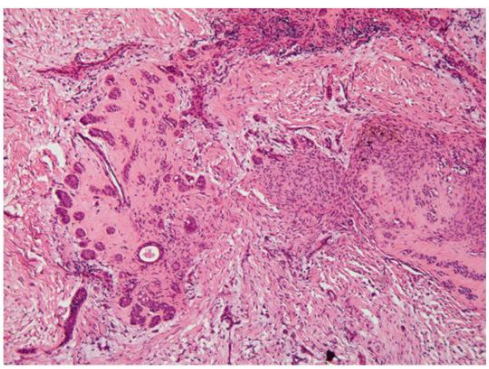
Microscopically, HDF lesions show fragments loosely or densely arranged connective tissue containing odontogenic epithelial rests scattered among the delicate or mature collagen fibrils with or without myxomatous change and inflammation. (Figure 6 A&B) Due to these histopathologic findings, HDF can often be confused with odontogenic fibroma, myxoma, myxofibroma and ameloblastic fibroma. (Figure 7) (Chrcanovic et al., 2014; Robinson, 2017) Within the fibrous connective tissue, epithelial components in HDF have been reported to be of three types: reduced enamel epithelium, remnants of dental lamina, and scattered odontogenic epithelial rests. Epithelial cells in reduced enamel epithelium are columnar or cuboidal with pyknotic nuclei and eosinophilic cytoplasm. Cells of the remnants of dental lamina are round in shape with round nuclei and pale cytoplasm. Scattered odontogenic epithelial rests have been reported in the majority of the lesions with vacuolar degeneration and occasional keratinization.(FUKUTA et al., 1991) Microscopically, the calcified areas were seen with osteodentin, cementum, psammomatous calcification, or Liesegang ring-like structures.(Figure 8) (Cho et al., 2011; Jamshidi et al., 2013; Roquebert et al., 2008),(Schmitd et al., 2014),(Gomes et al., 2019)

Figure 6A & B: High-power (400X), hematoxylin-eosin-stained photomicrographs of the hyperplastic dental follicle. A: Exhibits fibromyxomatous connective tissue with hyalinized induction around the nests and strands of inactive odontogenic rests. B: Highlights the epithelial lining of reduced enamel epithelium, ranging from tall columnar ameloblastic cells to flattened cuboidal cells.



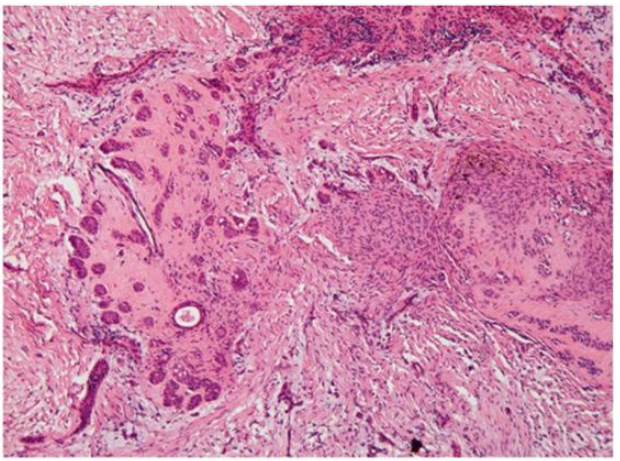
Reference: (Ghods, 2022)

Figure 7. In the fibrous tissue of a hyperplastic dental follicle, noduli resembling ameloblastic fibroma may be present. They should be considered tiny hamartomas of odontogenic tissue. Their nodular appearance allows distinction from ameloblastic fibroma that lacks any nodular architecture.



Reference: (S., 2015)

Figure 8. Hyperplastic dental follicle showing fibrous tissue containing islands of odontogenic epithelium partly surrounded by poorly mineralized collagenous matrix considered to represent dysplastic dentin because of the relationship with the epithelial nests.



Reference:(S., 2015)

Management

While HDF is generally considered a non-pathologic entity, radiographic monitoring is recommended to rule out more aggressive odontogenic lesions. Periodic follow-up imaging is particularly important if the follicular space continues to expand, as this could suggest pathological transformation. If an impacted tooth remains unerupted beyond the expected time frame, surgical intervention may be necessary to facilitate its eruption.(Kim et al., 2008)

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ERUPTION CYSTS

ESRA NUR AKGÜL[1]

Introduction

Eruption cysts (ECs) are benign, developmental odontogenic cysts that occur in the soft tissues overlying an erupting primary or permanent tooth (Aguiló et al., 1998). It is categorized within the dentigerous cyst group and considered a variant occurring in the soft tissues covering an erupting tooth by the World Health Organization (WHO) in its 2017 classification of odontogenic cysts (Soluk-Tekkesin & Wright, 2018).

ECs typically present as dome-shaped, translucent swellings on the alveolar ridge, often associated with the eruption of teeth. Although they are generally asymptomatic and self-limiting, they can occasionally cause discomfort, infection, or delay in tooth eruption, necessitating intervention (Gaddehosur et al., 2014). This chapter provides a detailed overview of etiology, prevalence, clinical, radiographic and histological features, differential diagnosis, and treatment of eruption cysts.

[1] Uzm.Dt. Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı, Orcid: 0000-0002-3320-1525

Etiology

The exact etiology of eruption cysts remains unclear, but several theories have been proposed. Some researchers attribute their development to degenerative cystic changes in the reduced enamel epithelium following the completion of amelogenesis, while others suggest that they arise from epithelial remnants of the dental lamina surrounding the erupting tooth (Radden & Reade, 1973; Toller, 1967). Local factors such as trauma, infection, early caries, and inadequate space for eruption have also been implicated in the development of ECs (Aguiló et al., 1998). Additionally, the use of certain medications, such as Cyclosporin A, has been associated with the formation of eruption cysts in some cases. Kuczek et al. reported a case of EC formation in a patient undergoing Cyclosporin A therapy, suggesting that systemic medications may influence the pathogenesis of these cysts (Kuczek et al., 2003). Other systemic conditions, such as Menkes kinky hair syndrome, have also been associated with the development of ECs. Nomura et al. reported a case of multiple ECs in a patient with Menkes syndrome, suggesting that genetic and metabolic factors may play a role in the pathogenesis of these lesions (Nomura et al., 1996).

Prevalence

Eruption cysts are predominantly observed in the pediatric population. Their occurrence aligns closely with the developmental stages of dentition, with most cases appearing during the eruption of primary or permanent teeth (Anderson, 1990). However, despite numerous case reports and retrospective studies, accurately determining the prevalence of ECs remains challenging.

Tekkesin et al. reported an incidence of 0.1% in a pediatric population, while Jones et al. and Manor et al. reported higher incidences of 1.8% and 21%, respectively (Jones & Franklin, 2006; Manor et al., 2012; Tekkesin et al., 2012). Variations in reported rates

are attributable to differences in study populations, diagnostic criteria, and the fact that many eruption cysts are asymptomatic and resolve spontaneously without ever being recorded. ECs are more commonly observed in the first decade of life, particularly between the ages of 6 and 9 years, coinciding with the eruption of permanent first molars and incisors (Aguiló et al., 1998; Anderson, 1990). Rare cases have also been documented in newborns (Bodner et al., 2005; Ramón Boj & García-Godoy, 2000). There is no clear gender predilection, although some studies suggest a slight male predominance (male: female ratio- 2:1) (Anderson, 1990; Bodner et al., 2005). Additionally, geographic and racial variations have been suggested, with certain reports indicating a higher incidence in Caucasian populations. Notably, the anatomical location of eruption cysts is often related to the tooth involved (Anderson, 1990; Bodner et al., 2005; Ramón Boj & García-Godoy, 2000). Most cases are reported in the first decade of life, coinciding with the eruption periods of primary and permanent teeth. Commonly, these lesions are found in the mandibular anterior region, particularly around the central incisors, although they are also frequently associated with permanent first molars and maxillary incisors (Bodner et al., 2005; Neville et al., 2015).

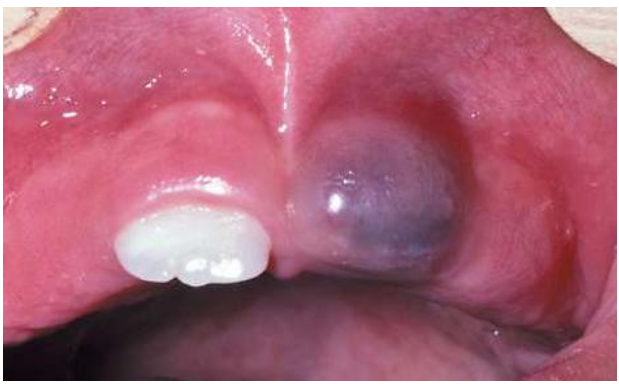
It is important to acknowledge that the true prevalence of eruption cysts may be underreported in clinical studies. Many lesions resolve without intervention or detection, leading to a potential underreporting in epidemiological surveys (Anderson, 1990; Ramón Boj & García-Godoy, 2000). Despite this, the clinical relevance of ECs is underscored by their potential to interfere with tooth eruption and by the occasional need for intervention when complications arise.

Clinical Features

Clinically, eruption cysts present as soft, fluctuant, dome-shaped swellings on the alveolar ridge, typically overlying the crown of an erupting tooth (Bodner et al., 2005). The color of the cyst can range from translucent to bluish or purple, depending on the presence of blood within the cystic cavity. When blood is present, the lesion is often referred to as an "eruption hematoma" (Neville et al., 2015).

ECs are usually asymptomatic but may become painful if infected or traumatized. They are most commonly associated with the eruption of mandibular molars and maxillary incisors (Bodner et al., 2005). The size of ECs can vary depending on the associated tooth, with cysts associated with permanent teeth generally being larger than those associated with primary teeth. The average diameter of ECs is approximately 0.6 cm, although larger cysts have been reported (Seward, 1973). ECs can occur unilaterally or bilaterally and may involve single or multiple teeth (Aguiló et al., 1998).

Figure 1: Intraoral view of eruption cyst



Reference: (Neville et al., 2015)

In some cases, ECs may be associated with natal or neonatal teeth. Natal teeth are present at birth, while neonatal teeth erupt within the first 30 days of life. The presence of ECs with natal or neonatal teeth is rare, with only a few cases reported in the literature (Bodner et al., 2005; Ricci et al., 2008). These cases often present unique challenges in terms of diagnosis and management, as the cysts may interfere with feeding or cause discomfort for the infant (Boras et al., 2007).

The clinical presentation of ECs can vary depending on the stage of the lesion. In the early stages, the cyst may appear as a small, translucent swelling on the alveolar ridge (Gaddehosur et al., 2014). As the cyst enlarges, it may become more fluctuant and take on a bluish or purple color due to the accumulation of blood within the cystic cavity. In some cases, the cyst may rupture spontaneously, leading to the drainage of cystic fluid and the exposure of the underlying tooth (Bodner et al., 2005).

Radiographic Features

Eruption cysts are soft tissue lesions and therefore do not typically show radiographic changes. However, in some cases, a radiolucent area may be observed over the crown of the erupting tooth, resembling a "half-moon" shape. Radiographs are primarily used to evaluate the position of the underlying tooth and to rule out other pathologies, such as dentigerous cysts or other odontogenic lesions (Anderson, 1990).

In cases where the cyst is large or symptomatic, radiographic examination may reveal a soft tissue shadow overlying the crown of the erupting tooth. However, there is usually no bone involvement, which helps differentiate ECs from dentigerous cysts. Radiographic evaluation is also important for assessing the morphology of the involved tooth and surrounding bone (Woldenberg et al., 2004).

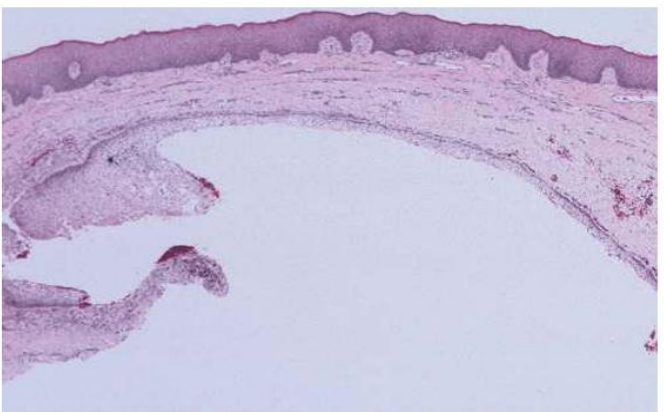
Histological Features

The histological features of ECs are not pathognomonic, and the diagnosis is often based on clinical and radiographic findings. However, histopathological examination may be necessary in cases where the clinical presentation is atypical or where other lesions are suspected.

Histologically, eruption cysts are lined by non-keratinized stratified squamous epithelium, similar to dentigerous cysts. The cystic wall consists of fibrous connective tissue, often with an inflammatory cell infiltrate in the lamina propria. The cystic cavity may contain fluid, blood, or both, depending on the presence of trauma or infection (Neville et al., 2015).

In some cases, the cystic lining may show areas of ulceration or inflammation, particularly if the cyst has been traumatized or infected. The underlying connective tissue may also show evidence of fibrosis, particularly in cases where the cyst has been present for an extended period of time (Neville et al., 2015).

Figure 2: Histologic appearance of eruption cyst



Reference: (Neville et al., 2015)

Differential Diagnosis

ECs must be distinguished from other oral lesions with similar clinical presentations. The differential diagnosis of eruption cysts includes several other soft tissue lesions, such as dentigerous cysts, hemangiomas, neonatal alveolar lymphangiomas, pyogenic granulomas, amalgam tattoos and Bohn nodules (Aguiló et al., 1998; Woldenberg et al., 2004).

Dentigerous cysts, unlike ECs, are associated with impacted teeth and involve bony changes that can be identified radiographically. Hemangiomas, which are vascular lesions, may be presented as bluish swellings. Transillumination can be a useful diagnostic tool, as eruption cysts typically glow under transillumination, whereas hematomas do not (Aguiló et al., 1998; Seward, 1973). Neonatal alveolar lymphangiomas, another possible differential diagnosis, are rare vascular anomalies that can resemble ECs in appearance but have distinct histological characteristics. Pyogenic granulomas present as erythematous, lobulated masses and are often associated with a history of trauma or irritation. Amalgam tattoos, caused by embedded amalgam particles, can appear as localized pigmentation, and while they may be mistaken for ECs, their etiology and clinical behavior are distinct (Aguiló et al., 1998; Seward, 1973). A thorough clinical examination, combined with radiographic and histopathological assessment, is essential for an accurate diagnosis.

Treatment

Most eruption cysts do not require treatment, as they often rupture spontaneously, allowing the underlying tooth to erupt normally. However, due to the lack of spontaneous regression, progressive enlargement, delayed tooth eruption, pain, and interference with feeding or aesthetic concerns, surgical intervention is required in eruption cysts (Hayes, 2000; Ricci et al., 2008).

In such cases, a simple incision or partial excision of the overlying tissue may be performed to expose the crown of the tooth and drain the cystic fluid, which is especially useful if the cyst is causing discomfort or delaying eruption. Marsupialization, the most commonly used surgical technique, involves excising the dome of the cyst to drain its contents and expose the crown, thereby facilitating normal tooth eruption; this procedure is usually performed under local anesthesia and carries a low risk of complications (Bodner et al., 2005; Boj et al., 2006). In certain situations, complete enucleation of the cyst may be required, and when the cyst is associated with a natal or neonatal tooth that is hypermobile or interferes with feeding, extraction of the tooth might be necessary. Additionally, laser therapy using Er,Cr:YSGG lasers has been employed as a minimally invasive alternative that can reduce bleeding, minimize postoperative pain, and promote faster healing, although its high cost and technical sensitivity may limit its widespread use (Boj et al., 2006).

A comprehensive understanding of the clinical, radiological, histological, and therapeutic aspects of eruption cysts is essential for clinicians managing these lesions. While eruption cysts are generally benign and self-resolving, a thorough knowledge of their clinical presentation, diagnostic evaluation, and management options enables dental professionals to provide accurate diagnoses and develop appropriate treatment plans. By integrating clinical experience with current research, practitioners can ensure effective management, minimize complications, and offer appropriate reassurance to patients and their caregivers, ultimately supporting the normal eruption and development of teeth.

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DENTIGEROUS CYST (FOLLICULAR CYST)

İSMAİL HAKTAN ÇELİK [1]

INTRODUCTION

Dentigerous cysts, also known as follicular cysts, are defined as developmental odontogenic cysts that attach to the cementoenamel junction of an unerupted tooth and surround its crown (Benn & Altini, 1996). They are the second most common type of odontogenic cysts in the jaws, accounting for approximately 20% of all odontogenic cysts and mandibular cysts (Wang & Olmo, 2021). Although their histogenesis is not fully understood, they are generally considered to originate from the reduced enamel epithelium. In its pathogenesis, the basic mechanism is suggested to be the accumulation of pathological fluid between the crown of the unerupted tooth and the reduced enamel epithelium, causing the expansion of the follicular tissue and, as a result, preventing the tooth from erupting. (Menditti & et al., 2018).

Although dentigerous cysts are usually of developmental origin, they may rarely develop in children with inflammatory origin as a result of chronic periapical inflammation caused by necrotic and

[1] Dr.Öğr.Üyesi, Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi , Çocuk Diş Hekimliği, Orcid: 0000-0003-1662-3790

infected deciduous teeth due to caries or trauma. (Asián-González & ark., 2007)(Gondim & ark., 2008). Three main mechanisms have been described for the formation of inflammatory dentigerous cysts: firstly, developing intrafollicular dentigerous cysts become infected by periapical inflammation emanating from primary teeth; secondly, inflammatory exudate from primary teeth leads to detachment of the reduced enamel epithelium from the enamel; and thirdly, radicular cysts developing in primary teeth fuse with the underlying permanent tooth germ to form extrafollicular dentigerous cysts.(Narang & ark., 2012) In addition, dentigerous cysts can develop when inflammatory stimulation in the periradicular area after pulp treatment of primary teeth affects the underlying permanent tooth follicle. Because of its close anatomical relationship to the permanent tooth germ and the high incidence of caries in primary teeth, it is reported to be the most common type of inflammatory dentigerous cyst associated with primary mandibular second molars. (Asián-González & et al., 2007)(W., 1980).

HISTOLOGY

A dentigerous cyst is a cyst of odontogenic origin, typically alternating with a thin non-keratinised epithelium. Histologically, it consists of three basic elements: cyst walls, cyst epithelium and cyst contents.

Cyst Epithelium: The dentigerous cyst is typically lined by a non-keratinized, stratified squamous epithelium consisting of 2 to 4 cell layers. The epithelial layer is usually well-organized and does not exhibit significant cellular atypia. However, in the presence of chronic inflammation, the epithelium may thicken and develop a reticular morphology**.**(Nayyer & et al.., 2017)(Siozopoulou & Vanhoenacker, 2020)

Cyst Wall: The cyst wall consists of fibrovascular connective tissue and typically has a thin stroma without

inflammatory cells. However, in cases of secondary infection or inflammation, lymphocytes, plasma cells, and macrophages may be present in the cyst wall (Siozopoulou & Vanhoenacker, 2020)(Rajendra Santosh, 2020).

Cyst Epithelium: The dentigerous cyst is lined with a flat, non-keratinised, soft epithelium, usually consisting of 2-4 cell layers. The epithelial layer is usually quite regular and shows no obvious cellular atypia. However, in the presence of chronic inflammation, the epithelium may thicken and acquire a reticular morphology. (Rajendra Santosh, 2020)(Siozopoulou & Vanhoenacker, 2020)

CLINICAL FEATURES

Dentigerous cysts are most commonly found in the mandibular third molar region (77%) and the maxillary canine region (11%). They can also be associated with a supernumerary tooth or an odontoma. (Bilodeau & Hunter, 2021). It usually progresses asymptomatically, and most cases are incidentally detected during routine radiographic examinations. However, when it reaches a certain size or becomes infected, various clinical findings may appear. When the cyst is small, it usually does not cause any symptoms. Patients typically do not notice any changes in their mouths, and the cyst is often detected incidentally during a dental visit for another reason (Bilodeau & Collins, 2017). As the cyst enlarges and causes bone expansion, patients may notice a visible swelling on their face. Large cysts, especially in the mandible, can lead to a noticeable widening of the lower jaw contour. Dentigerous cysts in the maxilla can expand toward the nasal floor, sinus cavity, or infraorbital region, leading to facial asymmetry. The dentigerous cyst is usually a painless lesion, but if secondary infection develops, pain, tenderness and sometimes increased temperature may be present. Infected cysts can cause inflammation of the surrounding

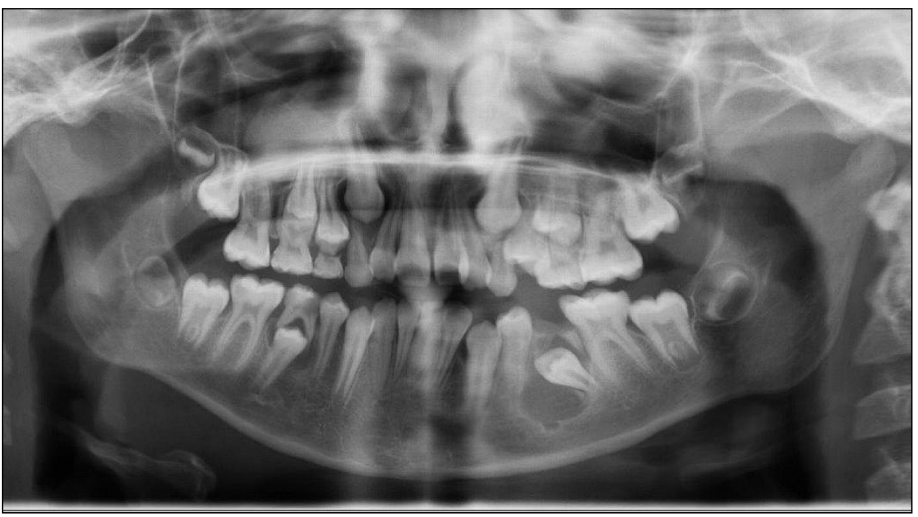
soft tissues, resulting in extraoral swelling and intraoral pain and tenderness (Freitas & ark., 2006).

As dentigerous cysts enlarge, they can exert pressure on the roots of adjacent teeth, leading to root resorption. This process may result in the displacement, inclination, or tipping of adjacent teeth. Large cysts, particularly those observed in the premolar and molar regions, can cause adjacent teeth to tilt, thereby disrupting occlusal relationships. As root resorption progresses, increased tooth mobility may occur, potentially leading to tooth loss in the long term (Bilodeau & Collins, 2017)(Wang & Olmo, 2021).

RADIOGRAPHIC FEATURES

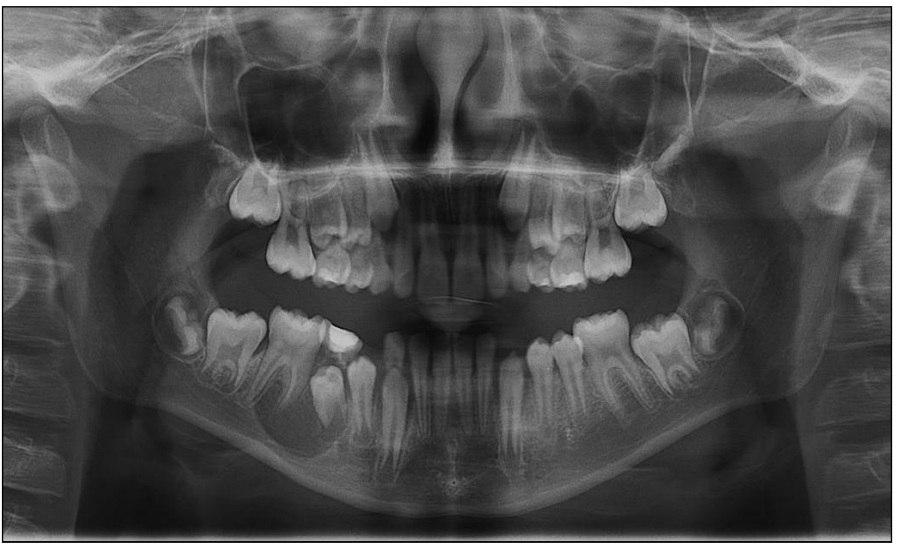
Radiographically, they usually appear as well-circumscribed unilocular lesions around an impacted tooth [Figure-1] (Bilodeau & Hunter, 2021). Rarely, large dentigerous cysts may appear multilocular on x-ray and may be confused with ameloblastoma.

***Figure 1**: Well-circumscribed unilocular dentigerous cyst around tooth 34*



Irregular and indistinct borders may be seen on radiographs when secondary infection develops in dentigerous cysts (Rajendra Santosh, 2020) . It may appear as a pericoronal radiolucency surrounding the germ of the underlying permanent tooth in relation to the lamina dura of the primary tooth in cases of inflammatory origin [Figure-2] (Narang & et al., 2012).

Figure 2 *: Dentigerous cyst resulting from the infection of primary tooth number 85.*



The radiographic distinction between a small dentigerous cyst and an enlarged follicle around the crown of an impacted tooth is difficult to make. According to some investigators, for a lesion to be considered a cyst, the radiolucent area surrounding the crown of the tooth must be at least 3-4 mm in diameter. (Wang & Olmo, 2021) (Farah & Savage, 2002).

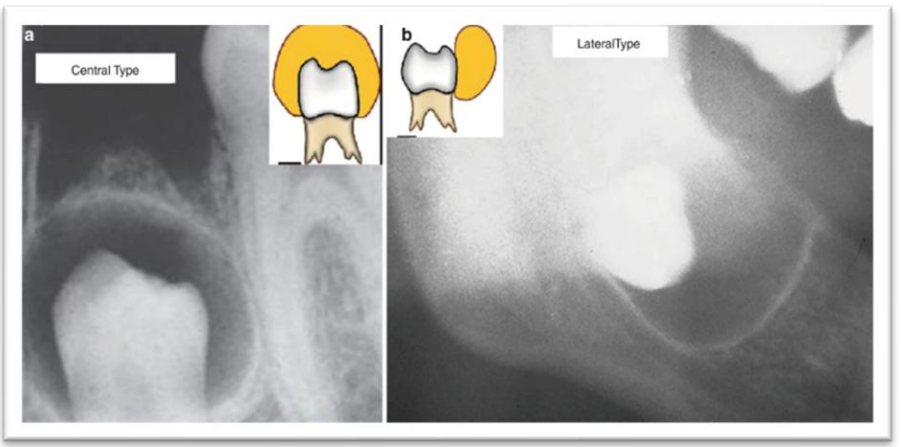
There are three different types of dentigerous cysts radiographically:

Central Type: This is the most common type and appears as a radiolucent area symmetrically surrounding the tooth's crown. Large cysts may extend to the lower border of the mandible or ramus, maxillary sinus, orbital floor or nasal floor and may cause the tooth to move away from the eruption direction [Figure-3a]. (Rajendra Santosh, 2020)(Nayyer & et al., 2017).

Lateral Type: It manifests as a radiolucent area surrounding the root surface laterally and part of the crown. It is most frequently observed in conjunction with partially erupted teeth, and its development is attributed to dilatation occurring on one side of the follicle [Figure -3b] (Nayyer & et al., 2017).

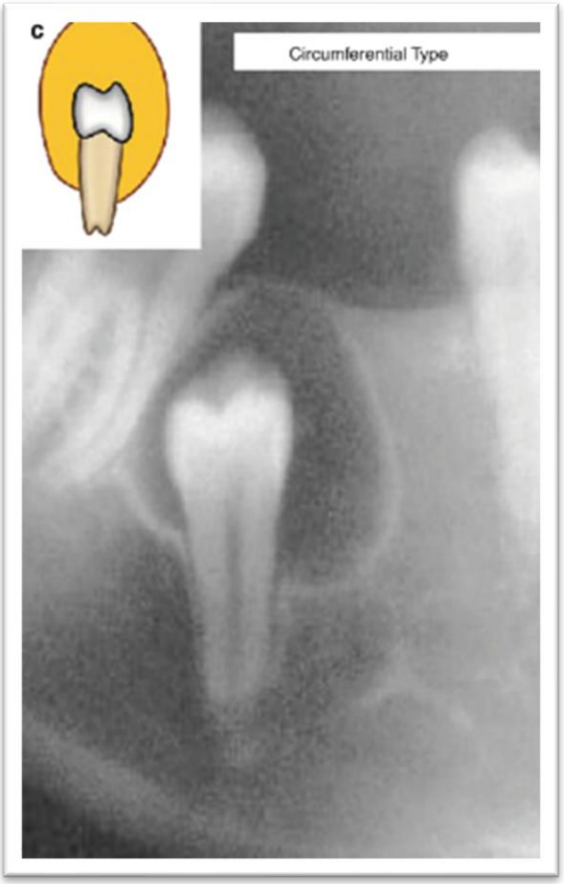
Circumferential Type: This is characterized by the presence of a radiolucent area surrounding the entire tooth, which can be more challenging to identify [Figure 4] (Nayyer & et al., 2017).

Figure 3: a: Central type dentigerous cyst, b: Lateral type dentigerous cyst



(Malik, 2021)

Figure 4: Circumferantial type dentigerous cyst



(Malik, 2021)

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of dentigerous cysts, other odontogenic and non-odontogenic cystic and tumoral lesions must be considered. The principal differential lesions given consideration are as follows: hyperplastic follicle keratocystic odontogenic tumor (KCOT), unicystic ameloblastoma, adenomatoid odontogenic tumor, unilocular radicular cysts, and, on occasion, non-odontogenic

cysts (e.g. nasopalatine canal cyst). One of the most commonly confused lesions is hyperplastic dental follicles, which produce a radiolucent appearance around impacted teeth. However, the hyperplastic dental follicle is generally characterized by a smaller diameter (typically less than 3 mm). Histopathological analysis reveals that the hyperplastic dental follicle is characterized by a thin layer of epithelium, in contrast to the thicker and more prominent epithelial tissue observed in dentigerous cysts. (Austin & Nelson, 2021). KCOTs usually show aggressive growth and tend to spread within the mandible. While most dentigerous cysts have a unilocular appearance, KCOT may be more multilocular. Since it may be difficult to differentiate these two cysts radiologically, histopathologic examination with biopsy is necessary (Konouchi & et al., 2014). Unicystic ameloblastomas can also be misdiagnosed as dentigerous cysts due to their similar association with impacted teeth. However, ameloblastomas are generally more aggressive and are histologically distinguished by a palisaded arrangement of basal cells. (Gunawardhana & et al., 2014). Another rare lesion that requires differentiation is an adenomatoid odontogenic tumor (AOT). AOT is a lesion that is often observed in young patients, typically located in the maxilla and associated with impacted teeth. While radiographically similar to a dentigerous cyst, AOT frequently exhibits small radiopacities and is histopathologically distinguished by the presence of islets of epithelial cells and rosette-like structures. (Borrás-Ferreres & et al., 2018). Finally, some non-odontogenic cysts and tumors, especially nasopalatinal canal cysts located in the maxilla or cystic lesions originating from the sinuses, may also be confused with dentigerous cysts. Consequently, advanced radiological modalities such as cone beam computed tomography (CBCT) and magnetic resonance imaging (MRI) should be employed to reach a definitive diagnosis. (Kiran & et al., 2015). When arriving at a diagnosis of the dentigerous cyst, it is essential to

take into consideration radiographic findings, clinical features, and histopathological evaluation as integrated components of the diagnostic process.

TREATMENT

The management of dentigerous cysts can be undertaken via marsupialization or enucleation, with the approach selected based on factors such as the dimensions and anatomic location of the lesion, the condition of the unerupted tooth, and the feasibility of subsequent follow-up. Whilst the recurrence rate is generally low, inadequate curettage and incomplete enucleation can result in the presence of cyst epithelium remnants, which can lead to recurrence (Kharis & et al., 2022). Particularly in large cysts with extensive bone loss, marsupialization may be preferred to protect the surrounding tissues and reduce the risk of pathological fracture. This method can also be applied in pediatric patients as it preserves the tooth, but it is reported that the recurrence rate may be higher. However, the disadvantages of marsupialization include the inability to completely remove the pathological tissue and the need for a second surgical intervention afterwards (Rioux-Forker & et al., 2019).

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ODONTOMA

ESRA AKÇAY [1]

Introductıon

The World Health Organization classifies odontomas as a benign odontogenic tumor composed of odontogenic epithelium and odontogenic ectomesenchyme with dental hard tissue formation (Praetorius F & Piatelli A, 2005). Odontomas are composed of enamel, dentin, cementum, and pulp tissues. These lesions found in the jaws represent a large percentage (%23-%77) of all odontogenic tumors. (Buchner et al., 2006; Servato et al., 2013; Siriwardena et al., 2019) Many studies have examined large-scale case series of these tumors and have described odontomas as the most common odontogenic tumors (Servato et al., 2013; Siriwardena et al., 2019). Odontomas are considered developmental hamartomas rather than true neoplasms of odontogenic origin. The distinguishing characteristic of a neoplasm is continuous and uncontrolled growth. However, several diagnosed cases of odontomas in the literature have reported reaching a sufficient size, leading to various complications such as jawbone expansion, tooth displacement, and tooth absence, thereby demonstrating neoplastic potential (An et al.,

[1] Uzm.Dt. Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı, Orcid: 0009-0003-9155-2379

2012; Bagewadi et al., 2015; Perumal et al., 2013). It remains unclear whether these odontogenic lesions represent a true neoplasm or merely a hamartoma (Soluk Tekkesin et al., 2012).

Odontomas are generally benign and asymptomatic lesions, typically diagnosed during routine radiographic examinations. The most prevalent clinical symptom observed in cases of odontoma is the delayed eruption of permanent teeth, a consequence of the obstruction of tooth eruption by the odontoma (Amado Cuesta et al., 2003; Garcia-Consuegra et al., 2000). As odontomas most frequently manifest in the anterior region of the maxilla, the maxillary incisors and canines are the teeth most commonly affected in such cases (Batra et al., 2004; Fernandes et al., 2005; Garcia-Consuegra et al., 2000). A further symptom that may be encountered in cases of odontoma is the palpation of the tumour lesion during clinical examination. The delayed eruption of permanent teeth and palpation of the odontoma lesion during clinical examination constitute almost all of the clinical signs of odontomas. Other findings that are very rare in odontoma cases are symptoms such as agenesis of the permanent tooth, pain, inflammation or infection (Batra et al., 2004; Fernandes et al., 2005; Garcia-Consuegra et al., 2000; Hidalgo-Sánchez et al., 2008; Kämmerer et al., 2016).

Etiology

Etiology of theses tumors is unknown, nevertheless, some authors have described the possible relationship with trauma in primary dentition, Malassez paradental remains, inflammation processes, odontoblastic hyperactivity and hereditary anomalies (Hidalgo-Sánchez et al., 2008). Odontomas may also present as part of certain syndromes such as Gardner syndrome, Hermann syndrome, familial colonic adenomatosis and basal cell nevus syndrome (Hidalgo-Sánchez et al., 2008; Iatrou et al., 2010).

Clinical Characteristics

Odontomas are generally benign and asymptomatic lesions, typically diagnosed during routine radiographic examinations. The most prevalent clinical symptom observed in cases of odontoma is the delayed eruption of permanent teeth, a consequence of the obstruction of tooth eruption by the odontoma (Amado Cuesta et al., 2003; Garcia-Consuegra et al., 2000). As odontomas most frequently manifest in the anterior region of the maxilla, the maxillary incisors and canines are the teeth most commonly affected in such cases (Batra et al., 2004; Fernandes et al., 2005; Garcia-Consuegra et al., 2000). A further symptom that may be encountered in cases of odontoma is the palpation of the tumour lesion during clinical examination. The delayed eruption of permanent teeth and palpation of the odontoma lesion during clinical examination constitute almost all of the clinical signs of odontomas. Other findings that are very rare in odontoma cases are symptoms such as agenesis of the permanent tooth, pain, inflammation or infection (Batra et al., 2004; Fernandes et al., 2005; Garcia-Consuegra et al., 2000; Hidalgo-Sánchez et al., 2008; Kämmerer et al., 2016).

In general, these lesions are diagnosed between the second and third-fourth decades of life. Complex odontomas are more frequent in children and adolescents. No significant gender differences have been reported (Amado Cuesta et al., 2003; Ferrer Ramírez et al., 2001; Garcia-Consuegra et al., 2000). In case series studies examining many odontoma cases, an attempt was made to establish a relationship between odontoma type and patient gender, but no significant findings were found (Hidalgo-Sánchez et al., 2008).

Radiographic Characteristics

Odontomas are typically diagnosed during routine radiographic examinations. Radiographically, an odontoma appears

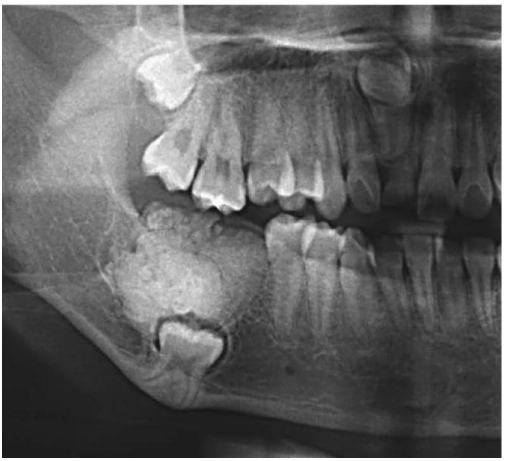
as a well-defined, radiopaque lesion surrounded by a thin radiolucent area, which is bordered by a thin soft tissue capsule (Amado Cuesta et al., 2003; Ferrer Ramírez et al., 2001; Garcia-Consuegra et al., 2000). Depending on the degree of calcification of the odontoma, three distinct developmental stages can be radiographically differentiated: the first stage, characterized by the lesion appearing radiolucent (due to the absence of calcification in the dental tissues); the second stage, marked by partial calcification; and the third stage, in which the odontoma appears radiopaque and is surrounded by a thin radiolucent halo (Garcia-Consuegra et al., 2000). Odontomas are classified based on their radiographic appearances and histological features. The World Health Organization classifies odontomas histopathologically into two variants: complex odontomas and compound odontomas (Amado Cuesta et al., 2003; Ferrer Ramírez et al., 2001; Garcia-Consuegra et al., 2000).

Classification of Odontomas

Complex odontomas

Complex odontomas appear as radiopaque, irregular, and amorphous masses, with the radiopaque lesion not resembling dental structures. A typical feature is the description of a sunburst-like appearance on the X-ray image (Amado Cuesta et al., 2003; Ferrer Ramírez et al., 2001). Complex odontoma lesions are more commonly found in the mandibular premolar and molar regions (Amado Cuesta et al., 2003). Often an impacted tooth or a dentigerous cyst can be observed with the odontomas (Kämmerer et al., 2016).

Figure 1: Representative panoramic radiograph of complex odontoma

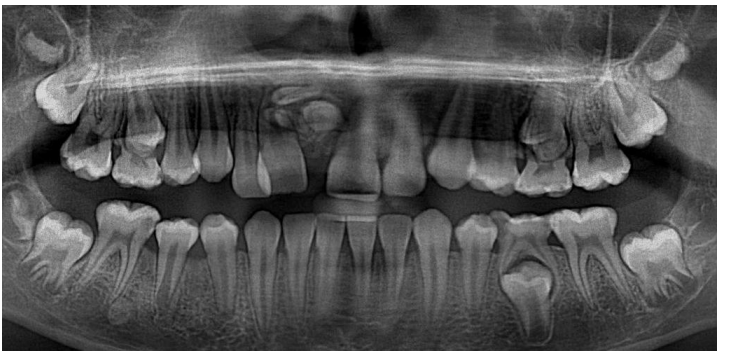


Reference:(Nguyen & Van Huynh, 2023)

Compound Odontomas

Compound odontomas appear radiographically as well-defined lesions containing radiopaque areas within a radiolucent halo. In compound odontomas, multiple follicular formations are observed, and these follicular formations are separated by fibrous septa. Compound odontomas are lesions that exhibit multiple small tooth-like structures called denticles. Compound odontoma lesions are commonly located in the anterior region of the maxilla. A review of the existing literature reports that compound odontomas are more commonly observed than complex odontomas (Amado Cuesta et al., 2003; Ferrer Ramírez et al., 2001; Garcia-Consuegra et al., 2000).

Figure 1: Representative panoramic radiograph of compound odontoma



In the literature, there are case reports in which radiological and histological features of compaund and complex odontoma types are observed together. This type of odontoma is known as a compound-complex odontoma (Khalifa et al., 2022; Soluk-Tekkeşin & Wright, 2018) .

A review of the available literature reveals that many odontoma cases are associated with teeth and are located in the alveolar bone (Hidalgo-Sánchez et al., 2008). However, isolated odontoma cases located within the maxillary sinus have also been reported. Although odontomas in the maxillary sinus are very rare, one reported case described clinical symptoms such as pain in the posterior maxillary region, unilateral nasal obstruction, and diplopia. In many cases of maxillary sinus odontomas, chronic sinusitis symptoms are commonly observed, including pain in the maxillary sinus region and/or posterior maxillary area, nasal obstruction, decreased sense of smell, dark nasal discharge, pressure sensation around the eyes, and halitosis (Ćabov et al., 2005; Crespo Del Hierro et al., 2008; Sotobori et al., 2013).

A review of the existing literature on odontoma cases reveals reports of "erupted odontomas," which spontaneously erupt into the oral cavity. Erupted odontomas are rare lesions, with the first case

reported by Rumel et al. in 1980 (Pró et al., n.d.; Rumel et al., 1980). Literature includes case reports of both complex and compound odontomas spontaneously erupting into the oral cavity. However, it has been reported that erupted odontomas are mostly of the complex type. This type of odontoma is typically observed in older individuals and is usually associated with impacted teeth. These lesions may present with pain, inflammation, and infection (Pró et al., n.d.).

Differential Diagnosis

The differential diagnosis of odontomas should include ameloblastic fibroodontoma, odontoameloblastoma, and ameloblastic fibroamilioma. If they present with a similar radiographic appearance to an odontoma and are located in the intra-radicular area, the differential diagnosis should also consider odonto-ameloblastoma, focal residual osteitis, cementoma, calcifying epithelial odontogenic tumor, adenomatoid odontogenic tumor, supernumerary tooth, cement fibrooma, or benign osteoblastoma (Amado Cuesta et al., 2003). During its intermediate stage, complex odontomas may resemble other lesions such as fibro-osseous lesions, calcifying cystic odontogenic tumors, fibrous dysplasia, and chronic osteomyelitis (Philipsen et al., 1997).

Treatment

The treatment of odontomas is performed through conservative surgical enucleation, planned according to the location and size of the odontoma (Philipsen et al., 1997). Surgical enucleation can be carried out under local or general anesthesia. However, the intrabuccal approach with local anesthesia is the most preferred treatment option (Amado Cuesta et al., 2003). Following excision, bone grafts may be necessary depending on the need for further treatment or the size and location of the odontoma (Lee et al., 2015). In odontoma cases, the connective tissue capsule surrounding

the odontoma, resembling the follicle of a tooth, helps in the complete removal of the lesion by separating it from the surrounding healthy bone. As a result, the recurrence rate after surgery is very low (Moore JR, 1985). It has been reported that the recurrence rate increases when enucleation is performed during the initial calcification stage of the odontoma (Amado Cuesta et al., 2003; Hisatomi et al., 2002). Postoperative follow-up of the case is crucial, and if necessary, preventive and interceptive orthodontic treatments can be applied to prevent future malocclusions (Barba et al., 2016; Zidane et al., 2022).

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ODONTOGENIC KERATOCYSTS

MEHMET ÜNAL[1]
RUMEYSA ÇAKIRCALI[2]

Introduction

Cysts are pathological cavities filled with fluid or semi-fluid material, lined by multilayered squamous epithelium, and surrounded by a connective tissue capsule. They are more commonly observed in the jawbones compared to other bones in the body. This is attributed to the higher prevalence of embryological remnants of odontogenic epithelial origin in this region. (Borghesi ve ark.; 2018)

In 2005, the WHO reclassified Odontogenic Keratocysts (OCCs) as tumors, revising the previous two editions of the WHO classification. The term keratocystic odontogenic tumor (KCOT) was proposed as the appropriate name. However, in 2017, the WHO reverted to the term odontogenic keratocyst (OCCs), reflecting the extensive and ongoing research conducted on this condition. (Soluk &Wright, 2022)

[1] Doktor Öğretim Üyesi, Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı 0000-0001-9111-6962
[2] Araştırma Görevlisi, Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi, Pedodonti Anabilim Dalı 0009-0006-3480-6266

OCCs is a benign but aggressive intraosseous tumor derived from remnants of the original dental lamina or tooth germ. It is thought to arise from traumatic implantation, downward growth of the basal cell layer of surface epithelium, or the reduced enamel epithelium of the dental follicle. Unlike most other jaw cysts, it has a very high recurrence rate and a tendency to transform into malignancy. OCCs can occur in any part of the jaw, but it is more commonly found in the ascending ramus and molar regions. However, there are very few reports indicating that most OCCs occur in soft tissues, such as the gingiva, buccal cavity, retromolar trigone, deep facial regions, or the area of the temporalis muscle. (Borghesi ve ark.; 2018)

OCCs Description

OCCs is most commonly observed in adults aged 30-50, though it has been reported across all age groups, ranging from 7 to 93 years. It is estimated to account for 4% to 16.5% of all odontogenic tumor cases. In its early stages, patients with OCCs often exhibit no noticeable symptoms or signs. Even when the lesion is large, it typically does not cause significant jaw expansion, making it unnoticeable in facial appearance. (Avril ve ark.; 2014, Johnson; 2014)

In some cases, patients may experience symptoms such as swelling, pain, abnormal sensations, discharge of pus, and mobile teeth. Among these, swelling is the most common clinical indication of OCCs. If an OCCs continues to grow and the bone gradually expands, facial deformity may occur. As a result, the surface bone becomes a very thin bony plate, which, upon palpation, may produce a ping-pong-like sensation and a so-called parchment-like brittle sound. (Johnson; 2014)

In severe cases, pathological fractures may occur, or the thin bony plate may eventually be resorbed, resulting in a wave-like

movement upon palpation. Tumor cells in OCCs can also invade the nasal cavity and maxillary sinus. In advanced cases, these cells may affect vision and even lead to diplopia. If OCCs is adjacent to teeth, it may cause compression of the neighboring teeth, leading to their displacement, loosening, or tilting. As a result, tooth loss may clinically accompany OCCs. It is worth noting that the extraction of mobile teeth in OCCs patients must be performed with extreme caution to avoid complications. If a mobile tooth originates from OCCs, tooth extraction or trauma may rupture the cyst, revealing yellow and white keratin-like substances. If the tumor is secondarily infected, symptoms such as swelling, pain, fever, and general discomfort may occur. Importantly, if swelling appears in other parts of the body, the patient's medical history should be examined in detail to avoid misdiagnosis or missed diagnosis. (Gupta ve ark.; 2011)

OCCs Localization

Similar to other odontogenic entities, OCCs originate from tooth-bearing areas. They are reported to occur twice as frequently in the mandible as in the maxilla. (Harmon ve ark.; 2015) When OCCs arise in the mandible, the most common locations are the posterior sextant, the angle, or the ramus. (MacDonald ;2016) Conversely, in the maxilla, the anterior sextant—primarily between the canine and lateral incisor—and the third molar region are the most frequent sites of origin. (Ali & Baughman ; 2003)

Large lesions are particularly common in the angle and ramus of the mandible (Mendes, Carvalho & van der Waal; 2010) According to the literature, OCCs can be found in periapical positions, pericoronal positions, or lateral root positions. In approximately 30% of cases, they have no association with any dental structure. Despite their aggressive behavior, OCCs typically cause minimal bone expansion in most cases, due to their tendency

to spread along the intramedullary cavity, described as "growing in the length of the bone" (Avril ve ark.; 2014, Ali & Baughman ; 2003, Scarfe, Toghyani & Azevedo; 2018)

Large lesions causing significant cortical plate erosion and involving surrounding structures may occur in asymptomatic patients. (Hyun, Hong & Kim ;2009) As a result, especially in Western countries, the presence of OCCs might be incidentally discovered at later stages during routine radiological investigations. A systematic review of the literature published by MacDonald-Jankowski in 2011 indicated that patients of East Asian descent may display early symptoms characterized by swelling and pain, while lower alveolar nerve anesthesia is more frequently reported in Latin American populations. Unlike ameloblastomas and other odontogenic lesions with similarly aggressive behavior, OCCs rarely cause root resorption of adjacent teeth. (Jankowski; 2011)

Diagnosis

The most commonly used radiological imaging techniques in the study of OCCs s are conventional radiography (mainly panoramic radiography), computed tomography (CT), and magnetic resonance imaging (MRI). These imaging methods differ significantly in terms of their technical characteristics, acquisition techniques, indications, and the information they provide

Radiographically, OCCs may show tooth displacement and root resorption; the latter is a rare radiographic feature of OCCs, with a reported incidence ranging from 1.3% to 11%. The literature indicates that cortical bone perforation is not an unusual characteristic of OCCs, with an intraoperative incidence ranging from 39% to 51%. However, this finding is very rarely detected in panoramic radiographs and is typically confined to the alveolar ridge. (Chirapathomsakul, Sastravaha & Jansisyanont ;2006)

CT is considered superior to conventional radiography in distinguishing OCCs from other unilocular or multilocular osteolytic lesions and in preoperative evaluation. In the assessment of cystic lesions of the jaws, MRI is primarily used as a complementary technique to CT (CBCT or MDCT) and can be beneficial in selected cases to provide better visualization of internal characteristics and soft tissue involvement. Some studies have summarized that these MRI signal characteristics are useful in differentiating OCCs from ameloblastomas (Hisatomi ve ark.; 2003, Fujita ve ark.; 2013, Konouchi, Asaumi &Yanagi; 2006)

The presence of multiple OCCs is considered one of the primary diagnostic criteria for NBCCS (Gorlin-Goltz syndrome) and may be the initial manifestation of the disease. Multiple OCCs are also observed in other syndromes, such as Noonan syndrome, Ehlers-Danlos syndrome, and oral-facial-digital syndrome. In syndromic OCCs, cysts tend to appear at an early age (during the first or second decades of life), more frequently arise from the posterior sextants of the maxilla, exhibit more aggressive behavior, and have higher recurrence rates compared to non-syndromic OCCs (Uğurlu, Özkan & Bulut, 2024)

Differential diagnosis

The differential diagnosis of OCCs includes odontogenic myxoma, ameloblastoma, central giant cell granuloma, adenomatoid odontogenic tumor, and dentigerous cyst (follicular cyst) when evaluated radiologically. Histologically, the differentiation is made from orthokeratocysts, radicular cysts, and ameloblastoma.

Histology

Odontogenic keratocysts (OCCs) possess distinctive histological features that are crucial for diagnostic purposes. Under microscopic examination, OCCs bear a subtle resemblance to

keratinized squamous epithelium; however, they lack rete ridges and frequently exhibit an artifactual separation from their basal membrane. (Thompson LD; 2006)

The fibrous wall of the cyst is typically thin and non-inflamed. The epithelial lining is thin, uniformly thick, and parakeratinized with columnar cells in the basal layer exhibiting focal reverse polarization (nuclei are positioned at the opposite pole of the cell). Basal cells resemble pre-ameloblasts, indicating an odontogenic origin. The epithelium may separate from the wall, resulting in epithelial islands. These can form 'satellite' or 'daughter' cysts, contributing to an overall multilocular cyst. The presence of daughter cysts is particularly observed in individuals with NBCCS. Inflamed cysts no longer exhibit the characteristic features of OCCs and instead display hyperplastic epithelium resembling radicular cysts. Due to areas of focal inflammation, a larger biopsy is required for the accurate diagnosis of odontogenic keratocysts. (Odell & Cawson ;2017, Coulthard ve ark.; 2013, Sharif ve ark.; 2016)

Treatment and Follow-up

The management of odontogenic keratocysts (OCCs) aims to reduce the risk of recurrence while minimizing morbidity for the patient. Currently, there is no consensus on the optimal treatment method. Various factors influence the selection of the most appropriate treatment, including the size and location of the lesion, unilocular or multilocular nature, the presence of cortical perforation or soft tissue involvement, and the age of the patient. (de Castro ve ark.; 2018, Dioguardi ve ark.; 2024)

Various surgical options have been considered, including enucleation alone or in combination with adjunctive measures (ostectomy, Carnoy’s solution, cryotherapy), marsupialization and decompression, as well as marginal or segmental resection. (Stoelinga;2005)

A systematic review of the literature by Johnson et al. demonstrated that enucleation was associated with the highest recurrence rate of approximately 30%, followed by marsupialization alone with a recurrence rate of around 18%. The association of lesion enucleation with an adjunctive chemical cauterization technique using Carnoy's solution—a mixture of chloroform, absolute ethanol, glacial acetic acid, and ferric chloride—significantly reduced recurrence rates to approximately 8%. (Johnson, Batstone & Savage; 2013)

Therefore, periodic radiographic monitoring of patients with surgically treated OCCs is recommended annually for the first 5 years, and thereafter at least every 2 to 3 years. (Johnson, Batstone & Savage; 2013) Patients with NBCCS are particularly prone to the formation of new lesions, both at previous surgical sites and in different sextants of the dental arches. (Bresler, Padwa & Granter; 2016) Consequently, long-term rigorous radiological follow-up is essential for these patients.

Conclucion

OCCs has received increasing attention due to its high recurrence rate and tendency to malignant transformation. The clinical presentation, diagnosis, and treatment options should be carefully evaluated. Before selecting a treatment for keratocystic odontogenic tumors, preoperative imaging data must be carefully read to ensure that the surgical field is clear, omission of the tumor is avoided, and the tumor is completely removed. All these precautions will significantly help to prevent recurrence or malignant transformation of OCCs.

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MANDIBULAR BUCCAL BIFURCATION CYST

HATİCE TÜRKOĞLU[1]

Introduction

The mandibular buccal bifurcation cyst (MBBC) is a rare inflammatory odontogenic cyst associated with the mandibular permanent first or second molar.(Kim et al., 2018) This cyst was first described by Stoneman and Worth in 1983 and was termed the "buccal bifurcation cyst."(Bautista et al., 2019) The term "paradental cyst" has also been used over time to describe this cyst.(Ruddocks et al., 2022) However, the World Health Organization included this lesion in the histological classification of odontogenic tumors in 1992 and named it the "mandibular infected buccal cyst." (Kim et al., 2018; Rao et al., 2023) It has been reported that the term "paradental cyst" should be used for cysts associated with mandibular third molars.(Ruddocks et al., 2022) Finally, the WHO classified this lesion under the category of "collateral inflammatory cysts" alongside the paradental cyst and referred to it as MBBC.(De Souza et al., 2025) Nevertheless, many cases of MBBC are still found in the literature under the name paradental cyst.(Philipsen et

[1] Dr.Öğrt.Üyesi, Afyonkarahisar Sağlık Bilimleri Üniversitesi Diş Hekimliği Fakültesi,Pedodonti Anabilim Dalı, Orcid: 0000-0003-4271-2463

al., 2004) Studies have reported many cases of MBBC that were diagnosed and treated as paradental cysts.(Philipsen et al., 2004)

MBBC accounts for approximately 0.9% to 4.7% of all odontogenic cysts. It is most commonly observed in the pediatric population, particularly in children between the ages of 4 andc 14. It is typically observed on the buccal surface of partially erupted or fully erupted permanent first and second molars. Although rare cases have been reported in maxillary canines, it is most commonly seen in the mandible.(Aloyouny et al., 2021; Bautista et al., 2019) Although most MBBCs are observed unilaterally, they can also occur bilaterally (Borgonovo et al., 2012a; Lacaita et al., 2006).

Etiology

The etiology of MBBC remains unclear. Although it was previously believed that these cysts originated from the reduced enamel epithelium, it is now considered more likely that they arise from the sulcular or junctional epithelium. It has been suggested that inflammation initiated by perforation of the epithelium during tooth eruption and disruption of the oral epithelial integrity triggers epithelial proliferation, leading to the formation of MBBC.(Gallego et al., 2007) This hypothesis seems reasonable, considering that MBBC typically manifests during or immediately following tooth eruption. Additionally, it is suggested that the cyst developed on the buccal surface of the tooth due to the fact that the mesiobuccal tubercle of the first molar tooth was the first to rupture from the epithelium.(Stoneman & Worth, 1983) It is also believed that factors such as crown morphology, fissure pattern, adjacent teeth, and periodontal structures may influence the location of the cyst.(Derindağ et al., 2019)

It is also suggested that the cyst epithelium may originate from various sources, including the residues of the dental lamina epithelium, Malassez epithelial rests, or Serres' rests.(Borgonovo et

al., 2012b) Other theories suggest that these cysts may be variants of lateral periodontal cysts, potentially arising from a unilaterally expanded dental follicle or serving as a source of inflammation in deep periodontal pockets.(Aloyouny et al., 2021)

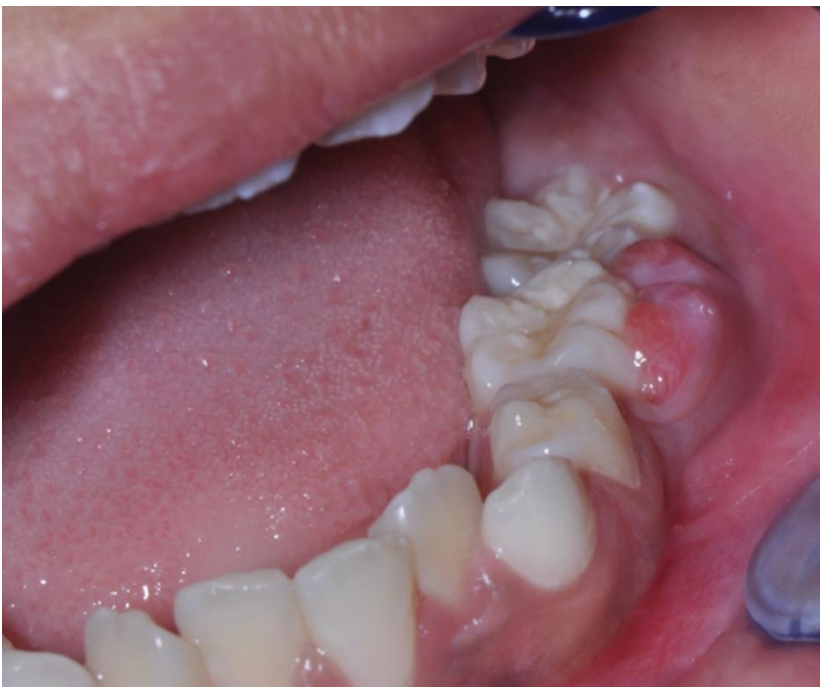
In addition to the origin of the cystic epithelium, the cause of the inflammatory process is also a subject of debate. Inflammation may be triggered by food impaction (pericoronitis) in an opened pericoronal pocket. Obstruction results in the accumulation of fluid within the impacted pockets, leading to cystic expansion via osmosis. Furthermore, it has been suggested that enamel projections into the furcation area of the tooth may act as a triggering factor for MBBC. (Colgan et al., 2002) According to the World Health Organization Classification of Head and Neck Tumors (2017), the etiopathogenesis of MBBC is the same as that of the paradental cyst, and this is noteworthy.(Aloyouny et al., 2021)

Clinical Characteristics

The mandibular buccal bifurcation cyst is most commonly seen in children between the ages of 4 and 14. Reported cases show no gender predilection. Although it primarily affects the mandibular first molar (65.51%), it has also been observed in the mandibular second molar (9.65%).(Philipsen et al., 2004) The involved tooth is typically vital. A deep periodontal pocket in the buccal gingiva, expansion of the buccal cortex, and the crown's inclination toward the buccal surface are characteristic features of MBBC (Figure 1).(Ramos et al., 2012; Shohat et al., 2003) Eruption of the tooth is usually impaired. Displacement may also be observed in adjacent unerupted teeth. Symptoms typically appear at the time of eruption of the affected tooth. The affected tooth may be asymptomatic, or it may present with symptoms such as pain and localized swelling. In the case of infection, pus discharge may be observed.(Kim et al., 2018)

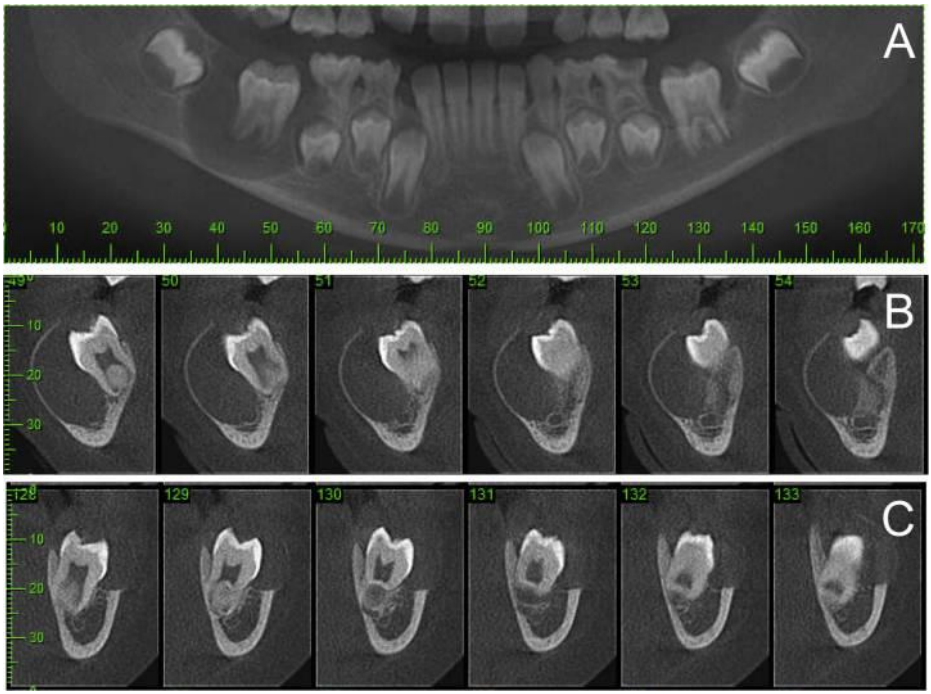
A common radiographic finding is well-defined radiolucency in the furcation area surrounding the roots of the affected tooth (Figure 2).(Bautista et al., 2019) Unlike lesions of endodontic origin, the lamina dura and periodontal ligament appear normal.(Thikkurissy et al., 2010) A superficial periosteal reaction may be observed in the cortical bone. The apices are displaced lingually, and the lingual tubercles are more prominent. The lower border of the mandibular bone is intact.(David et al., 1998) Displacement may be observed in adjacent unerupted teeth. In computed tomography images, perforation of the mandibular buccal cortex associated with the cyst may be observed.(Rao et al., 2023) Since it can also occur bilaterally, it is important to carefully examine the teeth in the opposing arch.(Bautista et al., 2019)

Figure 1: Buccal swelling associated with the mandibular left permanent first molar



Reference:(Ruddocks et al., 2022)

Figure 2: Central panoramic view in which a hypodense lesion may be observed bilaterally in the regions of permanent mandibular first molars. B and C – Transverse view of the right and left sides, respectively, in which buccal expansion of the cortical bone as well as the tilting of the teeth may be seen.



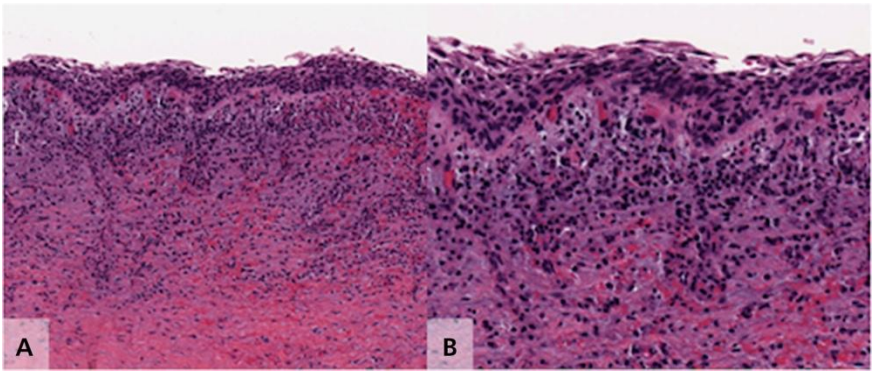
Reference:(Bautista et al., 2019)

Histology

The mandibular buccal bifurcation cyst is an inflammatory cyst, and it is difficult to diagnose based solely on histological sections. Its histopathological features resemble those of radicular and paradental cysts, both of which are inflammatory lesions.(Thikkurissy et al., 2010) The cyst epithelium is lined with non-keratinized squamous epithelium of variable thickness (Figure 3). A cystic capsule composed of vascularized connective tissue

exhibiting chronic inflammatory cell infiltration has been observed. Due to the non-specific microscopic features of this lesion, diagnosis should be made by integrating clinical, radiographic, surgical, and histological data.(Ramos et al., 2012; Thikkurissy et al., 2010)

Figure 3: Histologic features of mandibular buccal bifurcation cyst. Cystic lining of nonkeratinizing stratified squamous epithelium and underlying chronic inflammation (hematoxylin-eosin, 4 magnification) (A) and (hematoxylin-eosin, 20 magnification) (B).



Reference:(Ruddocks et al., 2022)

Treatment

Treatment methods for mandibular buccal bifurcation cysts have evolved over time. In many cases, treatments involving curettage of the cyst following the extraction of the affected tooth have proven successful.(Stoneman & Worth, 1983) However, since the extraction of permanent teeth may have negative effects on occlusion, more conservative treatments have been adopted over time. Conservative surgical enucleation is the preferred approach as it promotes bone healing and helps prevent recurrence.(Ruddocks et al., 2022) In this method, molar teeth, which are important for occlusion, can maintain their presence in the oral cavity without extraction. The literature reports success in many cases of MBBC

treated with enucleation. After enucleation, the teeth have been able to erupt normally, and bone healing has been observed.(Bautista et al., 2019; Pompura et al., 1997) In cases where a bone graft is placed after cyst enucleation, it has been reported to enhance bone regeneration, provide stability, and facilitate soft tissue reattachment.(Levarek et al., 2014)

Recently, a more conservative treatment method that does not involve surgical intervention has been proposed. Among these, a treatment method has been developed in which a small opening is created in the cyst epithelium using a periodontal probe from the buccal gingival pocket, thought to perform micromarsupialization. By rinsing this opening with daily saline or hydrogen peroxide, the pressure within the cyst may decrease over time, leading to the shrinkage and healing of the cyst.(Tseng et al., 2023; Zadik et al., 2011)

Recently, cases of MBBC that have spontaneously healed without any intervention have also been reported. In these cases, it is believed that automarsupialization occurred as a result of microtrauma.(Lizio et al., 2011; Santos et al., 2011) Marsupialization is a non-surgical treatment method that can be applied in small, asymptomatic cases. In these cases, regular follow-up and monitoring are crucial. In larger lesions, cyst enucleation is a successful and effective treatment option.(Stepic et al., 2011) The prognosis of MBBC is favorable, and recurrence is rare when treated appropriately. In most cases, conservative treatment is sufficient, and healing can occur without recurrence. Accurate diagnosis is crucial to avoid unnecessary larger procedures and to preserve the affected teeth in the oral cavity.(Lacaita et al., 2006; Ruddocks et al., 2022)

Differential Diagnosis

Since the mandibular buccal bifurcation cyst is an inflammatory lesion, its histological findings are quite similar to

those of other inflammatory cysts. The diagnosis of MBBC should be made by combining clinical, radiographic, and histological findings.The differential diagnosis of mandibular buccal bifurcation cyst includes dentigerous cyst, lateral radicular cyst, and periodontal pocket.(Chrcanovic et al., 2010) Circumferential dentigerous cyst, similar to MBBC, attaches to the tooth at the cemento-enamel junction, can be seen around the first and second molars, and may inhibit tooth eruption.(Ramos et al., 2012; Shibata et al., 2004) Histological sections may also show inflammation, hyperplasia, and leukocyte exocytosis, similar to MBBC.(Chrcanovic et al., 2010)

Paradental cysts are inflammatory lesions that commonly affect a vital tooth, typically found on the lateral surface, and are most often observed around mandibular third molars. They typically present a clinical picture similar to that of pericoronitis. Although classical paradental cysts and MBBCs are considered the same inflammatory lesions due to similarities in etiological factors and microscopic features, they differ in terms of anatomical location, clinical presentations, radiographic characteristics, and treatment methods. (Tseng et al., 2023; Zadik et al., 2011)

CBCT will be an important aid in making the differential diagnosis, as the sections obtained can reveal buccal cortical expansion and lingual inclination of the tooth apices, which are characteristic of MBBC. It is important to distinguish MBBC from other lesions and make an accurate diagnosis, as in most cases, conservative treatment is sufficient, leading to healing without recurrence.(Bautista et al., 2019)

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PRE-ERUPTIVE INTRACORONAL RESORPTION

MELİKŞAH URAL [1]

Introduction

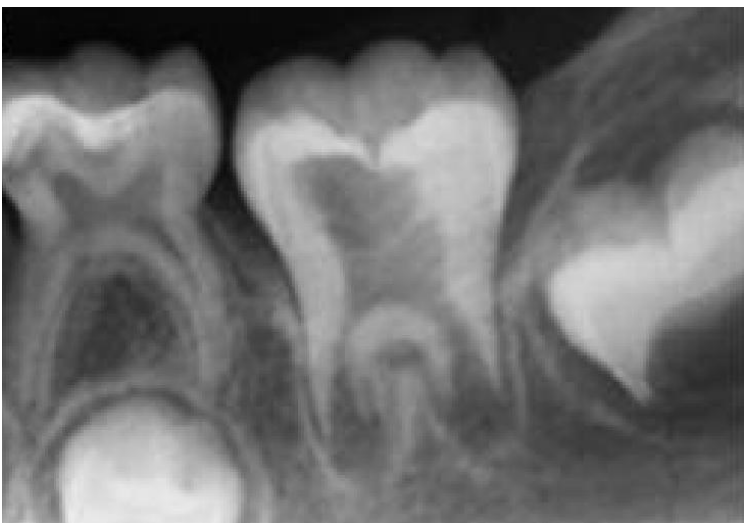
The term “Pre-eruptive Intracoronal Resorption” is defined as an abnormal, well-circumscribed radiolucent area within the coronal dentin tissue that occurs close to the enamel-dentin junction on the occlusal surfaces of unerupted teeth. These lesions have been variously referred to as “Pre-eruptive Intracoronal Resorption (PIR)”, “pre-eruptive caries”, “PIR or defect or radiolucency”, “intra-follicular caries” and “idiopathic coronal resorption” (W. Seow et al., 1999) . In this section, the term “PIR” is preferred.

Pre-eruptive intracoronal resorption is usually diagnosed incidentally. On radiographs of unerupted teeth, these affected teeth usually appear as well-demarcated radiolucencies. These lesions often resemble dental caries, and the term 'pre-eruptive caries' has sometimes been applied. Evidence from clinical and histologic case reports suggests that these lesions may be resorptive in nature (W. Seow et al., 1999).

[1] Uzm. Dt., Afyonkarahisar Sağlık Bilimleri Üniversitesi, Diş Hekimliği Fakültesi, Orcid: 0000-0003-0940-9913

Diagnosing these lesions at an early stage will help to prevent resorption of the pulp. It can be identified and treated without affecting the tooth (Holan et al., 1994). PIR defects do not involve cariogenic bacteria as in dental caries. However, upon eruption of the tooth, the area affected by resorption comes into contact with the oral flora, accelerating the demineralisation of the tooth. Clinically, these caries have been reported to look like normal dental caries (Moskovitz & Holan, 2004). No association between intracoronal resorption and race, gender, medical conditions, systemic factors or fluoride supplementation has been reported (W. Seow et al., 1999).

Figure 1: Radiographic view of PIR



Reference: (Elbay et al., 2015)

Etiology

In the literature, this condition was first described by Skillen in 1941 and various theories have been proposed for its etiology (Skillen, 1941). In the literature, the etiology includes an acquired pathologic condition resulting from apical inflammation of the deciduous teeth, dental caries, local developmental defects of dentin

with or without concomitant enamel defects, and internal or external resorption (W. K. Seow & Hackley, 1996). In addition, local factors and developmental defects, especially ectopic positioning of permanent teeth, have been suggested to play a role. Periapical inflammation in the deciduous tooth can cause disruption of the dental epithelium, favoring the formation of inflammatory resorptive cells that cause a resorptive defect in the developing tooth crown. However, the majority of defects were found in teeth unrelated to deciduous tooth germ, such as first and second permanent molars, suggesting that other factors are involved and that this theory does not explain all radiolucencies in posterior teeth (Al-Batayneh & AlTawashi, 2020a).

Systemic factors do not seem to play an etiologic role. Local factors such as ectopic eruption, in which 13-28% of PIR occurs, may be due to pressure from abnormal tooth position leading to local damage to the protective layer of the tooth and resorptive cells can easily initiate dentin resorption, which usually does not resorb until later stages as enamel is much harder (Özden & Acikgoz, 2009). Similarly, impacted teeth in ectopic position can cause resorption of neighboring teeth.

The fact that retrospective studies are more common and that it is difficult to obtain sufficient numbers of teeth or tissues for examination has led to a poor understanding of the etiology of these lesions (Counihan & O'Connell, 2012).

Most likely, all the theories could be possible explanations for each reported case. However, only one of the theories, the possibility of pre-eruptive caries without exposure to the oral environment, is highly doubtful and has been rejected according to the commonly known caries pathophysiology. Currently, the most acceptable theory for the cause of PIR is intra-coronal resorption

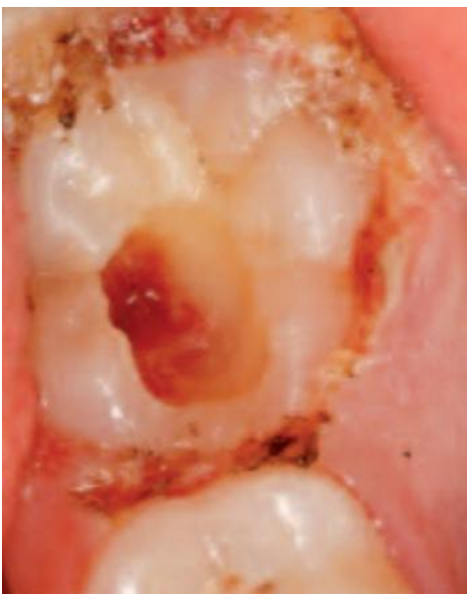
through dentine invasion by resorptive cells interrupting crown formation (Al-Batayneh & AlTawashi, 2020a).

Clinical Appearance of PIR Defects

The clinical appearance of PIR can vary; in cases where the resorbed cavity is exposed after tooth eruption is complete, the lesion often appears empty or filled with a structure consisting mostly of soft dentin (Rutar, 1997).

Pre-eruptive intracoronal resorptions that occur before eruption are often not recognized until the tooth erupts and are easily misdiagnosed because they resemble caries. Intracoronal resorptions are typically observed as prominent radiolucencies in the dentin in the immediate vicinity of the enamel-dentin junction and are often detected incidentally on the radiograph of an unerupted tooth (Elbay et al., 2015).

Figure 2: Intraoral view of PIR



Reference: (Elbay et al., 2015)

Treatment of PIR

From a clinical perspective, an accurate diagnosis is very important in treatment of PIR defects. After eruption of the affected teeth, a caries-like lesion may appear with the presence of any enamel microporosity, defect or deep fissures forming an entryway into the defect for oral microorganisms (Grundy et al., 1984). The clinical management of these cases is complex in terms of the stage of root development in newly erupted teeth, the need for treatment of the pulp, the possible loss of pulp vitality, the lifespan of a non-viable tooth and, in some cases, the true value of retaining a tooth whose early loss may not be critical to overall arch form and occlusal function (Al-Batayneh & AlTawashi, 2020b).

The clinical management of PIR defects depends on the size of the lesion and the rate of progression of the lesion at the time of detection in relation to the expected time until the affected tooth emerges into the oral cavity and erupts to the occlusal level, to determine whether the lesion is growing or has reached the pulp (Rutar, 1997). Other factors affecting the treatment of PIR defects include the rate of progression of the lesion as assessed from serial radiographs, the patient's behaviour, age, cooperation for routine examination and the need to preserve the tooth (skeletal relationship, spacing/complexity, hypodontia and supernumerary teeth) (Moskovitz & Holan, 2004). Therefore, after lesion diagnosis, the timing of intervention should be determined based on periodic radiographs. The rate of progression of lesions may vary even on different teeth in the same individual and it has been recommended that the initial abnormality should require annual follow-up of other unerupted teeth. Therefore, observation of the lesion to determine its behaviour is important to decide on definitive treatment. In the literature, different follow-up intervals were reported for each case report and the duration ranged from 10 months to 5 years (Hata et al., 2007; Moskovitz & Holan, 2004).

The literature generally recommends immediate treatment in large lesions where the tooth is not close to eruption, in order to stop the progression of the resorptive process and prevent its penetration of the dental pulp (Holan et al., 1994). When the decision is made to treat the tooth before eruption, a mucosal flap is raised and the occlusal intact enamel and tissue filling the defect are removed by curettage. This procedure can be performed under conscious sedation or general analgesia if difficulty in managing patient behaviour is anticipated; however, it is not always necessary as pediatric patients may be old enough and/or co-operative enough to tolerate the procedure under local anaesthesia (Spierer & Fuks, 2014). The use of hand instruments is contraindicated in the removal of resorptive tissues and should be considered when the risk of pulpal exposure is high. Pulp exposures can be successfully treated by direct pulp capping with calcium hydroxide and tooth eruption can proceed smoothly (Davidovich et al., 2005). Recent studies have shown that MTA and Biodentine have superior properties compared to calcium hydroxide in the treatment of vital pulp and can also be recommended for direct pulp capping of these teeth (Brizuela et al., 2017; Paula et al., 2018). Coronal sealing is important to limit the extent of resorption and prevent contamination of tissues and underlying dentin following tooth eruption. The use of glass ionomer cement is recommended as a restorative material for teeth after surgical exposure due to its documented benefits such as adhesive properties, less moisture sensitivity, rapid hardening, high viscosity, easy handling and fluoride release into the cavity (Klambani et al., 2005). The use of amalgam or zinc oxide eugenol as a restorative material has also been reported to be successful in such cases (Grundy et al., 1984; Wood & Crozier, 1985).

If the lesion is large or causes symptoms such as pain, swelling or fistula, extraction of the affected tooth may be the right treatment and orthodontic treatment may be required afterwards. The

ideal time for extraction is just before or just after the tooth erupts, so that the procedure is simple and the pulp is not infected by oral pathogens (Klambani et al., 2005).

Conclusions

Accurate and careful examination of radiographs of unerupted teeth is crucial for the early diagnosis and treatment of PEIR defects. The most acceptable theory for the cause of PIR is that coronal resorption, most likely by invasion of dentin by resorptive cells following interruption of crown formation, may be caused by local factors such as ectopic position of the affected tooth. Invasive lesions should be treated promptly by surgical exposure of the defect if the tooth is to be preserved. We recommend long-term prospective clinical studies to elucidate the aetiology of the lesions, factors influencing their progression, effects on neighbouring teeth and long-term prognosis after treatment.

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SURGICAL MANAGEMENT OF LESIONS ASSOCIATED WITH UNERUPTED OR ERUPTING TEETH IN CHILD AND ADOLESCENT PATIENTS

OLGUN TOPAL[1]

INTRODUCTION

Tooth eruption is a complex biological process involving the coordinated action of cellular and molecular mechanisms that guide the movement of a developing tooth from its formation site within the alveolar bone to its final functional position in the oral cavity. Whilst this process is generally without incident, various pathological conditions have the capacity to disrupt normal eruption patterns, leading to the development of lesions associated with unerupted or erupting teeth. These lesions can range from benign cysts and hamartomas, such as dentigerous cysts and odontomas, to more aggressive entities like odontogenic keratocysts (Crasnean et al., 2023, p. 335); (Hariadi et al., 2023, p. 1058–1063). Early diagnosis and appropriate surgical intervention are crucial to prevent complications such as bone resorption, infection, malocclusion, and

[1] Dr. Öğretim Üyesi, Afyonkarahisar Sağlık Bilimleri Üniversitesi, Diş Hekimliği Fakültesi, Ağız, Diş ve Çene Cerrahisi Anabilim Dalı, Orcid: 0000-0003-3550-8739

in severe cases, destruction of adjacent anatomical structures (Tkaczuk et al., 2015, p. 834-839).

The surgical management of lesions associated with unerupted or erupting teeth in paediatric and adolescent patients presents unique challenges due to factors such as ongoing craniofacial growth, the presence of developing dentition, and the need for minimally invasive approaches to preserve future functional and esthetic outcomes. The treatment modality is selected based on the nature, size, and location of the lesion, as well as the age and overall health status of the patient. Surgical interventions encompass a range of procedures, including marsupialization, enucleation, curettage, excision, and in more extensive cases, resection and reconstructive procedures (Padmakumar et al., 2015, p. 532-536).

The objective of this chapter is to provide a comprehensive overview of the surgical methodologies used in managing such lesions in young patients. The discussion covers indications for surgical intervention, preoperative evaluation, lesion-specific surgical techniques, potential complications, and postoperative care strategies. Achieving optimal outcomes while minimizing morbidity and preserving the integrity of the dentition and surrounding structures requires a thorough understanding of these aspects.

INDICATIONS FOR SURGERY AND PREOPERATIVE EVALUATION

Surgical intervention for lesions associated with unerupted or erupting teeth is indicated when these lesions threaten normal dental development, adjacent structures, or overall oral function. Persistent delayed eruption—defined as the failure of teeth to erupt within the expected timeframe due to obstruction by cystic lesions, supernumerary teeth, or developmental anomalies—is a primary indication. Radiographic evidence of pathology, such as radiolucent or radiopaque lesions, suggests the presence of odontogenic cysts,

tumors, or other abnormalities requiring intervention. The progressive enlargement of cystic or tumoral lesions may lead to cortical bone thinning, resorption, or facial asymmetry, necessitating timely surgical management. Additionally, lesions causing pain, infection, or discomfort require prompt intervention to alleviate symptoms and prevent complications. Moreover, space-occupying lesions that induce malocclusion or displacement of adjacent teeth should be surgically removed early to prevent functional and aesthetic disturbances (Padmakumar et al., 2015, pp. 532–536; Crasnean et al., 2023, p. 335).

A comprehensive preoperative evaluation is essential to ensure optimal surgical outcomes and minimize complications. A thorough clinical examination should include an assessment of facial symmetry, swelling, and signs of infection. Additionally, palpation of the lesion is necessary to determine its consistency and the mobility of the involved teeth. Occlusal and soft tissue evaluations are crucial in assessing the lesion's functional impact.

Radiographic imaging plays a pivotal role in preoperative planning. Panoramic radiography provides a broad view of the maxillofacial region, enabling an assessment of lesion extent and its relationship with vital structures. Cone-beam computed tomography (CBCT) offers three-dimensional visualization of the lesion's boundaries, internal structure, and potential effects on adjacent teeth and bone (Caminiti et al., 2020, pp. 814–821). Furthermore, periapical radiographs may be valuable for evaluating periapical involvement and root integrity.

Recognizing the significance of histopathological and laboratory evaluations is essential, as they constitute integral components of the preoperative assessment protocol. In cases of cystic lesions, aspiration biopsy may be necessary to distinguish between odontogenic and non-odontogenic entities. Routine blood

tests are warranted when systemic involvement or underlying conditions affecting bone metabolism are suspected.

Effective treatment planning requires a multidisciplinary approach, involving collaboration among pediatric dentists, orthodontists, and oral and maxillofacial surgeons to ensure comprehensive patient care. Factors such as the patient's age, skeletal development, and future orthodontic needs must be carefully considered when determining the most appropriate surgical approach (Tkaczuk et al., 2015, pp. 834–839).

A thorough preoperative evaluation is paramount for optimizing patient outcomes and minimizing the risks associated with surgical intervention. This section establishes the foundation for understanding the rationale behind surgical management, which will be further explored in subsequent sections covering specific surgical techniques.

SURGICAL TECHNİQUES AND MANAGEMENT APPROACHES

The surgical management of lesions associated with unerupted or erupting teeth necessitates a systematic and individualised approach. A range of techniques is employed, with the choice of technique depending on the type of lesion, its size, and its location. The aim of the techniques is to optimise both the functional and aesthetic outcomes, while minimising the risk of recurrence and preserving surrounding structures.

Hyperplastic Dental Follicle

Hyperplastic dental follicles, though generally benign, may necessitate surgical intervention if they exhibit excessive enlargement, impinge on adjacent structures, or cause delayed eruption. The standard surgical approach is enucleation, which involves the careful removal of follicular tissue with minimal trauma

to surrounding bone and teeth. Curettage is performed following enucleation to eliminate any residual soft tissue remnants and reduce the risk of recurrence (Neville et al., 2023). Postoperative management includes routine monitoring for eruption progress and potential recurrence.

Enucleation: Enucleation involves the complete removal of the cystic lesion along with its epithelial lining, thereby minimizing the risk of recurrence. The procedure begins with the elevation of a mucoperiosteal flap to access the lesion, followed by meticulous dissection and removal using sharp curettes. Careful handling is essential to avoid damage to adjacent structures, including nerves and developing tooth buds. After excision, the cavity is irrigated with sterile saline, and hemostasis is achieved. Whenever possible, primary closure of the surgical site is performed; however, in certain cases, an open-packing approach may be employed to facilitate secondary healing (Couto et al., 2022, pp. 1–5).

Curettage: Curettage is typically performed following enucleation to ensure the complete removal of any residual epithelial remnants, thereby minimizing the risk of recurrence. A sharp curette is used to meticulously scrape the cystic lining from the bony walls of the defect, ensuring thorough eradication.

Eruption Cyst

Eruption cysts, also referred to as eruption hematomas when containing blood, are a type of soft tissue cyst that forms over a tooth on the verge of erupting through the gingiva. Clinically, they present as dome-shaped, translucent swellings on the alveolar ridge, typically in children under 10 years of age (Bilodeau & Hunter, 2021, pp. 71–84). If bleeding occurs within the cyst, it may appear bluish or purple. Small eruption cysts are generally painless and do not significantly delay tooth eruption. Most cases resolve spontaneously as the tooth erupts into the oral cavity, making

observation the primary management approach. In the absence of symptoms such as discomfort or infection, no surgical intervention is required, as the cyst typically ruptures naturally with tooth eruption. Parents should be reassured and advised to maintain good oral hygiene in the affected area.

However, if the cyst becomes painful, infected, or persists beyond the expected eruption period, surgical intervention may be indicated. This minor procedure, performed under local anesthesia, involves creating a cruciate incision or excising a small window from the cyst's roof to drain its fluid contents. This exposure facilitates normal tooth eruption. The procedure, often referred to as "unroofing" of the eruption cyst, is minimally invasive. Sutures are generally unnecessary, though a single suture may be placed if excess tissue flaps are created during the excision.

Postoperatively, gentle rinsing with chlorhexidine or saline helps maintain cleanliness and prevent secondary infection. Children typically experience minimal postoperative discomfort, which can be managed with over-the-counter analgesics if needed. Healing occurs as the mucosa re-epithelializes over the erupting tooth. In summary, surgical management of eruption cysts is rarely required, and when performed, it is a straightforward procedure with rapid resolution (Nagaveni et al., 2011, pp. 148–151).

Dentigerous Cyst

A dentigerous cyst is defined as a developmental odontogenic cyst that originates from the accumulation of fluid between the crown of an unerupted tooth and the surrounding reduced enamel epithelium. It is characterised by its attachment at the cervical region of the tooth and its capacity to enclose the crown. Dentigerous cysts are among the most prevalent jaw cysts in paediatric patients, constituting approximately 20% of jaw cysts in general (Bilodeau & Hunter, 2021, p. 71-84). These cysts most frequently involve

permanent molars or canines, for example the mandibular first molar or maxillary canine in younger patients, and third molars in older adolescents. As dentigerous cysts have the potential to expand and displace teeth, or even cause pathologic fractures in extreme cases, treatment is indicated upon diagnosis. Furthermore, while the cyst lining is generally benign, there exists a potential for neoplastic change over time (e.g., to ameloblastoma), thus necessitating definitive management (Guerrisi et al., 2007, p. 180-185). The two main surgical approaches are enucleation (complete removal of the cyst lining) and marsupialization (decompression). The choice of approach depends on factors such as the size of the cyst, the age of the patient, and the importance of the associated tooth (Muzio et al., 2017, p. 128-138).

Enucleation and Extraction: Conventional management of a dentigerous cyst involves surgical enucleation of the cyst along with removal of the unerupted tooth involved. This approach is frequently regarded as the treatment of choice for smaller cysts or in cases where the involved tooth is a supernumerary or a third molar of limited utility The surgical procedure is performed under appropriate anaesthesia (typically general anaesthesia or deep sedation for paediatric patients) and involves the elevation of a mucoperiosteal flap to expose the cyst. The removal of the overlying bone is then undertaken with meticulous burring. The cyst is then enucleated by separating and removing the entire cyst sac from the surrounding bone, which should include the thin epithelial lining. In cases where the tooth is attached to the cyst, it is imperative to extract the tooth along with the cyst to ensure a comprehensive removal procedure. The cavity is then irrigated and examined for any remnants. It is widely accepted that enucleation is a curative procedure; the recurrence of a dentigerous cyst after complete removal is exceptionally rare. However, it should be noted that this procedure does carry with it the potential loss of a developing tooth.

In the case of a child, this may require future orthodontic or prosthetic management to address the absence of the tooth (Li et al., 2014, p. 795-800).

Marsupialisation: Marsupialization is a conservative surgical approach used in the management of large odontogenic cysts, particularly when the preservation of the associated tooth is desirable. This technique is indicated for cases Wheel complete enucleation may pose risks to adjacent structures or when maintaining the integrity of developing teeth is a priority.

The procedure involves creating an opening in a portion of the cyst wall, typically by removing a segment of bone and cyst lining, resulting in a 5–10 mm opening. The cyst contents are then evacuated. The margins of the cyst lining may be sutured to the oral mucosa to establish a permanent stoma, or a drain or stent may be placed to maintain patency. Various techniques are employed to prevent premature closure of the opening, including the insertion of a plastic tube, a custom-made acrylic obturator, or a modified removable appliance designed to function as a marsupialization stent. Postoperative care includes daily irrigation of the cyst cavity to promote cleanliness and prevent secondary infection.

Marsupialization is effective in reducing cyst size while preserving the associated tooth. The likelihood of spontaneous tooth eruption is influenced by factors such as patient age and the initial position of the impacted tooth. Younger patients and cases with shallow impaction generally have higher success rates. If spontaneous eruption does not occur within a few months, orthodontic traction may be applied by bonding a bracket to the exposed crown to facilitate proper positioning. In some cases, a secondary procedure (cystectomy) may be necessary to remove any residual cyst lining once the lesion has decreased in size and become more accessible.

This approach offers excellent long-term outcomes, minimizing the risk of recurrence while maintaining dental arch integrity and occlusion. However, marsupialization requires patient compliance and regular follow-up visits to monitor progress. Treatment duration is typically longer than enucleation, making individualized treatment planning essential. Periodic radiographic imaging is recommended to assess the resolution of the lesion and ensure normal development of the affected area (de Oliveira et al., 2024, p. 314).

Odontoma

Odontomas are the most common odontogenic tumors in pediatric patients and are classified as developmental hamartomas rather than true neoplasms. These lesions are generally asymptomatic and are often discovered incidentally when an unerupted tooth fails to emerge within the expected timeframe. Odontomas are classified into two primary types: **compound odontomas**, which consist of multiple small, tooth-like structures, and **complex odontomas**, which present as an irregular mass of dental tissues without distinct tooth morphology.

Although odontomas are benign and non-recurrent, surgical removal is typically recommended due to their potential to impede tooth eruption, induce cyst formation, and disrupt normal occlusion development. Retaining an odontoma in situ may result in space loss, malalignment of adjacent teeth, and secondary complications such as permanent dentition impaction.

The primary treatment for odontomas is surgical enucleation with curettage of the surrounding tissue. These lesions are typically well-encapsulated, allowing for complete removal with minimal difficulty. The procedure is performed under local anesthesia for small, accessible odontomas or general anesthesia for larger lesions or younger patients.

The surgical process involves elevating a full-thickness mucoperiosteal flap over the site, followed by careful removal of overlying bone using a surgical bur to expose the lesion. The odontoma is then meticulously separated from the surrounding bone and extracted. Compound odontomas typically "shell out" as discrete tooth-like structures, whereas complex odontomas may require meticulous dissection. The bony cavity is then gently curetted to ensure complete elimination of any residual odontogenic tissue.

Histopathological examination of all excised specimens is essential to confirm the diagnosis and rule out rarer odontogenic tumors. Following lesion removal, the flap is repositioned and sutured, and postoperative healing is monitored. Odontoma excision is generally associated with minimal morbidity, and most patients recover uneventfully within a few weeks.

After odontoma removal, orthodontic intervention is often initiated to facilitate the eruption of impacted teeth. If the impacted tooth is sufficiently exposed, an orthodontic bracket and traction chain may be attached to guide eruption. If spontaneous eruption does not occur within several months, active orthodontic traction may be applied. Close monitoring is necessary to determine whether additional intervention, such as extraction or prosthetic replacement, is required.

Early detection and removal of odontomas have been shown to support normal occlusal development and reduce the need for extensive orthodontic or prosthetic intervention (Guerrisi et al., 2007, pp. 180–185).

Odontogenic Keratocysts

Odontogenic keratocysts (OKCs) are developmental cysts that originate from dental lamina remnants and are distinguished by their aggressive behavior and high recurrence rates. Previously

classified as keratocystic odontogenic tumors due to their neoplastic potential, the 2017 WHO classification re-designated them as odontogenic cysts.

OKCs can occur at any age, though they most commonly present in the second and third decades of life, with approximately 17% of cases occurring in patients under 20 years of age. They are relatively rare in children under 10 years old. These cysts exhibit a strong predilection for the mandible, particularly the posterior body and ramus region.

Radiographically, OKCs typically present as well-defined radiolucent lesions, often unilocular but occasionally multilocular in larger cases. Unlike other odontogenic cysts, OKCs tend to grow along the internal aspect of the jaw, resulting in minimal cortical expansion. While many OKCs are discovered incidentally on radiographs, symptomatic cases may present with pain, swelling, or drainage, particularly if secondary infection occurs. The presence of multiple OKCs in adolescents should raise suspicion for Nevoid Basal Cell Carcinoma Syndrome (Gorlin Syndrome).

Given their aggressive nature and high recurrence rates, OKCs require meticulous enucleation with curettage, often supplemented by chemical cauterization with Carnoy's solution to reduce the likelihood of recurrence. In cases involving extensive bone involvement, segmental resection with subsequent reconstruction may be necessary to achieve definitive management (Pejović et al., 2016, pp. 129–134).

Enucleation with Curettage: This is considered a primary treatment option for solitary lesions in younger patients. A mucoperiosteal flap is reflected, and the entire cyst lining is meticulously enucleated. The OKC lining is characterised by its thin (6–10 cell layers thick) and fragile nature, necessitating meticulous technique to ensure its removal in a single piece. The surrounding

bony walls are then curetted to remove any epithelial remnants or daughter cysts. Some surgeons utilise peripheral ostectomy, defined as the removal of 1–2 mm of bone, to mitigate the risk of recurrence. The recurrence rate, in the absence of adjunctive treatment, ranges from 3% to 60%. In paediatric patients, enucleation allows for the preservation of teeth and jaw continuity, which is a significant advantage over resection (Li et al., 2014, p. 795-800).

Enucleation with Chemical or Cryotherapy Adjuncts: In order to enhance outcomes, adjunctive therapies are frequently administered post-enucleation. Carnoy's solution, a chemical therapy, is used to minimise recurrence rates in aggressive odontogenic keratocysts. This solution, a fixative composed of ethanol, chloroform, acetic acid, and ferric chloride, is applied to the bony cavity for 3–5 minutes following enucleation and curettage. The chemical cauterization effect of the solution destroys residual epithelial cells, thereby reducing the likelihood of cyst recurrence. Following application, the cavity is thoroughly irrigated with sterile saline to neutralise any residual solution. Despite its efficacy, this technique must be employed with caution, particularly in proximity to neurovascular structures, due to its potential for toxicity.

Segmental resection: Segmental resection is a radical approach reserved for extensive lesions that compromise the structural integrity of the jaw. This procedure involves the complete removal of a segment of the jaw, including both the lesion and surrounding bone. Indications for segmental resection include aggressive odontogenic tumours and recurrent keratocysts that have eroded significant portions of bone. Post-operative reconstruction is typically necessitated by the procedure, and is commonly achieved through the utilisation of bone grafts or alloplastic materials, with the aim of restoring both the aesthetic appearance and the functionality of the affected region. This procedure necessitates a multidisciplinary approach, often involving maxillofacial surgeons,

prosthodontists, and orthodontists to achieve optimal functional and esthetic outcomes (Crasnean et al., 2023, p. 335).

Buccal Bifurcation Cysts

Buccal bifurcation cysts are treated in a conservative manner with enucleation, ensuring that the affected tooth is preserved. In larger lesions, marsupialization may be performed to allow spontaneous resolution while maintaining normal anatomical structures. Appropriate surgical techniques, when employed systematically and based on evidence, can optimise outcomes for paediatric and adolescent patients with lesions associated with unerupted or erupting teeth. A comprehensive preoperative evaluation, meticulous surgical execution, and diligent postoperative care are integral to achieving long-term success, while minimising morbidity and preserving oral function (Pompura et al., 1997, p.215).

POSTOPERATIVE CARE

Postoperative care is of paramount importance in ensuring the success of surgical management for lesions associated with unerupted or erupting teeth in paediatric and adolescent patients. Meticulous postoperative care and long-term follow-up are essential components of a comprehensive postoperative care plan. Given the unique anatomical and developmental considerations in young patients, ensuring optimal healing while preventing complications is a priority.

Immediate Postoperative Care: Postoperative care commences immediately following the surgical procedure and encompasses pain management, infection control, and soft tissue healing. Analgesics such as ibuprofen or acetaminophen are typically prescribed to manage discomfort. In cases where there is a high risk of infection, prophylactic antibiotics may be administered, particularly following procedures such as enucleation or segmental

resection. Postoperative bleeding is managed through gentle pressure application with sterile gauze, and patients are advised to avoid vigorous rinsing for the first 24 hours.

Soft tissue healing is a critical component of postoperative recovery. Patients should adhere to a stringent oral hygiene regime, encompassing gentle rinsing with chlorhexidine mouthwash to prevent bacterial colonization. In cases of procedures such as marsupialization, where an open cavity remains, daily irrigation with saline is required to prevent premature closure and fluid accumulation. The removal of sutures is typically performed within 7 to 14 days, contingent on the extent of the surgical procedure.

Monitoring and Management of Postoperative Complications: Postoperative complications, including but not limited to swelling, infection, and delayed healing, are a potential outcome of paediatric oral surgeries. The management of postoperative oedema typically involves the application of cold compresses during the initial 24-hour period, followed by the administration of warm compresses to enhance circulation and reduce swelling. Infections, although uncommon with adequate antibiotic coverage, may present with erythema, purulent discharge, and persistent pain. In such cases, an antibiotic course is extended, and wound debridement may be required One of the most significant complications following surgical removal of odontogenic cysts, particularly odontogenic keratocysts, is recurrence. The recurrence rate varies depending on the lesion type and surgical technique. For instance, marsupialization has been observed to demonstrate a higher recurrence rate in comparison to enucleation, particularly when augmented by therapies such as Carnoy's solution. Consequently, close radiographic monitoring is imperative, with the recommendation of follow-up imaging at 6-month intervals during the first two years post-surgery (Padmakumar et al., 2015, p. 532-536).

CONCLUSIONS AND FUTURE DIRECTIONS

The surgical management of lesions associated with unerupted or erupting teeth in pediatric and adolescent patients requires a multidisciplinary approach, integrating expertise from oral and maxillofacial surgery, pediatric dentistry, orthodontics, and pathology. The complexity of these cases necessitates individualized treatment planning, focusing on lesion eradication, functional and esthetic rehabilitation, and long-term patient monitoring. While current surgical techniques—such as marsupialization, enucleation, curettage, and resection—provide effective management options, advancements in surgical and diagnostic technologies continue to offer promising avenues for improving patient outcomes.

A significant development in this field is the introduction of minimally invasive surgical techniques, such as guided bone regeneration and laser-assisted cyst enucleation, which aim to reduce surgical morbidity and enhance healing. The primary objective of these techniques is to preserve the structural integrity of surrounding anatomical structures, a critical consideration in growing patients, where excessive tissue loss may adversely impact long-term dentofacial development. Further research is needed to establish standardized protocols and evaluate long-term efficacy (Padmakumar et al., 2015, pp. 532–536).

Another emerging area is the integration of biological and regenerative approaches in the management of odontogenic lesions. Stem cell therapy and platelet-rich fibrin (PRF) applications have demonstrated promising results in promoting bone regeneration following cystic lesion removal. PRF has been shown to accelerate soft and hard tissue healing by promoting angiogenesis and collagen deposition, potentially leading to reduced recovery times and improved outcomes. Future investigations into biomaterial-based

scaffolds and growth factor applications could further revolutionize pediatric oral surgery.

The widespread implementation of cone-beam computed tomography (CBCT) and digital pathology has significantly improved the accuracy of lesion characterization. CBCT provides high-resolution, three-dimensional visualization, enabling precise assessment of lesion margins, cortical bone involvement, and proximity to vital structures. This imaging modality has enhanced preoperative planning, reduced intraoperative risks, and optimized surgical outcomes.

Additionally, advancements in molecular pathology, such as the utilization of immunohistochemical markers (e.g., Ki-67 and p53), have facilitated differentiation between aggressive and indolent lesions, allowing for more personalized treatment strategies (Caminiti et al., 2020, pp. 814–821).

Given the high recurrence rates associated with odontogenic keratocysts and aggressive tumors, structured follow-up protocols are essential. Periodic radiographic evaluations and clinical monitoring can aid in the early detection of recurrence, enabling timely intervention.

Recent investigations are also exploring the potential of artificial intelligence (AI) and machine learning algorithms in enhancing lesion detection and risk assessment. AI-driven models may improve early diagnosis, personalized treatment planning, and postoperative surveillance, potentially transforming clinical decision-making (Crasnean et al., 2023, p. 335).

Interdisciplinary collaboration remains fundamental to successful treatment outcomes. Pediatric dental specialists, orthodontists, and maxillofacial surgeons play a crucial role in comprehensive patient care, addressing both surgical and functional rehabilitation. Orthodontic intervention is particularly critical in

cases where tooth eruption pathways are altered due to cystic or tumoral obstructions. Coordinated treatment planning between orthodontists and surgeons has been shown to enhance occlusal harmony and long-term stability (Tkaczuk et al., 2015, pp. 834–839).

The future of surgical management for lesions associated with unerupted or erupting teeth is dependent on ongoing advancements in minimally invasive techniques, regenerative therapies, and diagnostic innovations. By leveraging these advancements, clinicians can optimize patient outcomes, minimize surgical morbidity, and ensure long-term functional and esthetic success. Interdisciplinary collaboration and continued research will be essential in shaping the next generation of treatment strategies, ultimately improving the quality of life for pediatric and adolescent patients affected by these conditions.

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LONG-TERM PROGNOSIS OF LESIONS ASSOCIATED WITH UNERUPTED OR ERUPTING TEETH IN CHILD AND ADOLESCENT PATIENTS

MEHMET FAİK YÜCEL[1]
OLGUN TOPAL[2]

The Tooth Eruption Process and Its Stages

The process of tooth eruption begins during the embryonic stage and extends through adolescence. In some cases, it may continue beyond this period, as observed with third molars. Any disruption in this process can result in teeth remaining impacted or experiencing delayed eruption. The development of primary teeth starts in the fourth month of intrauterine life from the dental lamina, which is intimately connected to the lingual side of the enamel organ, whereas permanent tooth development initiates during the bell stage.(Nanci, 2012, p. 352) Järvinen, Tummers, & Thesleff, pp. (2009, pp. 91–281) The stages of tooth eruption are as follows:

[1] Research assistant, Afyonkarahisar Sağlık Bilimleri University Faculty of Dentistry Department of Oral and Maxillofacial Surgery, Orcid: 0009-0009-4109-8327
[2] Assoc. Prof, Afyonkarahisar Sağlık Bilimleri University Faculty of Dentistry Department of Oral and Maxillofacial Surgery, Orcid: 0000-0003-3550-8739

Tooth Germ Formation: The tooth germ originates during the embryonic phase and continues its development within the bone.

Root Formation: As the tooth germ matures, the root begins to take shape.

Eruptive Movement: The tooth gradually migrates toward the oral cavity, passing through bone and surrounding soft tissues.

Establishment of Occlusal Position: The tooth achieves its final functional position by making contact with the opposing dentition.

The Importance of the Tooth Eruption Process

The tooth eruption process is crucial for the following reasons:

• Functional Development: Erupting teeth are essential for acquiring fundamental functions, such as chewing and speaking.

• Maxillofacial Complex Growth: The formation of tooth germs and the eruption process promote the development of jawbones and play a key role in shaping facial aesthetics.

• Dental Arch Formation: The proper eruption of teeth in their correct positions is vital for the formation and proper alignment of the dental arch.

• Psychosocial Effects: The timing and proper eruption of teeth positively influence children's self-confidence and social development.

Factors Influencing Prognosis Determination Lesions

The long-term prognosis of lesions related to impacted or erupting teeth is influenced by various factors. These include the characteristics of the lesion, the selected treatment method, the patient’s unique attributes, and any underlying medical conditions.

Below, the primary factors affecting the long-term prognosis of lesions associated with erupted or unerupted teeth are explained in detail.

Lesion Type and Behavior

• Non-Aggressive Lesions: Benign lesions such as dentigerous cysts, odontomas, and periapical granulomas typically exhibit a good prognosis after surgical treatment, with a low likelihood of recurrence.

• Aggressive Lesions: Lesions like ameloblastoma and odontogenic keratocyst are characterized by aggressive growth, a high recurrence rate, and the need for prolonged monitoring.

Lesion Size and Spread

• Small and Well-Defined Lesions: Lesions that are limited in size and clearly demarcated can typically be removed with ease through surgical intervention and are associated with a more favorable prognosis.

• Large and Extensive Lesions: Expansive lesions may infiltrate adjacent tissues, leading to potential structural damage. Surgical management can be more complex depending on the lesion's dimensions and its relationship with surrounding anatomical structures. In such cases, prognosis requires a more cautious evaluation.

Treatment Approach

• Surgical Treatment: Complete excision of the lesion significantly improves prognosis. However, inadequate surgical removal, lesion extension into surrounding tissues, or the presence of satellite cysts, as seen in odontogenic keratocysts, increases the risk of recurrence.

• Conservative Treatment: In certain cases, conservative treatment methods may be preferred; however, these approaches can be associated with a higher risk of recurrence.

Patient's Age and Overall Health Status

• Pediatric and Adolescent Patients: In patients undergoing growth and development, lesions can have a more pronounced impact on dental and maxillofacial development. Early intervention enhances long-term prognosis.

• Systemic Conditions: A patient's overall health status can influence their response to treatment and the healing process.

Monitoring and Follow-Up

• Consistent Follow-Up: Regular clinical and radiographic assessments post-surgery facilitate the early identification of recurrences, positively influencing the long-term outcome.

• Patient Adherence: The patient's commitment to both treatment and follow-up care is essential for ensuring a favorable prognosis.

Protection of the Tooth Germ

Preserving the tooth germ during the removal of the lesion allows the tooth eruption process to proceed, playing a significant role in maintaining long-term dental health.

Developmental Cysts and Prognosis

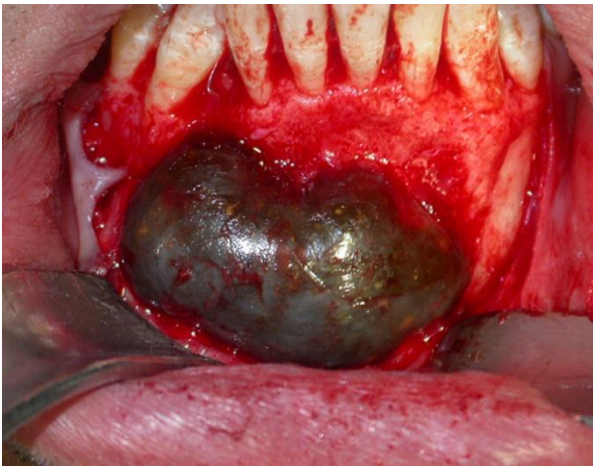
Developmental cysts of the jaws are a diverse group of lesions that arise from odontogenic or non-odontogenic epithelial remnants during embryogenesis. These cysts, which include dentigerous cysts, odontogenic keratocysts, and lateral periodontal cysts, among others, can present as incidental radiographic findings or manifest with clinical symptoms such as swelling, pain, or

displacement of adjacent teeth. While many developmental cysts follow a benign course, some exhibit aggressive behavior, with a tendency for recurrence or significant bone destruction. Understanding the classification, pathogenesis, and biological behavior of these cystic lesions is crucial for establishing an accurate diagnosis and selecting the most appropriate treatment approach. Moreover, long-term prognosis varies depending on factors such as lesion type, size, location, and response to surgical or conservative management. This chapter provides a comprehensive overview of developmental cysts, discussing their classification, clinical and radiographic characteristics, histopathological features, and prognosis to aid clinicians in optimal patient management.

Dentigerous Cyst (Follicular Cyst)

Clinical Characteristics: It is one of the most commonly observed cysts in the jaws.(Helm & Seidler, 1974, pp. 122–129; Lysell, Magnusson, & Thilander, 1962, pp. 217–234; Sivapathasundharam, 2020, pp. 5–260) Typically asymptomatic. As the cyst enlarges, symptoms such as swelling, delayed tooth eruption, or displacement of adjacent teeth may develop.(Daley, Wysocki, & Pringle, 1994, pp. 80–286; Regezi, Sciubba, & Jordan, 2011, pp. 32–326)

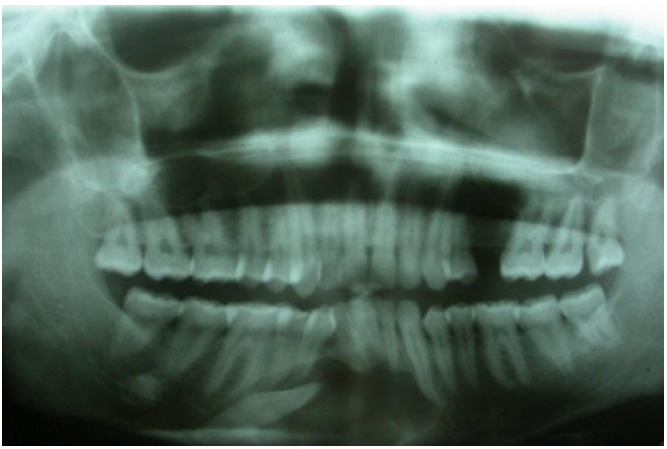
Dentigerous Cyst



(Rajae & Karima, 2021)

Radiographic Findings: A well-demarcated, unilocular radiolucent area surrounding the crown of an unerupted tooth, extending from the cementoenamel junction.(Brown, Berkman, Cohen, Kaplan, & Rosenberg, 1982, pp. 30–627)

Dentigerous Cyst



(Rajae & Karima, 2021)

Histopathology: Consists of a thin epithelial lining and fibrous connective tissue.(Gorlin, 1957, pp. 84–271)

Key Features: Most frequently associated with impacted third molars and canines. The recurrence risk is minimal following complete excision.

Prognosis: Dentigerous cysts generally heal completely following surgical intervention. Complete enucleation of the cyst and preservation of the tooth germ are essential for achieving long-term success.(Daley & Wysocki, 1995) The recurrence rate is low (5-10%). However, incomplete cyst removal or injury to the tooth germ can increase the risk of recurrence. It is advisable to perform regular radiographic evaluations during the first 1-2 years after surgery. Early identification of recurrence enables prompt intervention.

Eruption Cyst

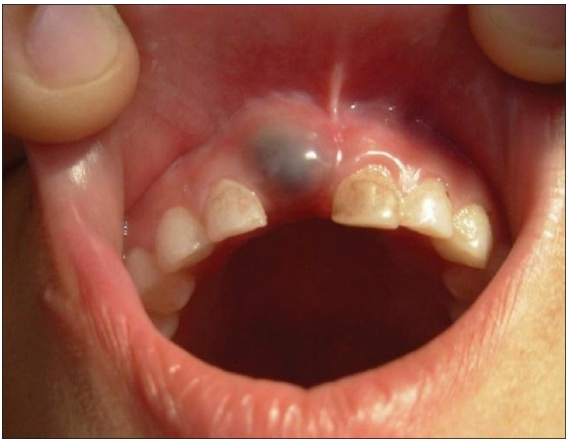
Clinical Features: Appears as a soft, bluish-purple swelling over erupting teeth.(Nunn, 1993, pp. 207–209)

Radiographic Findings: Typically does not present radiographic evidence due to its location within soft tissue.

Histopathology: Contains a thin epithelial lining and inflammatory cells.

Key Features: Generally transient and resolves spontaneously as the tooth erupts. Surgical intervention may be required in symptomatic cases.(Bansal, Kumari, Asrani, & Yadav, 2022)

Eruption cyst



(Nagaveni, Umashankara, Radhika, & Maj Satisha, 2011)

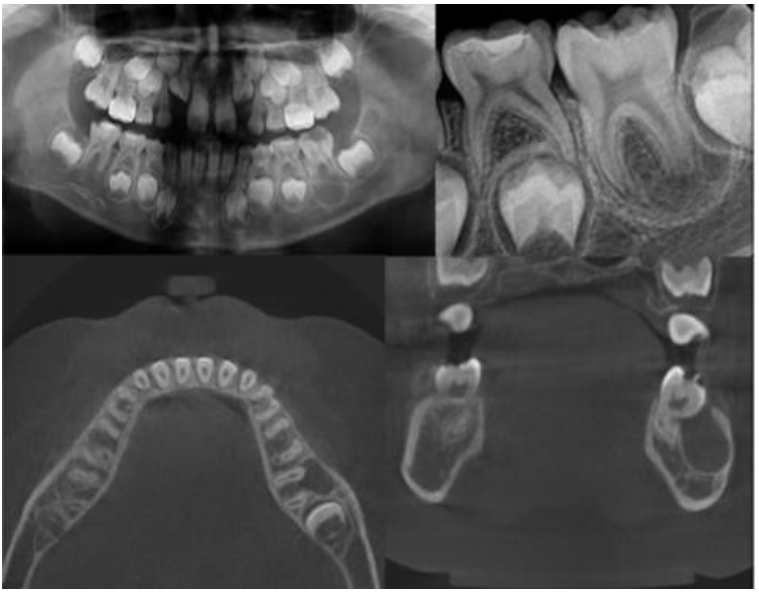
Prognosis: Eruption cysts typically resolve on their own as the tooth erupts. Surgical treatment is rarely necessary. (Sivapathasundharam, Biswas, & Preethi, 2019)The risk of recurrence is low, and the cyst does not recur once the tooth eruption process is complete.(Nagaveni et al., 2011) Follow-up is usually not required. However, if there are delays in eruption or complications arise, regular follow-up every 6 months or 1 year is recommended.

Buccal Bifurcation Cyst

Clinical Features: Buccal bifurcation cyst is a cyst that usually develops in the buccal region of mandibular first or second molars at the bifurcation of the roots of the tooth. It is a rare cyst. It is usually diagnosed in children aged 5-13 years. Painless swelling (the most common symptom).(Pompura, Sándor, & Stoneman, 1997) It can cause teeth to delay eruption or erupt in an abnormal position. Rarely accompanied by infection or pain.

Radiographic Findings: Panoramic or periapical radiographs show a well-circumscribed, radiolucent (dark) lesion in the furcation zone of the roots of the tooth. There may be resorption or displacement of the roots of the tooth.

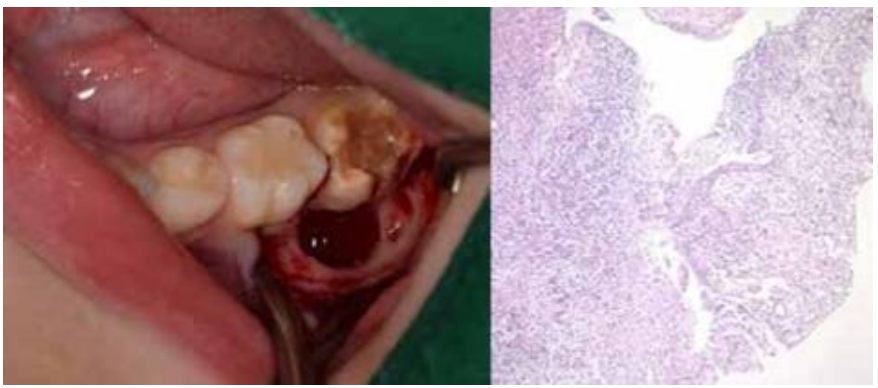
Buccal bifurcation cyst



(Kim, Nam, Kim, & Choi, 2018)

Histopathology: The cyst wall consists of a thin epithelial lining and fibrous connective tissue. Inflammatory cells (lymphocytes, plasma cells) can be seen. Cyst fluid may be clear or yellowish.

Buccal bifurcation cyst



(Kim et al., 2018)

Prognosis: The prognosis after treatment is usually very good. It is rare, especially after surgical enucleation. With early diagnosis and treatment, the tooth can be preserved and resume normal function.(Philipsen, Reichart, Ogawa, Suei, & Takata, 2004; Pompura et al., 1997)

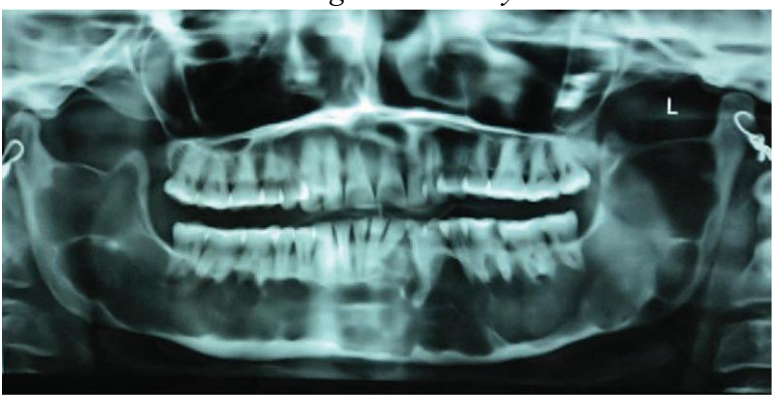
Odontogenic Keratocyst

Clinical Features: Often asymptomatic unless infection occurs. Radiographic Findings: Well-defined, multilocular or unilocular radiolucent area, often displaying a septated appearance.(Stoelinga, 2001)

Histopathology: Parakeratinized epithelial lining with basal cells arranged in a palisaded pattern.

Key Features: Recurrence risk is high due to the presence of dormant cysts (25-60%).(Stoelinga, 2001, pp. 14–25) The lesion exhibits aggressive behavior.(Vedtofte & Prætorius, 1979)

Odontogenic keratocyst



(Khan et al., 2019)

Prognosis: Due to its aggressive nature and high recurrence rate (25-60%), odontogenic keratocysts must be monitored carefully. Complete removal of the lesion and curettage of the surrounding bone are essential. In cases where the lesion is large or close to

critical anatomical structures (such as the inferior alveolar nerve), marsupialization can be performed to reduce the cyst size before further surgical intervention. (E. A. Al-Moraissi, Kaur, Gomez, & Ellis, 2023; Essam Ahmed Al-Moraissi et al., 2017, pp. 141–144)The surgical approach depends on the lesion's size and its proximity to surrounding structures. Due to the high recurrence risk, extended follow-up is necessary. Most recurrences occur within the first 5 years after treatment. Annual radiographic examinations are advised for the first 5 years after surgery. If recurrence is detected, additional surgical procedures may be needed.(Stoelinga, 2001, pp. 14–25)

Tumoral Lesions and Prognosis

Tumoral lesions associated with unerupted or erupting teeth in children and adolescents represent a significant clinical challenge due to their potential for aggressive behavior and impact on dental development. These lesions can range from benign odontogenic tumors to more aggressive neoplastic conditions, necessitating careful diagnosis and management. Early detection and appropriate intervention are crucial for minimizing complications, preserving normal occlusal function, and ensuring favorable long-term outcomes. This section will discuss the most common tumoral lesions encountered in this context, their clinical and radiographic features, as well as factors influencing their prognosis.

Other Lesions and Prognosis

Odontoma

Clinical Features: Typically asymptomatic, may obstruct tooth eruption or cause complete impaction of teeth.

Radiographic Findings: Radiolucent area with radiopaque foci.

Histopathology: Structures resembling dentin and enamel.(Maltagliati et al., 2020)

Key Characteristics: The most common odontogenic tumor; no risk of recurrence.(Thompson, 2021)

Prognosis Odontomas generally heal completely after surgical removal. If the tooth germ is preserved, the eruption process continues normally. The risk of recurrence is extremely low, and in fact, recurrence is rarely observed.(Owosho, Potluri, & Bilodeau, 2013; Thompson, 2021) No follow-up is typically required after surgery. However, if there is a delay in tooth eruption, regular follow-up appointments are recommended.

Conclusion

The long-term prognosis of lesions associated with unerupted or erupting teeth varies depending on the type of lesion and the treatment approach. Early diagnosis, appropriate treatment, and regular follow-up ensure favorable outcomes. Particularly for aggressive lesions, long-term monitoring is crucial for the early detection of recurrences.

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DIFFERENTIAL DIAGNOSIS OF LESIONS ASSOCIATED WITH UNERUPTED OR ERUPTING TEETH IN CHILD AND ADOLESCENT PATIENTS

BURCU GÜÇYETMEZ TOPAL[1]
BÜŞRA YILDIZ[2]
AYŞENUR KULA[3]

Introduction

Cystic lesions are frequently encountered in the jaws, with their incidence varying between children and adults. During childhood, the three-dimensional growth of the maxillofacial skeleton and the developmental process of odontogenesis influence the distribution of cysts. It is essential to have a thorough understanding of the clinical and radiographic findings of these pathologies, which are commonly observed during this period (Özkurt, B., & Bodrumlu, E. H.,2022).

[1] Assoc. Prof. Burcu Güçyetmez Topal, Afyonkarahisar Health Sciences University Department of Pediatric Dentistry,0000-0002-9932-9169.
[2] Res. Asst. Büşra Yıldız, Afyonkarahisar Health Sciences University Department of Pediatric Dentistry, 0000-0001-5438-3834.
[3] Res. Asst. Ayşenur Kula, Afyonkarahisar Health Sciences University Department of Pediatric Dentistry,0000-0002-9752-6765.

Certain pathologies associated with impacted teeth in pediatric patients may often be confused with one another. This may be due to the fact that cysts exhibit similar symptoms, are frequently asymptomatic, and children may have difficulty clearly expressing their complaints. Additionally, these lesions may not always present with well-defined borders in radiographic examinations, often displaying similar radiographic appearances. Since dental development is not yet complete in pediatric patients, diagnosing these lesions may be more challenging, and they may have a tendency to spread over a wider area. Moreover, the limitations of routine imaging techniques may further contribute to this difficulty (Neville et al., 2023; Shear & Speight, 2008).

The treatment approaches for two clinically similar pathologies in children may differ significantly. For instance, a pathology identified through radiographic imaging may be diagnosed as apical periodontitis, a radicular cyst, or a periapical granuloma. While root canal treatment is generally sufficient for the healing of apical granulomas, the management of radicular cysts, being true cysts, may necessitate additional apical surgery alongside root canal treatment (Lizio et al., 2018).

Accurate differential diagnosis is crucial for ensuring proper diagnosis and appropriate treatment planning. Inadequate or incorrect treatment planning may result in the mismanagement of the condition. A precise diagnosis can prevent the adverse effects of lesions associated with impacted teeth on maxillofacial development in pediatric and adolescent patients.

Differential Diagnosis of Hyperplastic Follicle

The hyperplastic follicle is a pathology commonly confused with a dentigerous cyst, especially in children during the mixed dentition period. The differential diagnosis includes eruption cyst, dentigerous cyst, odontogenic keratocyst, and ameloblastoma. It

should be remembered that the hyperplastic follicle does not cause displacement of surrounding teeth or bone expansion. Additionally, if the follicle shows asymmetric growth, the possibility of a dentigerous cyst should be considered (White & Pharoah, 2018).

	Hyperplastic Follicle (White & Pharoah, 2018)	**Eruption Cyst** (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Dentigerous Cyst** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)	**Ameloblastoma** (Neville et al., 2023)
Nature	Epithelial, resulting from excessive growth of the dental follicle.	Inflammatory, a cyst that prevents tooth eruption.	Odontogenic, develops from the dental follicle and surrounds the crown of the tooth.	Odontogenic, a rapidly growing and aggressive cyst.	Tumorous, invasive growth, can cause bone destruction.
Growth Pattern	Slow, limited growth.	Grows slowly, prevents the eruption of the tooth.	Grows slowly, expands around the tooth.	Aggressive growth, can cause bone destruction.	Slow but invasive growth, can cause bone destruction.
Age Range	Typically between 10-30 years.	Children and adolescents (7-15 years).	10-30 years.	Young adults (20-40 years).	30-50 years.
Location	Around the dental follicle, typically located in the mandible.	Around the tooth, especially in the vicinity of unerupted teeth.	Typically around the unerupted tooth, most often seen in the mandible.	Observed in the mandible or maxilla.	Observed in the mandible (most common) or maxilla.

	Hyperplastic Follicle (White & Pharoah, 2018)	**Eruption Cyst** (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Dentigerous Cyst** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)	**Ameloblastoma** (Neville et al., 2023)
Radiographic Appearance	A well-defined cystic area around the tooth. Radiolucent areas larger than 3mm may indicate a change in the dental follicle.	No significant radiographic findings due to being a soft tissue lesion, but a crescent-shaped radiolucent area may be seen at the apex of the unerupted tooth.	A wide cystic lesion around the tooth crown, can cause bone loss. For it to be diagnosed as a dentigerous cyst, the space between the tooth and follicle must be greater than 3mm.	Invasive, large cystic area with scalloped borders independent of the tooth roots.	Bone destruction, expansion, and tooth mobility are observed.
Histopathology	Thin epithelium, fibrotic tissue, basal cells.	Thin epithelial layer, inflammatory cells.	Keratinized epithelium, thin fibrous tissue, fluid content.	Multilayered thin epithelium, thickened basal cells.	Ameloblastic cells, cystic structures.
Treatment Approach	Generally self-resolving without treatment. In some cases, enucleation or	Surgical enucleation.	Surgical enucleation, wide surgery may be necessary.	Surgical enucleation, bone graft may be	Surgical treatment.

Hyperplastic Follicle (White & Pharoah, 2018)	**Eruption Cyst** (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Dentigerous Cyst** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)	**Ameloblastoma** (Neville et al., 2023)
marsupialization may be needed.			necessary in some cases.	

Differential Diagnosis of Eruption Cyst

An eruption cyst is an inflammatory-origin pathology associated with an unerupted tooth. Its differential diagnosis includes hemangioma, pyogenic granuloma, Bohn nodule, and amalgam pigmentation. The eruption cyst is located in the alveolar crest in the area of the tooth eruption and can be differentiated from hemangioma based on its location. It grows much slower compared to pyogenic granuloma(Aguilo et al., n.d.).

	Eruption Cyst (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Hemangioma** (Neville et al., 2023)	**Pyogenic Granuloma** (Cummings & Laskin, 2013)	**Bohn Nodule** (Neville et al., 2023)	**Amalgam Pigmentation** (Shafer et al., n.d.)
Nature	Benign, fluid-filled cyst with a blue-purple color that forms during tooth eruption.	Abnormal growth of blood vessels.	Rapidly growing benign tumor with high vascular density.	Soft tissue lesion, usually congenital.	Accumulation of amalgam material around the tooth filling.
Growth Pattern	Grows slowly, occurs before the tooth erupts.	Grows slowly, may grow rapidly in some cases.	Grows rapidly, typically associated with bleeding and inflammation.	Limited, usually grows slowly.	Slow, typically seen after long-term accumulation.
Age Range	Typically in childhood and adolescence.	Typically in childhood.	Can occur in any age group, especially common in young individuals.	Newborns and infants.	Adults.

	Eruption Cyst (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Hemangioma** (Neville et al., 2023)	**Pyogenic Granuloma** (Cummings & Laskin, 2013)	**Bohn Nodule** (Neville et al., 2023)	**Amalgam Pigmentation** (Shafer et al., n.d.)
Location	Around the tooth eruption area.	Often found on the face, mouth, and tongue.	Typically located in the gums.	More common in the buccal segment of the alveolar ridge.	Around the tooth, near amalgam fillings.
Radiographic Appearance	Due to being a soft tissue lesion, no significant radiographic findings, but a crescent-shaped radiolucent area may be seen at the apex of the unerupted tooth.	Prominent blood vessels with cystic structures around the vessels.	Blood vessels and dense granulation tissue with areas of bleeding.	Typically small, round, soft tissue cysts.	Amalgam particles can be seen around fillings.
Histopathology	Follicular epithelium, cystic area filled with fluid, may contain keratin.	Blood vessels, endothelial cells, and clotted blood.	Vascular density, granulation tissue, inflammatory cells.	Keratinized epithelium, minimal inflammation.	Amalgam particles, metallic pigments.

	Eruption Cyst (Aguilo et al., n.d.; Figueiredo et al., n.d.; Oliveira et al., 2018)	**Hemangioma** (Neville et al., 2023)	**Pyogenic Granuloma** (Cummings & Laskin, 2013)	**Bohn Nodule** (Neville et al., 2023)	**Amalgam Pigmentation** (Shafer et al., n.d.)
Treatment Approach	Generally monitored, may require surgical intervention in some cases.	Generally monitored, may require surgical intervention in some cases.	Surgical treatment (excision).	Monitoring, usually no surgical treatment required.	No treatment typically needed, cosmetic treatment may be performed.

Differential Diagnosis of Dentigerous Cyst

The differential diagnosis of dentigerous cyst includes radicular cyst, traumatic bone cyst, ameloblastoma, ameloblastic fibroma, adenomatoid odontogenic tumor, and odontogenic keratocyst. Radicular cysts are typically distinguished by the presence of a non-vital tooth (White & Pharoah, 2018).

	Dentigerous Cyst (White & Pharoah, 2018)	**Hyperplastic Follicle** (White & Pharoah, 2018)	**Radicular Cyst** (Oliveira et al., 2018)(Figueiredo et al., n.d.)	**Traumatic Bone Cyst** (Neville et al., 2023)	**Ameloblastoma** (Cummings & Laskin, 2013)	**Ameloblastic Fibroma** (White & Pharoah, 2018)	**Adenomatoid Odontogenic Tumor** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)
Nature	Benign cyst that prevents tooth eruption.	Benign, tooth-associated follicular structure.	Benign cyst that develops around the tooth root.	Bone lesion.	Benign tumor associated with the tooth.	Benign odontogenic tumor.	Benign odontogenic tumor.	Benign pathology containing keratin fluid.
Growth Pattern	Surrounds the crown of the tooth, forming a cyst around the tooth.	Grows slowly, surrounds the tooth, typically seen before eruption.	Grows slowly, typically develops around the tooth root.	Slow-growing, fluid-filled space within bone.	Grows slowly, cystic, solid structures.	Grows slowly, typically around the tooth root.	Grows slowly, develops around the tooth.	Grows slowly, contains dense cystic structure.

	Dentigerous Cyst (White & Pharoah, 2018)	**Hyperplastic Follicle** (White & Pharoah, 2018)	**Radicular Cyst** (Oliveira et al., 2018)(Figueiredo et al., n.d.)	**Traumatic Bone Cyst** (Neville et al., 2023)	**Ameloblastoma** (Cummings & Laskin, 2013)	**Ameloblastic Fibroma** (White & Pharoah, 2018)	**Adenomatoid Odontogenic Tumor** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)
Location	Surrounds the tooth root, observed with an impacted tooth.	Surrounds the tooth, in the development area.	Surrounds the tooth root, typically in the periapical region.	In jawbones, usually unilateral.	Located in the jawbones, especially in the molar regions.	Located in the jawbones, around teeth.	Develops around the tooth root, especially in the canine area.	Develops in jawbones, around teeth.
Age Range	10-30 years.	Children and young adults.	30-60 years	Young adults.	30-60 years.	Adult adolescence.	Adult adolescence.	20-40 years.
Radiographic Appearance	Well-corticated, well-	A smooth, well-defined follicular	Associated with a non-vital tooth.	Fluid-filled, sharply	Typically contains tooth remnants or	Cystic areas around the tooth root,	May show radiopaque areas within	Sometimes containing

	Dentigerous Cyst (White & Pharoah, 2018)	**Hyperplastic Follicle** (White & Pharoah, 2018)	**Radicular Cyst** (Oliveira et al., 2018)(Figueiredo et al., n.d.)	**Traumatic Bone Cyst** (Neville et al., 2023)	**Ameloblastoma** (Cummings & Laskin, 2013)	**Ameloblastic Fibroma** (White & Pharoah, 2018)	**Adenomatoid Odontogenic Tumor** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)
	defined borders.	area around the tooth.		defined areas within bone.	causes bone resorption.	dense fibrous tissue.	the cystic borders.	tooth remnants.
Histopathology	Contains fluid, keratin, and epithelial cells.	Limited collagen tissue, increased stromal cell activity.	Fluid-filled, lined with epithelial cells.	Soft tissue and fluid-filled area.	Odontogenic epithelium, ameloblast-like cells.	Collagen tissue, epithelial cells.	Ameloblast-like cells, fibrotic tissue.	Keratinized epithelium, sometimes odontogenic cells.
Treatment Approach	Typically monitored.	Typically monitored, sometimes	Surgical treatment.	Surgical treatment	Surgical treatment.	Surgical treatment.	Surgical treatment.	Surgical treatment.

Dentigerous Cyst (White & Pharoah, 2018)	**Hyperplastic Follicle** (White & Pharoah, 2018) surgical intervention needed.	**Radicular Cyst** (Oliveira et al., 2018)(Figueiredo et al., n.d.)	**Traumatic Bone Cyst** (Neville et al., 2023)	**Ameloblastoma** (Cummings & Laskin, 2013)	**Ameloblastic Fibroma** (White & Pharoah, 2018)	**Adenomatoid Odontogenic Tumor** (White & Pharoah, 2018)	**Odontogenic Keratocyst** (White & Pharoah, 2018)

Differential Diagnosis of Odontoma

Odontomas are composed of fully differentiated dental tissues, including mature enamel, dentin, cementum, and pulp, resulting from the differentiation of odontogenic tissues. Rather than true neoplasms, they are considered hamartomas and represent the most common odontogenic tumor. They can be distinguished from ossifying fibromas by their tendency to affect unerupted molar teeth and their higher prevalence in younger individuals. Odontomas may also be confused with periapical cemental dysplasia; however, the latter typically presents as multiple lesions located at the periapical regions of teeth, differentiating them from odontomas. Additionally, if an ossifying dysplasia is solitary and located in an edentulous area, it may be mistaken for an odontoma. However, osseous dysplasia tends to exhibit a wider and more sclerotic margin, whereas odontomas are characterized by well-defined cortical borders. The primary conditions considered in the clinical differential diagnosis of odontomas include adenomatoid odontogenic tumor, ameloblastoma, odontogenic cysts—particularly dentigerous cyst and keratocystic odontogenic tumor—ossifying fibroma, and fibrous dysplasia(White & Pharoah, 2018).

	Odontoma(Neville et al., 2023; Regezi et al., 2016)	**Adenomatoid Odontogenic Tumor**(Philipsen & Reichart, 2006)	**Ameloblastoma**(Wright & Vered, 2017)	**Odontogenic Cysts (Dentigerous Cyst, Odontogenic Keratocystic Tumor)**(Boffano & Gallesio, 2011)	**Ossifying Fibroma**(White & Pharoah, 2018)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
Nature	Developmental malformation (hamartoma).	Benign neoplastic tumor.	Malignant neoplastic tumor.	Cystic structures.	Benign neoplastic tumor.	Developmental malformation.
Growth Pattern	Slow, well-defined.	Slow, well-defined.	Aggressive, invasive growth tendency.	Expansile cystic growth.	Slow, expansile.	Expands within the bone.
Age Range	10–20 years	10–20 years	20–40 years	Any age	20–30 years	10–20 years
Location	Found in both the mandible and maxilla (more	More common in the maxilla (canine region).	More common in the mandible but also found in the maxilla.	Surrounds impacted teeth.	More common in the mandible	More common in the maxilla but

	Odontoma(Neville et al., 2023; Regezi et al., 2016)	**Adenomatoid Odontogenic Tumor**(Philipsen & Reichart, 2006)	**Ameloblastoma**(Wright & Vered, 2017)	**Odontogenic Cysts (Dentigerous Cyst, Odontogenic Keratocystic Tumor)**(Boffano & Gallesio, 2011)	**Ossifying Fibroma**(White & Pharoah, 2018)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
	common in the posterior region).				but also found in the maxilla.	also found in the mandible.
Radiographic Appearance	Radiopaque with tooth-like structures.	Radiolucent with calcifications.	Can be multilocular or unilocular, radiolucent.	Can be unilocular or multilocular, radiolucent.	Radiolucent, may contain opacities.	"Ground-glass" appearance.[5]
Histopathology	Contains dentin, enamel, and cementum.	Contains epithelial cell clusters and amyloid-like material.	Contains ameloblastic epithelium and mesenchymal cells.	Has a cystic epithelial lining.	Contains calcifications within fibrous tissue.	Contains immature bone trabeculae.

	Odontoma(Neville et al., 2023; Regezi et al., 2016)	**Adenomatoid Odontogenic Tumor**(Philipsen & Reichart, 2006)	**Ameloblastoma**(Wright & Vered, 2017)	**Odontogenic Cysts (Dentigerous Cyst, Odontogenic Keratocystic Tumor)**(Boffano & Gallesio, 2011)	**Ossifying Fibroma**(White & Pharoah, 2018)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
Treatment Approach	Surgical excision, low recurrence risk.	Surgical excision, low recurrence risk.	Surgical excision, high recurrence risk.	Curettage or excision.	Surgical excision.	Observation, surgery if necessary.

Differential Diagnosis of Odontogenic Keratocystic Tumor (OKT)

Its epithelial component demonstrates a growth potential similar to that of benign tumors, distinguishing it from other cystic lesions. Typically asymptomatic, OKT is less likely to cause root resorption and tooth displacement compared to dentigerous cysts. Due to its multilocular appearance, it may be misdiagnosed as ameloblastoma; however, ameloblastoma exhibits a more aggressive behavior. The primary conditions considered in the differential diagnosis of OKT include adenomatoid odontogenic tumor, ameloblastoma, dentigerous cyst, ossifying fibroma, and fibrous dysplasia. The most distinguishing features of OKT are its keratinized epithelial lining and high recurrence rate(Boffano & Gallesio, 2011; Neville et al., 2023; Philipsen & Reichart, 2006; White & Pharoah, n.d.; Wright & Vered, 2017).

	Odontogenic Keratocystic Tumor(Philipsen & Reichart, 2006)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)	**Ameloblastoma**(Boffano & Gallesio, 2011)	**Dentigerous Cyst**(Wright & Vered, 2017)	**Ossifying Fibroma**(Boffano & Gallesio, 2011)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
Nature	Benign, aggressive, high recurrence tendency, cystic tumor.	Benign, well-circumscribed, cystic tumor.	Benign but aggressive, invasive, high recurrence rate.	Benign, cystic structure surrounding the associated tooth.	Benign, contains calcifications, slow-growing.	Benign, composed of fibrous tissue and bone formations.
Growth Pattern	Slow-growing, locally invasive, sometimes multifocal.	Slow, well-circumscribed, expands around the tooth.	Rapid, expansile within bone, invasively affects surrounding tissues.	Expands as a cystic structure surrounding the associated tooth.	Slow, expansile growth within bone with calcifications.	Slow, asymmetric expansion within bone.
Age Range	Typically 20–40 years, but can occur at any age.	More common in young individuals (typically 10–20 years).	More common between 20–40 years, rarely seen in children.	Can occur at any age, more common in younger individuals.	Most common between 20–30 years.	More common in children and young adults.

	Odontogenic Keratocystic Tumor(Philipsen & Reichart, 2006)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)	**Ameloblastoma**(Boffano & Gallesio, 2011)	**Dentigerous Cyst**(Wright & Vered, 2017)	**Ossifying Fibroma**(Boffano & Gallesio, 2011)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
Location	More common in the mandible but can also occur in the maxilla.	More common in the maxilla, especially in the canine region.	More common in the mandible, particularly in the molar regions, but can also occur in the maxilla.	Mostly found in the mandible, surrounding impacted teeth.	More common in the mandible, typically in the premolar and molar regions.	More common in the maxilla, usually unilateral.
Radiographic Appearance	Radiolucent with scalloped borders, sometimes multilocular, expansile.	Radiolucent, well-defined, contains calcifications.	Radiolucent, multilocular, expansile, sometimes with a "honeycomb" appearance.	Radiolucent, cystic structure surrounding the associated tooth.	Radiolucent areas with internal calcifications.	Radiolucent with a "ground-glass" appearance, expansile.
Histopathology	Keratinized epithelium with a prominent basal cell layer, contains keratin.	Contains epithelial cell clusters and amyloid-like material.	Contains ameloblastic epithelium, mesenchymal cells, and multiple cystic structures.	Cystic epithelial lining, keratinized, with a distinct basal cell layer.	Contains calcifications and bone tissue within fibrous stroma.	Contains immature bone trabeculae and fibrous tissue.

	Odontogenic Keratocystic Tumor(Philipsen & Reichart, 2006)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)	**Ameloblastoma**(Boffano & Gallesio, 2011)	**Dentigerous Cyst**(Wright & Vered, 2017)	**Ossifying Fibroma**(Boffano & Gallesio, 2011)	**Fibrous Dysplasia**(Kramer & Pindborg, 1971)
Treatment Approach	Surgical excision, requires long-term follow-up due to high recurrence risk.	Surgical excision, low recurrence risk.	Surgical excision, high risk of expansion and recurrence.	Curettage or excision, marsupialization may be necessary.	Surgical excision, low recurrence risk.	Observation, surgical excision, or bone remodeling if necessary.

Differential Diagnosis of Buccal Bifurcation Cyst

Buccal bifurcation cyst is a benign odontogenic cyst that typically develops around the roots of teeth in the bifurcation area and is often asymptomatic. Its most distinguishing feature is its occurrence in the buccal bifurcation region of mandibular molars, where cyst expansion causes the crown of the affected tooth to tilt buccally. This characteristic displacement of molars differentiates it from other lesions. It should be distinguished from inflammatory periosteal lesions, such as periodontal abscesses or Langerhans cell histiocytosis, which may induce a periosteal response on the buccal surfaces of mandibular molars. Additionally, since it originates from the furcation area of the tooth rather than enveloping the crown, it can be differentiated from dentigerous cysts. In its differential diagnosis, odontogenic keratocystic tumor, dentigerous cyst, ameloblastoma, and adenomatoid odontogenic tumor are considered(White & Pharoah, 2018).

	Buccal Bifurcation Cyst(Wright & Vered, 2017)	**Odontogenic Keratocystic Tumor**(Neville et al., 2023; Philipsen & Reichart, 2006)	**Dentigerous Cyst**(Boffano & Gallesio, 2011)	**Ameloblastoma**(Cummings & Laskin, 2013)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)
Nature	A benign, odontogenic cystic lesion that develops around the roots of teeth.	A benign, aggressive, and recurrent-prone cystic tumor.	A benign cystic lesion, usually developing around an impacted tooth.	A benign but aggressive and invasive lesion with a high recurrence tendency.	A benign, well-defined, cystic tumor.
Growth Pattern	Slow-growing, expanding around the roots of teeth.	Slow-growing, locally invasive, and sometimes multifocal.	Slow-growing, expanding around an impacted tooth.	Rapid, causing bone expansion and invasive effects on surrounding tissues.	Slow, localized, and expands around the tooth.
Age Range	20-40 years	20-40 years	10-20 years	20-40 years, rarely seen in children.	10-20 years

	Buccal Bifurcation Cyst(Wright & Vered, 2017)	**Odontogenic Keratocystic Tumor**(Neville et al., 2023; Philipsen & Reichart, 2006)	**Dentigerous Cyst**(Boffano & Gallesio, 2011)	**Ameloblastoma**(Cummings & Laskin, 2013)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)
Location	Found in the mandible, particularly in the molar and premolar regions.	More common in the mandible but can also occur in the maxilla.	Primarily in the mandible, usually around impacted teeth.	More common in the mandible but can also occur in the maxilla, predominantly in the molar region.	Found in the maxilla, especially in the canine region.
Radiographic Appearance	Radiolucent, well-defined, expanding around the roots of teeth.	Radiolucent, well-defined, sometimes multilocular with areas of expansion.	Radiolucent, cystic lesion surrounding the tooth.	Radiolucent, multilocular, causing bone expansion, sometimes with a "honeycomb" appearance.	Radiolucent, well-defined, may contain calcifications.
Histopathology	Cystic structure containing epithelial cells and keratin around the tooth roots.	Keratinized epithelium with a prominent basal cell layer and keratin content.	Cystic epithelial lining with keratin content.	Ameloblastic epithelium, mesenchymal cells, and multiple cystic structures.	Epithelial cell clusters containing amyloid-like material.

	Buccal Bifurcation Cyst(Wright & Vered, 2017)	**Odontogenic Keratocystic Tumor**(Neville et al., 2023; Philipsen & Reichart, 2006)	**Dentigerous Cyst**(Boffano & Gallesio, 2011)	**Ameloblastoma**(Cummings & Laskin, 2013)	**Adenomatoid Odontogenic Tumor**(Wright & Vered, 2017)
Treatment Approach	Surgical excision, root resection may be required.	Surgical excision, long-term follow-up due to high recurrence risk.	Curettage, excision, sometimes marsupialization is required.	Surgical excision, high recurrence risk.	Surgical excision, low recurrence risk.

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