PROBLEMS OF DENTAL MEDICINE

Problems of Dental Medicine 51
DOI: 10.3897/pdm.51.e178424

Review Article

Periapical inflammation in the primary dentition and its impact on permanent teeth

Maria Kirilova[1]

1 *Department of Pediatric Dentistry, Faculty of Dental Medicine, Medical University-Sofia, Bulgaria*

Corresponding author: Maria Kirilova, Faculty of Dental Medicine, Sofia, Bulgaria; Email: m.kirilova@fdm.mu-sofia.bg

Received: 17 November 2025 ♦ **Accepted:** 15 December 2025 ♦ **Published:** 29 December 2025

Citation: Kirilova M (2025) Periapical inflammation in the primary dentition and its impact on permanent teeth. Problems of Dental Medicine: 1-5. doi: 10.3897/pdm.51.e178424.

Abstract

Introduction: Untreated carious lesions in primary teeth can negatively affect a child's quality of life, but they can also cause long-term adverse effects on permanent teeth. Untreated carious lesions in primary teeth progress to inflammation of the dental pulp, which is followed by the development of apical periodontitis.

Aim: The aim is to review and summarize the available evidence in the specialized literature on the relationship between periapical complications in primary teeth and their association with pathological changes in the developing tooth structures of permanent teeth, such as the formation of follicular cysts or defects in the enamel structure.

Materials and methods: A systematic review of the scientific literature in PubMed, Scopus, and Web of Science was performed. An analysis was made of scientific publications that examine periapical inflammatory processes affecting mainly primary molars, as well as their potential for the development of follicular cysts.

Results: It was found that periapical infection of primary teeth is associated with defects in the enamel (hypoplasia, hypomineralization), eruption abnormalities, dilacerations, ankylosis, delayed or impaired root development, and the development of inflammatory follicular cysts.

Conclusion: Persistent inflammatory processes associated with periapical lesions have a negative effect on the follicular tissues of permanent teeth. This can lead to structural changes in these tissues, as well as to the formation of follicular cysts and eruption disorders.

Keywords

primary teeth; periapical inflammations; periapical lesions, complications; deciduous molars; follicular cyst

Introduction

Untreated carious lesions in primary teeth negatively affect the child's quality of life, but can also cause long-term adverse effects on permanent teeth. In addition to being a serious predictor of caries in the permanent dentition, they can also cause structural defects in the permanent tooth [1]. If left untreated, carious lesions in primary teeth progress to inflammation of the dental pulp, followed by the development of apical periodontitis.

Apical periodontitis is an inflammatory response associated with pathogens and toxins in the root canals of

Copyright by Maria Kirilova. This is an open access article distributed under the terms of the Creative Commons Attribution License (CC-BY 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

the tooth. It is often asymptomatic and may be discovered incidentally during a routine examination [2]. The cumulative effect of repeated restoration failures and secondary carious lesions further increases the prevalence of endodontic pathology [3]. Despite the available methods for treating pulpitis/periodontitis of primary teeth, which aim to preserve the tooth until its physiological exfoliation, there is evidence in the literature of risks to developing permanent teeth [4]. The problem stems from the fact that untreated infection of the preceding primary tooth, due to anatomical features, can negatively affect the underlying permanent tooth germ [5]. The impact of apical periodontitis is not limited to the specific primary tooth. The infection can lead to various complications of the permanent tooth germ, such as enamel hypoplasia, hypomineralization, follicular cyst formation, etc.[6, 7].

The aim of this article is to review and summarize the available evidence in the specialized literature on the relationship between periapical complications in primary teeth and their association with pathological changes in the developing tooth structures of permanent teeth, such as the formation of follicular cysts or defects in the enamel structure.

Materials and methods

Methods

The methodology of the review includes a systematic search of scientific literature in electronic databases such as PubMed, Scopus, and Web of Science. An analysis was made of scientific publications that examine periapical inflammatory processes affecting mainly primary molars, as well as their potential for the development of follicular cysts. The selection of scientific publications was made according to their scientific significance, relevance, and connection to the purpose of the study.

Literature review

Apical periodontitis – peculiarities in primary molars

Apical periodontitis (AP) is an inflammation and destruction of the periapical tissues. It is the result of etiological factors that are of endodontic origin or a consequence of trauma [8, 9, 10]. Various authors describe the interaction between bacteria and their toxins and the host's defenses at the border between the infected root pulp and the periodontal ligament. The result of this interaction is local inflammation accompanied by resorption of hard tissues and the periodontal ligament. This is followed by the development of periapical lesions [9, 10].

The main etiological factor for the development of apical periodontitis is microorganisms [11]. The goal of endodontic treatment in necrotic pulp is to prevent premature tooth loss and the resulting complex complications [12]. Preserving the primary tooth until it is replaced by the permanent tooth is the best solution for the child's oral health. This ensures space for the permanent tooth to erupt, preserves the integrity of the dental arch, and maintains chewing function [12, 13].

The main methods for treating pulpitis and apical periodontitis of primary teeth are pulpotomy and pulpectomy. This ensures the physiological preservation of primary teeth and the performance of their functions. [4]. The damage that can occur after the spread of infection from a necrotic primary tooth includes the development of abscesses, granulomas, cysts, internal resorption of the root of the primary tooth and the bone around the roots [18, 21].

Features of primary teeth that determine the risk of complications in the permanent tooth germ

The main problem arising from infection of primary teeth is the effect that this infection may have on the development of the underlying permanent tooth germ. This is due to their proximity and the anatomical features of primary teeth [5]. The literature discusses the anatomical, histological, and clinical aspects of damage to permanent teeth caused by periapical infection of primary teeth. [5]. The pathology of PA is not limited to primary teeth. Periapical infections can have consequences for permanent teeth. Various complications have been described in the literature, including enamel hypoplasia, impaired or delayed development, changes in eruption, partial or complete arrest of permanent tooth development, and the formation of follicular cysts [6, 7].

Periapical lesions such as cysts and granulomas develop as a result of chronic irritation caused by anaerobic bacterial infection originating in the root canal [14]. In primary molars, inflammatory changes resulting from pulp inflammation are observed in the bone structure in the area of their bifurcation [15]. There are studies in the literature that indicate that the furcation area of primary molars has multiple additional canals, openings, and accessory structures that connect the pulp chamber to the periapical structures [16]. The furcation of primary molars has reduced dentin thickness and a zone of resorption, which determines the permeability of the area [17]. Thus, the diffusion of products from the decomposition of dental pulp, microorganisms and their toxins, as well as medications used in endodontic treatment, can lead to periapical inflammatory reactions [16, 17]. A study has found that the topographical position of the roots of the deciduous tooth relative to the germ of the permanent tooth may explain the potential for possible disturbances in the development of the permanent tooth following infection originating from its predecessor [18]. Such infections, especially in untreated necrotic primary teeth, can lead to periapical inflammation. This inflammation can spread to the follicular tissues of the permanent tooth and can lead to disruption of tooth eruption, and the accumulation of inflammatory infiltrate can lead to the formation of a follicular cyst [19].

Authors indicate that in cases of untreated pulpitis in primary teeth or failure of endodontic treatment of primary teeth, the infection may progress to the

development of a periapical lesion. This lesion can range from acute, such as a periapical abscess, to chronic, including periapical granuloma, cyst, osteomyelitis, affecting the follicular tissues of the permanent tooth germ, and others [5,18]. Progression depends not only on the virulence of MO, but also on the individual's immune status, local predisposing factors, and the duration of the disease [5].

Effects on permanent teeth

Complications reported in the literature when the infection reaches the permanent tooth germ include enamel hypoplasia, hypomineralization, cessation of root development, follicular cyst formation, accelerated eruption, and ankylosis. [13,18] Complications such as crown/root distortion (dilaceration), premature eruption of permanent teeth, including the formation of a follicular cyst, are also described in the literature [5,22]. Other authors document that changes in shape, enamel defects, and eruption deviations may be observed [23]. The development of various enamel defects is irreversible damage [17]. This damage is caused by physicochemical and biological effects on the ameloblasts. [17].

The database contains studies that examine the interaction between inflammatory processes in primary teeth and their impact on the development of the permanent tooth germ, which show significant clinical correlations. Some authors suggest that structural changes in enamel may be caused by infection of an untreated necrotic tooth [24]. The frequency of enamel defects in permanent teeth is higher in cases of untreated infection of a primary tooth [13, 25]. Results from another study demonstrate that structural defects in the enamel of permanent teeth are the result of endodontic treatment (pulpotomy/pulpectomy) [25].

In addition to reports of hypoplasia and hypomineralization of permanent teeth, there are also reports of delayed development of the root of the permanent tooth [26]. In cases where the inflammation is more severe, it can lead to a complete halt in tooth development and sometimes to destructive changes that result in sequestration of the tooth germ [26]. The damage is explained by the anatomical connection between the developing permanent tooth and the interradicular region of the primary molar [26].

In addition to structural defects in the enamel, the literature describes another pathology that affects the underlying tissues and can lead to the formation of follicular cysts, especially if the inflammation is acute and spreads to the sensitive structures – the ameloblasts [26]. In addition to these, radicular cysts are also described in the literature. They originate from Malasse's epithelial remnants as a result of apical periodontitis. Radicular cysts in primary teeth are a rare condition [27]. There are various reasons why they are rare, one of which is the short period of presence of primary teeth and easy drainage due to the additional canals in the bifurcation area of primary teeth [27].

The second most common cyst described in the literature is the dentigerous cyst (also known as a follicular cyst), which is an odontogenic cyst associated with an unerupted permanent tooth, supernumerary tooth, or, rarely, deciduous teeth [28, 29, 32]. A follicular cyst is a pathology that covers the crown of an unerupted tooth and is attached to the enamel-cementum junction of the tooth [35, 36, 37]. The etiology of follicular cysts is unclear [32, 36]. The cyst may form in the periapical space after endodontic treatment of a primary tooth. [28]. The pathogenesis of the cyst is unclear, with authors describing that the formation of a follicular cyst may be the result of a change in the reduced enamel epithelium [28]. Prolonged inflammation can cause irritation of the germ sac of the permanent tooth. This, in turn, leads to the development of a follicular cyst [28].

There are various theories about the development of follicular cysts. Some authors suggest that the cause of follicular cyst development is related to abnormalities in the early stages of tooth formation. Others believe that it has an inflammatory origin from an infected primary tooth [38]. The literature contains data from authors who present various studies showing that there are two types of follicular cysts [29, 39]. The first type occurs during tooth development as a result of impacted teeth. The second type is inflammatory in origin, associated with inflammation from a non-vital deciduous tooth or another cause [25, 29, 40]. Most studies report that inflammatory odontogenic cysts are more common than those that occur during tooth development [41]. Inflammatory follicular cysts are found in mixed dentition [19, 32, 42]. In children, this type of cyst develops during this period because the risk of certain factors is increased. This includes retention of upper canine teeth, development of periapical inflammation from a necrotic deciduous tooth, or complications after failed endodontic treatment [32, 35, 36]. The infection from the necrotic tooth spreads to the follicle and involves it [35]. The teeth affected are the mandibular premolars, successors to the deciduous molars [19, 29, 36, 37, 44].

The literature describes that second primary molars are predisposed to the development of complicated carious lesions [19,28]. This is explained by the high frequency of endodontic treatment performed on these teeth and is associated with the development of follicular cysts. According to the authors, primary teeth that have undergone endodontic treatment show a higher frequency of such complications [33, 36]. Another study emphasizes the high frequency of odontogenic infections affecting the first primary molars [17]. Several possible reasons for this are indicated: early eruption and longer period of exposure to cariogenic factors; the possibility of developing lesions in the period between tooth eruption and dental examination; the young age of the child and the distal position of the teeth in the dental arches may contribute to more difficult and less effective cleaning [17].

Asian-Gonzalez reports that the association between a developing follicular cyst and a carious lesion or endodontically treated second primary molar is significant. [28]. This is supported by another study reporting that periapical lesions in primary teeth resulting

from persistent irritants have the potential to affect the follicular tissues of the developing permanent tooth [40]. A number of authors agree that persistent infection is a serious risk factor for the development of this type of complication. [29, 30, 31]. This is supported by studies that note that the materials used in endodontic treatment can cause cyst development [20]. These types of conditions are common in primary molars. They remain asymptomatic for a long period of time. Therefore, they require a strict follow-up protocol to prevent long-term complications [40].

Follicular cysts are most common in childhood. According to the literature, they occur in children aged 6-12 years [46, 48]. They are mainly located in the lower jaw [47, 48,50,51]. Follicular cysts develop asymptomatically over a long period of time [5, 38]. They are discovered incidentally on radiography during routine examination or when patients complain of pain and swelling [19, 29,]. According to the literature, radiographically, follicular cysts are well-defined, associated with the crown of an unerupted or impacted tooth in the bone [35], and are often associated with a non-vital primary tooth [19].

Conclusion

Persistent inflammatory processes associated with periapical lesions have a negative effect on the follicular tissues of permanent teeth. This can lead to structural changes in these tissues, as well as to the formation of follicular cysts and eruption disorders.

Additional information

Funding statement

National program “Young Scientists and Postdoctoral Students – 2’’.

Ethical compliance

Not applicable.

Conflict of interest

The authors declare that they have no conflict of interest and no affiliations with or involvement in any organization or entity with any financial interest in the subject matter or materials discussed in this manuscript.

Author contributions

No

Acknowledgements

National program “Young Scientists and Postdoctoral Students – 2’’.

References

1. Grund K, Goddon I, Schüler IM, Lehmann T, Heinrich-Weltzien R. Clinical consequences of untreated dental caries in German 5- and 8-year-olds. BMC Oral Health. 2015 Nov 4;15(1):140. doi: 10.1186/s12903-015-0121-8.
2. Tibúrcio-Machado CS, Michelon C, Zanatta FB, Gomes MS, Marin JA, Bier CA. The global prevalence of apical periodontitis: a systematic review and meta-analysis. Int Endod J. 2021 May;54(5):712-735. doi: 10.1111/iej.13467.
3. Fouad AF, Khan AA, Silva RM, Kang MK. Genetic and Epigenetic Characterization of Pulpal and Periapical Inflammation. Front Physiol. 2020 Feb 4;11:21
4. Yawaka Y, Kaga M, Osanai M, Fukui A, Oguchi H. Delayed eruption of premolars with periodontitis of primary predecessors and a cystic lesion: a case report. Int J Paediatr Dent. 2002 Jan;12(1):53-60. doi: 10.1046/j.0960-7439.2001.00323.x.
5. Gupta SS, Shetty DC, Urs AB, Nainani P. Role of inflammation in developmental odontogenic pathosis. J Oral Maxillofac Pathol. 2016 Jan-Apr;20(1):164.
6. Beltrame AP, Bolan M, Serratine AC, Rocha MJ. Bacterial intensity and localization in primary molars with caries disease. J Indian Soc Pedod Prev Dent. 2012 Jan-Mar;30(1):32-40. doi: 10.4103/0970-4388.95576.
7. Patil N, Vishwakarma AP, Singh R, Aggarwal M, Ali MO, Khan AR. Effect of Chronic Apical Periodontitis in Primary Molars on Succedaneous Permanent Teeth: An Observational Retrospective Study. Cureus. 2023 Sep 14;15(9):e45275. doi: 10.7759/cureus.45275.
8. Bharuka SB, Mandroli PS. Single- versus two-visit pulpectomy treatment in primary teeth with apical periodontitis: A double-blind, parallel group, randomized controlled trial. J Indian Soc Pedod Prev Dent. 2016 Oct-Dec;34(4):383-90. doi: 10.4103/0970-4388.191429.
9. Blake A, Tuttle T, McKinney R. Apical Periodontitis. 2023 Jul 17. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024
10. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. Crit Rev Oral Biol Med. 2004 Nov 1;15(6):348-81. doi: 10.1177/154411130401500604.
11. Shah HS, Patil VM, Kamath AP, Mathur AA. Comparative Evaluation of Instrumentation Time, Obturation Time, and Radiographic Quality of Obturation Using Two Rotary Systems and Manual Technique for Primary Molar Pulpectomies - *In vivo* Study. Contemp Clin Dent. 2021 Jan-Mar;12(1):55-62. doi: 10.4103/ccd.ccd_83_20.
12. Sirohi K, Marwaha M, Gupta A, Bansal K, Srivastava A. Comparison of Clinical and Radiographic Success Rates of Pulpotomy in Primary Molars using Ferric Sulfate and Bioactive Tricalcium Silicate Cement: An *in vivo* Study. Int J Clin Pediatr Dent. 2017 Apr-Jun;10(2):147-151. doi: 10.5005/jp-journals-10005-1425.
13. Coll JA, Dhar V, Vargas K, Chen CY, Crystal YO, AlShamali S, Marghalani AA. Use of Non-Vital Pulp Therapies in Primary Teeth. Pediatr Dent. 2020 Sep 15;42(5):337-349.
14. Lukić A, Vojvodic D, Majstorović I, Colić M. Production of interleukin-8 in vitro by mononuclear cells isolated from human periapical lesions. Oral Microbiol Immunol. 2006 Oct;21(5):296-300. doi: 10.1111/j.1399-302X.2006.00293.x.
15. Cleghorn, Blaine & Boorberg, Noriko & Christie, William. (2010). Primary human teeth and their root canal systems. Endodontic Topics. 23. 10.1111/etp.12000.
16. Moura LFAD, Lima MDM, Lima CCB, Bandeira AVL, Moura MS, Conde Júnior AM, Rizzo MDS. Cellular profile of primary molars with pulp necrosis after treatment with antibiotic paste. Int J Exp Pathol. 2018 Oct;99(5):264-268. doi: 10.1111/iep.12292. Epub 2018 Oct 15.
17. Sousa HCS, Lima MDM, Lima CCB, Moura MS, Bandeira AVL, Deus Moura LFA. Prevalence of Enamel Defects in Premolars Whose Predecessors Were Treated with Extractions or Antibiotic Paste. Oral Health Prev Dent. 2020 Sep 4;18(4):793-798. doi: 10.3290/j.ohpd.a45083.

18. Cordeiro MM, Rocha MJ. The effects of periradicular inflamation and infection on a primary tooth and permanent successor. J Clin Pediatr Dent. 2005 Spring;29(3):193-200. doi: 10.17796/jcpd.29.3.5238p10v21r2j162.
19. de Oliveira GA, Limongi MC, Garcia HS, Fonseca LC, Manzi FR. Success in Conservative Treatment of an Inflammatory Follicular Cyst: A 5-year Follow-up. Int J Clin Pediatr Dent. 2021 Mar-Apr;14(2):327-330. doi: 10.5005/jp-journals-10005-1924.
20. da Silva LA, Nelson-Filho P, Faria G, de Souza-Gugelmin MC, Ito IY. Bacterial profile in primary teeth with necrotic pulp and periapical lesions. Braz Dent J. 2006;17(2):144-8. doi: 10.1590/s0103-64402006000200012.
21. Valderhaug J. Periapical inflammation in primary teeth and its effect on the permanent successors. Int J Oral Surg. 1974;3(4):171-82.
22. Yildiz E, Tosun G, Sari I. Early loss of a permanent tooth due to preceding primary tooth infection. J Pediatr Dent 2014;2:25-7.
23. Santana Neto MC, Lopes Bandeira AV, Santos Sousa HC, de Moura MS, de Fátima Almeida de Deus Moura L, de Deus Moura de Lima M. Enamel of Premolars whose Predecessors Presented Rupture of the Follicle Bone Crypt from Periapical Infections. J Clin Pediatr Dent. 2020 Aug 1;44(4):256-261. doi: 10.17796/1053-4625-44.4.7.
24. Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. Pediatr Dent. 1996 Jan-Feb;18(1):57-63.
25. Pruhs RJ, Olen GA, Sharma PS. Relationship between formocresol pulpotomies on primary teeth and enamel defects on their permanent successors. J Am Dent Assoc. 1977 Apr;94(4):698-700. doi: 10.14219/jada.archive.1977.0353.
26. McDonnell ST, Liversidge H, Kinirons M. Temporary arrest of root development in a premolar of a child with hypodontia and extensive caries. Int J Paediatr Dent. 2004 Nov;14(6):455-60. doi: 10.1111/j.1365-263X.2004.00582.x.
27. Shetty S, Angadi PV, Rekha K. Radicular cyst in deciduous maxillary molars: a rarity. Head Neck Pathol. 2010 Mar;4(1):27-30. doi: 10.1007/s12105-009-0152-z. Epub 2009 Nov 25.
28. Asián-González E, Pereira-Maestre M, Conde-Fernández D, Vilchez I, Segura-Egea JJ, Gutiérrez-Pérez JL. Dentigerous cyst associated with a formocresol pulpotomized deciduous molar. J Endod. 2007 Apr;33(4):488-92. doi: 10.1016/j.joen.2006.10.011.
29. Benn A, Altini M. Dentigerous cysts of inflammatory origin. A clinicopathologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996 Feb;81(2):203-9. doi: 10.1016/s1079-2104(96)80416-1.
30. Lustig JP, Schwartz-Arad D, Shapira A. Odontogenic cysts related to pulpotomized deciduous molars: clinical features and treatment outcome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999 Apr;87(4):499-503. doi: 10.1016/s1079-2104(99)70251-9.
31. Gondim JO, Neto JJ, Nogueira RL, Giro EM. Conservative management of a dentigerous cyst secondary to primary tooth trauma. Dent Traumatol. 2008 Dec;24(6):676-9. doi: 10.1111/j.1600-9657.2008.00616.x.
32. Gupta K, Kohli A, Verma A, et al. Management of dentigerous cyst in mixed dentition with successful eruption of permanent tooth: a case report with long term follow up. Rama Univ J Dent Sci. 2015;2(1):67–72.
33. Khzam N. What do we know about dentigerous cysts in children? A review of the literature. J Res Med Dent Sci. 2017;5:1–6
34. Shibata Y, Asaumi J, Yanagi Y, Kawai N, Hisatomi M, Matsuzaki H, Konouchi H, Nagatsuka H, Kishi K. Radiographic examination of dentigerous cysts in the transitional dentition. Dentomaxillofac Radiol. 2004 Jan;33(1):17-20. doi: 10.1259/dmfr/24148363.
35. Huang G, Moore L, Logan RM, Gue S. Histological analysis of 41 dentigerous cysts in a paediatric population. J Oral Pathol Med. 2019 Jan;48(1):74-78.
36. Marques NP, Marques NCT, Sakai VT, Hanemann JAC. Inflammatory follicular cysts associated to necrotic primary teeth. Eur Arch Paediatr Dent. 2017 Aug;18(4):279-285. doi: 10.1007/s40368-017-0297-5.
37. McKinney SL, Lukes SM. Dentigerous cyst in a young child: a case report. Can J Dent Hyg. 2021 Oct 1;55(3):177-181.
38. Nahajowski M, Hnitecka S, Antoszewska-Smith J, Rumin K, Dubowik M, Sarul M. Factors influencing an eruption of teeth associated with a dentigerous cyst: a systematic review and meta-analysis. BMC Oral Health. 2021 Apr 7;21(1):180. doi: 10.1186/s12903-021-01542-y.
39. Sethi A, Shetty DC, Tandon A, Afroz N. Bilateral Nonsyndromic Cystic Lesions Involving Impacted Teeth: Nomenclature and Diagnostic Protocol. Int J Clin Pediatr Dent. 2020 Jul-Aug;13(4):429-432. doi: 10.5005/jp-journals-10005-1795..
40. Patil AS, Jathar PN, Panse AM, Bahutule SR, Patil RU, Patil M. Infected Dentigerous Cyst and its Conservative Management: A Report of Two Cases. Int J Clin Pediatr Dent. 2019 Jan-Feb;12(1):68-72. doi: 10.5005/jp-journals-10005-1578.
41. Rølling I, Poulsen S. Formocresol pulpotomy of primary teeth and occurrence of enamel defects on the permanent successors. Acta Odontol Scand. 1978;36(4):243-7. doi: 10.3109/00016357809004675.
42. Kammer PV, Mello FW, Rivero ERC. Comparative analysis between developmental and inflammatory odontogenic cysts: retrospective study and literature review. Oral Maxillofac Surg. 2020 Mar;24(1):73-84. doi: 10.1007/s10006-019-00816-8.
43. Kumar R, Singh RK, Pandey RK, Mohammad S, Ram H. Inflammatory dentigerous cyst in a ten-year-old child. Natl J Maxillofac Surg. 2012 Jan;3(1):80-3.
44. Berdén J, Koch G, Ullbro C. Case series: Treatment of large dentigerous cysts in children. Eur Arch Paediatr Dent. 2010 Jun;11(3):140-5. doi: 10.1007/BF03262730.
45. McKinney SL, Lukes SM. Dentigerous cyst in a young child: a case report. Can J Dent Hyg. 2021 Oct 1;55(3):177-181.
46. Sharma D, Dubey G, Agrawal R, Verma P, Kulkarni VK. Inflammatory dentigerous cyst in mixed dentition: a rare sequela of incomplete endodontic treatment of a primary molar. *Journal Name*. 2015;1:58–62
47. Kozelj V, Sotosek B. Inflammatory dentigerous cysts of children treated by tooth extraction and decompression--report of four cases. Br Dent J. 1999 Dec 11;187(11):587-90. doi: 10.1038/sj.bdj.4800339a.
48. Langă MC, Nica DF, Duma V-F, Heredea RE, Sinescu C. Dentigerous cysts in children: clinical, radiological, and healing aspects. Medicina (Kaunas). 2024;60(7):1133
49. Sohn C, Ryu J, Nam I, Shin SH, Lee JY. Cystic lesion between a deciduous tooth and the succeeding permanent tooth: a retrospective analysis of 87 cases. J Korean Assoc Oral Maxillofac Surg. 2022 Dec 31;48(6):342-347.
50. Rajae EG, Karima EH. Dentigerous cyst: enucleation or marsupialization? A case report. Pan Afr Med J. 2021;40:149. doi: 10.11604/pamj.2021.40.149.28645.
51. Noujeim Z, Nasr L. The prevalence, distribution, and radiological evaluation of dentigerous cysts in a Lebanese sample. Imaging Sci Dent. 2021;51(3):291-297. doi: 10.5624/isd.20210075.