Clinico-radiologic features of molar-incisor malformation in a case series of 38 patients
A retrospective observational study

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Abstract
Molar-incisor malformation (MIM) is a recently defined dental abnormality of molar root and incisors, and introduced as one of the causes of periapical and periodontal abscesses. The purpose of this study was to investigate the clinical and radiological features of MIM, with special emphasis on various medical history. A total of 38 patients with MIM were included in this study. Radiographic features and clinical data, including medical history, chief complaint, associated complications, treatment, and prognosis, were retrospectively investigated. On radiographs, the affected molars showed short, slender, underdeveloped roots and constricted pulp chambers. All affected incisors and canines exhibited dilacerated short roots, wedge-shaped defect on the cervical part of the crown. Complications included periodontal bone loss (52.6%), endodontic lesion (50.0%), and endodontic-periodontal lesion (28.9%). The medical histories of the patients with MIM indicate that almost all (94.7%) were hospitalized due to problems during the neonatal period. MIM may cause various dental problems, such as periapical and periodontal abscesses and early loss of the affected teeth. The early diagnosis of MIM on radiographs and appropriate treatment will contribute to a favorable prognosis, especially for young and adolescent patients.

Abbreviation: MIH = molar-incisor hypoplasia, MIM = molar-incisor malformation, SMIDD = symmetric multiquadrant isolated dentin dysplasia.

Keywords: complication, neonatal, pediatrics, radiography, tooth development

1. Introduction
A particular form of malformed root associated with an abnormally mineralized diaphram on cervical portion of the pulp was recently defined by Witt et al.1 At about the same time, Lee et al. named this condition molar-incisor malformation (MIM).2 MIM, also known as molar root-incisor malformation (MRIM), is typically identified by underdeveloped roots with a short, narrow, and spiky shape, appearing most often on permanent first molars,3 and less often on deciduous second molars and permanent maxillary central incisors.

MIM has been reported in relatively recent years. Until it is known, it has been mistaken for molar-incisor hypomineralization (MIH), a developmental tooth anomaly, which occurs in permanent first molars and incisors. MIH describes a clinical feature wherein one to all permanent first molars have hypomineralized enamel.4 This condition is also frequently associated with incisors. Affected teeth are fragile and susceptible to enamel breakdown and dental caries. Much research has been conducted on MIH and the prevalence ranges between 3.6% and 25%.4

In addition to the similarities in the areas that occur, there are similarities in the causes. Systemic complications or environmental factors during delivery or immediately after birth are a presumed cause of MIH.5-9 Although the etiology of MIM remains unclear, administration of various medications, congenital health problems, and neonatal infections are suggested as the cause of anomaly.5-9

There are differences between the two abnormalities. Even though both conditions commonly occur on permanent central incisors and first molars, MIH manifests only on the coronal portion of teeth and root involvement is yet to be reported. MIH usually can be diagnosed by visual inspection of enamel opacities; in contrast, MIM of molars can only be diagnosed by radiographic examination regardless of eruption because it manifests as root and pulp chamber malformations. In addition, the teeth with MIM have potential complications including tooth
devitalization with subsequent tooth mobility, abscess formation and alveolar bone loss.\cite{1,2}

Until recently, a few studies on MIM have been reported. The current study aimed to investigate clinical and radiological features of MIM with relatively large number of cases, with special emphasis on previous medical history and complications.

2. Materials and methods

This study was approved by the Institutional Review Board of Seoul National University Dental Hospital (SNUDH) [IRB No. CRI15008]. Radiographic data were retrospectively reviewed for the patients who visited SNUDH from January 2001 to October 2017. The conclusion section of radiology reports were searched using the Picture Archiving and Communications System (INFINIT PACS, INFINITT Healthcare, Seoul, South Korea) using the keywords malformed, malformation, dysplastic, dysplasia, hypoplastic, or hypoplasia, in combination with both tooth or root. Medical histories of all but 2 patients (94.7%) indicated special emphasis on previous medical history and complications. Permanent and deciduous teeth and their complications on radiographs such as periodontal bone loss, periapical lesion, or combined lesion were recorded. Review of both medical and dental charts was performed, focusing especially on medical history during the neonatal period. Clinical features, including chief complaint, associated complications, treatment, and prognosis, were also analyzed.

3. Results

Total of 38 patients with MIM, 24 males and 14 females, were analyzed. The age of the patients at the time of their first visit to SNUDH ranged from 3 to 23 years, the average being 8.5 years. The follow-up period ranged from 1 to 10 years, with an average of 4.8 years. Only 15 patients presented with a chief complaint of pain, mobility, or swelling related to MIM-affected teeth, whereas all other patients presented with no symptoms on the affected teeth. Further, 12 patients were referred from a local dental clinic due to malformation of the affected roots detected on panoramic radiograph, and 4 patients visited SNUDH for orthodontic treatment, with 2 of them complaining of severe labioversion and malformation of the permanent central incisors affected by MIM.

Medical histories of all but 2 patients (94.7%) indicated hospitalization due to various problems during the neonatal period (Table 1). Additionally, 15 patients (39.5%) exhibited diseases involving the central nervous system, among which 9 showed a history of infection, primarily meningitis. Furthermore, 12 patients (31.6%) were hospitalized with low body weight or dystocia due to premature birth, and 7 patients underwent surgery for cardiac, pulmonary, or duodenal problems and 3 patients exhibited infection or inflammation.

All 38 patients showed MIM on the permanent first molars. All four permanent first molars were affected, except 3 of the patients. Along with this, the permanent upper central incisors were affected in 17 patients (44.7%), the permanent lower central incisors were affected in 2 patients. The permanent upper canines were affected in 4 patients (10.5%), of whom two patients (5.3%) had affected lower canine. Additionally, 20 patients (52.6%) exhibited MIM on the deciduous molars. The permanent upper lateral incisors, the permanent lower lateral incisors, and the deciduous lower canines were affected in one patient, respectively.

In radiographs, the affected permanent first molars and deciduous molars showed short, slender, underdeveloped or undeveloped roots (Figs. 1–4). Also we could find severely obstructed pulp chambers evidenced by a thin horizontal radiolucent line, and increased radiopacity in the cervical portion of crown due to mineralized plate and enamel protuberance of constriction. No abnormalities in shape or color of the crown portion of the molars had been noted on visual inspection. All of the affected permanent incisors and canines exhibited dilacerated or slender, short roots. In most of the cases with affected incisors, wedge-shaped defects or notch on the cervical part of the crown were shown to the various degree (Fig. 1A, B, and Fig. 2). The cervical constriction of incisors and canines were characteristic features also. However, we could not see root malformations including underdevelopment and dilaceration frequently in incisors and canines (Table 2).

Complications of the affected teeth included endodontic-periodontal lesion (Fig. 3) in 11 patients (28.9%), periodontal bone loss in nine patients (23.7%), and periapical lesion in 8 patients (21.1%). Periapical or periodontal lesions occurred in MIM affected teeth without any evidence of infection such as dental caries, crown fracture, or calculus. A periapical cyst developed from an affected molar at 4 years of follow-up in one patient (Patient no. 10, Fig. 4). In another patient, cellulitis of the left buccal cheek was noted and was caused by a periapical lesion of an affected permanent first molar.

Prognosis of the affected teeth indicated that 16 patients (42.1%) lost 1 or more permanent teeth that were affected by MIM. In terms of numbers of lost teeth, early loss was found in 19.2% (38/198) of affected permanent teeth. The early loss of permanent teeth occurred for various reasons, including severe periodontal bone loss or tooth mobility due to a very short root. Among 34 permanent teeth in the 19 patients with periapical or endodontic-periodontal lesions, 18 teeth (52.9%) were lost early, 10 teeth were retained after root canal treatment, and 6 teeth were retained without any treatment. Treatments performed after extraction of the affected teeth included orthodontic treatment and implantation of prostheses. Of 17 patients with affected permanent incisors, 2 lost their incisors and 4 had incisors preserved after endodontic procedures or restoration; the remaining 11 patients had retained the incisors with periodic recall check-ups. For those with extracted incisors, space maintainers were applied with plans for dental implant surgery after the completion of alveolar bone growth.

4. Discussion

In the present study, patients with MIM showed significant radiological and clinical features. Permanent and deciduous molars were affected by MIM in 38 patients (100%) and 20 patients (52.6%), respectively, and they showed obstructed pulp chambers by the mineralized plate, with short and slender roots. Seventeen patients (44.7%) showed malformations on crowns of more than one incisors or canines. Affected incisors and canines had wedge-shaped defects or notch on the cervical portion of the
### Table 1
Summary of clinical information.

| Pt No. | Age (yr)/Sex | Affected tooth | Medical history | Chief complaint | Complication | Treatment and prognosis
|--------|--------------|----------------|----------------|----------------|-------------|------------------------|
| 1      | 9/M          | 6 1 6 1 6     | Bacterial meningitis in neonatal period | Referral from LDC due to root malformation | Early loss: #16,26 | #16,26 extraction |
| 2      | 7/M          | 6E 1 6E E6    | Hospitalized due to cerebral hemorrhage in neonatal period | Referral from LDC due to impacted mesiodens | Early loss: #55,65,75,85, Severe periodontal bone loss: #36,46 | #11,16,21,26,36,46: retained under periodic recall check for 2 years, #16,26,36,46 extraction |
| 3      | 8/M          | 6E 1 6E E6    | Hospitalized due to bacterial meningitis 15 days after birth | Orthodontic treatment | Early loss: #16,26,36,46 | #16,26,36,46 extraction |
| 4      | 8/M          | 6E 3 3 6 3 6  | Premature birth at 32 weeks, mental retardation, asthma, viral encephalitis at 4 years of age | Referral from LDC for caries treatment | Congenital missing: #35,45 | All affected teeth retained under periodic recall check for 10 years |
| 5      | 12/M         | 6E 6 6 6 6    | Cranial surgery due to craniolacunatosis 2 years after birth, pneumonia | Orthodontic treatment | Endodontic-periodontal lesion, and early loss: #16,26,36,46 | #16,26,36,46; extraction after treatment |
| 6      | 7/F          | 6E 6 6 6 6    | Lipomeningomyelocle surgery in neonatal period | Orthodontic treatment | Early loss: #36,46 | #16,26; retained under periodic recall check for 4 years |
| 7      | 7/M          | 6E 6 6 6 6    | Premature birth at 32 weeks, encephalopathy, cerebral hemorrhage | Referral from a medical hospital due to periapical abscess and cellulitis | Periapical abscess: #26. Cellulitis | #16,26,36,46; retained under periodic recall check for 1 year |
| 8      | 14/F         | 6E 6E 6E 6E   | Dystocia (care in incubator) | Tooth pain on mastication | Endodontic-periodontal lesion, and early loss: #16,36,46 | #16,36,46; extraction, implant prosthesis |
| 9      | 9/M          | 6 6 6 6 6     | Convulsion, brain capillary blockage in neonatal period | Tooth mobility and pain | Endodontic-periodontal lesion: #36,46 | #8,6; retained under periodic recall check for 5 years |
| 10     | 7/F          | 6E 1 6E 1 6E  | Cardiac surgery at 1 week after birth due to total anomalous pulmonary venous connection | Removal of foreign body around #75 | Periapical cyst at 4 years of follow-up: #46 Endodontic-periodontal lesion: #16,26, Early loss: 26, Congenital missing: #35,45 | #46,85; extraction and bone graft after curettage of periapical cyst |
| 11     | 9/F          | 6E3 6E 3E 3E  | Meningitis in neonatal period, epilepsy | Referral from LDC due to root malformation | Endodontic-periodontal lesion: #36 Early loss: #11,21 | #11,21 extraction, space maintainer, implant |
| 12     | 9/M          | 6E 1 6E 1 6E  | Premature birth at 28 weeks, genital operation in neonatal period | Referral from LDC due to root malformation | Endodontic-periodontal lesion: #36,75 Early loss: all affected teeth except #21 | #11,16,21,36,75; retained for 10 years |
| 13     | 5/F          | 6E 1 6D 1 6D  | Bacterial meningitis in neonatal period | Referral from LDC due to root malformation | Periapical lesion: #11,21, Congenital missing: #15,25,35,45 | #11,21; retained after RCT for 4 years, #16,26,36,46; retained for 4 years |
| 14     | 7/M          | 6E 6E 6E E6   | Spinal meningitis in neonatal period | Referral from LDC due to tooth malformation | Endodontic-periodontal lesion: #36 Early loss: #11,21 | #11,21 extraction, space maintainer, implant prosthesis |
| 15     | 5/M          | 6E 6E 6E 6E   | Chronic renal failure in neonatal period, hemodialysis | Referral from a medical hospital for oral exam before kidney transplantation | Endodontic-periodontal lesion: #36,46 Early loss: #36,46 | #16,26,36,46; retained under periodic recall check for 10 years |
| 16     | 8/F          | 6E 6E 6E CDE6 | Premature birth at 27 weeks | Referral from LDC due to root malformation | Periodontal bone loss: #16,26,36,46, Early loss: #36,46 | #36,46; extraction after RCT |
| 17     | 4/F          | 6E 6D 6D DE   | Premature birth at 29 weeks, chronic pulmonary disease | Caries treatment of #52 | Periodontal bone loss: #36 Sensitivity: #16,26,36,46 | #16,26,36,46; retained under periodic recall check for 3 years |
| 18     | 6/F          | 6E 6E 6E 6D   | Premature birth at 28 weeks | Tooth mobility | Early loss of #74,75 Periodontal bone loss: #36 Sensitivity: #16,26,36,46 | #16,26,36,46; retained under periodic recall check for 3 years |

(continued)
Table 1
(continued).

| Pt No. | Age/Sex | Affected tooth | Medical history | Chief complaint | Complication | Treatment and prognosis |
|--------|---------|----------------|-----------------|-----------------|--------------|------------------------|
| 19     | 7/F     | 6 1 6 1 6     | n/s             | Pain on #46     | Periodontal bone loss: #46 | #11,16,21,26,36,46: retained under periodic recall check for 1 years |
| 20     | 9/M     | 6 1 6 1 6     | Meningitis in neonatal period | Referral from LDC due to tooth malformation | Endodontic-periodontal lesion: #11,21,36 | #11,16,21,26,36,46: retained under periodic recall check for years |
| 21     | 9/M     | 6,3 1 3 6 1 1 6 | Cerebral hemorrhage, hydrocephaly surgery (3M) | Swelling and pus on #11 | Periapical lesion: #41 | #11,41: retained after RCT |
| 22     | 11/F    | 6 1 6 1 6     | Premature birth at 30 weeks | Pain on #46     | Periapical lesion with sinus tract, early loss: #16,46 | #11,21: retained with restoration for 6 months |
| 23     | 3/M     | 6,6 6,6       | Premature birth | Caries of deciduous teeth | Slight periodontal bone loss: #16,46 | #16,26,36,46: retained under periodic recall check for 1.5 years |
| 24     | 10/M    | 6 6 6,6       | Premature birth at 24 weeks | Referral from LDC due to tooth malformation | Slight periodontal bone loss: #36,46 | #11,16,21,22,26: retained under periodic recall check for 2 years |
| 25     | 6/F     | 6,6,6,6       | n/s             | Discomfort on chewing | Tooth mobility due to periodontal bone loss, early loss: #16,46 | #11,21: retained after RCT |
| 26     | 9/F     | 6,6 136 6,6   | Meningitis at 5 days after birth | Orthodontic treatment | Tooth mobility due to periodontal bone loss: #16,26,36,46 | #16,36,46: extraction of tooth malformation |
| 27     | 7/F     | 6,6 1 6,6     | Hospitalization due to sacrococcygeal inflammation at 19 days after birth | Swelling and pus on #11,21 | Early loss: #16,36,46 | - |
| 28     | 6/M     | 6,6 6,6       | Surgery due to pulmonary atresia in neonatal period | Referral from LDC due to tooth malformation | Early loss: #16,36,46 | - |
| 29     | 12/M    | 6 6 6,6       | Surgery due to biliary atresia in neonatal period | Pain on #36     | Periapical lesion, early loss: #36,46 | #16,26,36,46: retained under periodic recall check for 1 years |
| 30     | 8/M     | 6,6 1 6,6     | Hospitalization due to cerebrovascular disease in neonatal period | Referral from LDC due to tooth malformation | Early loss: #16,36,46 | #11,16,21,26,36,46: retained under periodic recall check for 1.5 years |
| 31     | 12/M    | 6 6 6,6       | Hospitalization due to jaundice and neonatal sepsis in neonatal period | Tooth mobility of #16,26 | Periodontal bone loss, early loss: #16,26,36,46 | #16,26,36,46: extraction of tooth malformation |
| 32     | 11/M    | 6 6 6,6       | Hospitalization due to premature birth at 33 weeks | Referral from LDC due to cyst like lesion on #46 | Endodontic-periodontal lesion, early loss: #46 | #16,26,36: extraction of tooth malformation |
| 33     | 23/F    | 6 6 6,6       | Hospitalization for 1 month after birth, due to heart disease | Swelling and fistula on #16 | Periapical cyst: #16,46 | #16,46: retained after RCT |
| 34     | 12/M    | 6 6 6,6       | Surgery due to duodenal atresia at neonatal period | Gingival fistula on #46 | Periodontal bone loss: #16,26 | #16,26,36,46: extraction of tooth malformation |
| 35     | 5/M     | 6,6 1 6,6     | Meningitis at neonatal period (10D) | Referral from LDC due to tooth malformation | Early loss of tooth: #55,65,85 | #11,16,21,26,36,46: retained under periodic recall check for 3.5 years |
| 36     | 4/M     | 6,6 6,6       | Hospitalization due to premature birth at 38 weeks and low birth weight (1.8kg). Surgery due to congenital heart disease at 1 month after birth | Referral from LDC due to tooth malformation | Early loss of tooth: #55,65,85 | #75,16,26,36,46: retained under periodic recall check |
| 37     | 10/M    | 6 6 6,6       | Hospitalization due to osteomyelitis of shoulder at neonatal period (10D) | Gingival swelling | Endodontic-periodontal lesion: #36,46 | #36,46: retained after RCT |
| 38     | 10/M    | 6 6 6,6       | Cerebral hemorrhagic at birth | Gingival fistula on #36 | Endodontic-periodontal lesion: #36,46 | #36: retained after drainage and irrigation |

LDC = local dental clinic, RCT = root canal treatment.
† Palmer system was used in this column for tooth numbering.
* Federation Dentaire Internationale (FDI) system was used in this column for tooth numbering.
Almost all patients (94.7%) had histories of hospitalization in the neonatal period. Including periapical cyst and cellulitis, endodontic and periodontal complications were commonly accompanied. Early loss of tooth was found in 16 patients (42.1%).

MIM, recently defined dental abnormality, could be confused with MIH because MIM and MIH both affect molars and incisors (Table 3). The clinical criteria for diagnosing MIH, including those of the European Academy of Pediatric Dentistry (EAPD), state that examination for MIH in epidemiological studies should include all permanent first molars and incisors. No consensus on diagnostic criteria has yet been suggested for the radiological features of MIH, probably because hypomineralization of affected teeth is not significant enough to be detected on radiographs. MIH-affected teeth can be identified by visual inspection due to their unique enamel opacities. In contrast, it is known that MIM-affected molars should be diagnosed using radiography because the teeth look normal upon visual inspection; however, MIM-affected permanent incisors and canines demonstrated prominent cervical defects observable by visual examination. As the name suggests, most MIM reports demonstrated molars and incisors affected. Only one other report and the present study reported MIM involved permanent canine teeth. Besides, the present study even found the MIM affected deciduous canine (Case no.16). The most characteristic radiologic features were pulp obstruction and cervical constriction in molars, crown defect and cervical constriction in incisors and canine. Cervical constriction is also a typical feature in dentinogenesis imperfecta, but unlike MIM, all teeth are affected, and pulp obstruction by the mineralized plate is not observed. A recent study proposed a new term for MIM—symmetric multi-quadrant isolated dentin dysplasia (SMIDD)—which is similar to dentin dysplasia type 1-b. As demonstrated in the present study, dentin and enamel layers of the crown of incisors and canines were also affected in MIM; therefore, we do not agree that the term “SMIDD” is appropriate for this disorder.

A few studies showed the histologic findings of MIM-affected molars, which showed ectopic mineralized plates at the pulp cavity floor which consisted of abnormal dentin and amorphous calcified tissue. However, the etiology of MIM remains unclear. Many

Figure 1. Panoramic radiograph (A) of a 9-year-old boy and periapical radiographs (B). Upper central incisors have a wedge-shaped defect on the crown and a severe cervical constriction of short dilacerated roots. Both lower first permanent molars show a normal crown contour but have only a single short and slender root. The pulp chambers of the permanent molars appear to be obstructed. Note that cervical portions of crowns show increased radiopacity due to mineralized plate and enamel protuberance of constriction.
conditions have been proposed to explain MIM. In the present study, patient histories of hospitalization were for various causes, including infection on the central nervous system, premature birth, surgery, or dystocia; notably, however, all had a medical history of intensive exposure to antibiotics. Even for the 12 patients with a history of premature birth, exposure to antibiotics is suspected, because antibiotic treatment tends to be used standardly for prematurely delivered babies in South Korea. Alaluusua suggested the use of antibiotics within six years after birth as a cause of MIH.\cite{13} We thought that intensive administration of antibiotics at critical periods in tooth development might be one of the causative factors of MIM as well. Other studies suggest brain-related systemic disease as an epigenetic factor explainable by neuro-osteology\cite{2,14}; however, less than half of the patients experienced diseases of the central nervous system in the present study, with the remainder hospitalized for other causes.

Most patients in the present study had a medical history of hospitalization during the neonatal period, suggesting an association between the environmental stressors of that period and specific malformation of molars and incisors. The development of the deciduous molars starts between birth and one year of age. If a risk factor exists during the neonatal period, root malformation of primary molars could occur; however, the timing of patient disease and root development of permanent molars does not match because roots develop around three years of age. Efforts to explain this marked difference in time intervals have been made by other researchers who speculate that certain stressors affect specific cells or that Hertwig epithelial root sheaths, formed long before root formation, are involved.\cite{3,8} These explanations do not fully clarify the mechanism by which only specific teeth in the same developmental stage are affected. Until now, the definite and reliable etiologic factors of MIM are not investigated. Reports of MIM have been made actively within the last 10 years; this implicates that the changes in modern society might be related in this disease. Multidisciplinary studies will be required to investigate the etiology of MIM.

Table 2

| Radiographic features | Incisors | Permanent or deciduous canines | Deciduous molars | Permanent first molars |
|-----------------------|----------|-------------------------------|------------------|------------------------|
| Wedge-shaped crown defect (17/17) | Cervical constriction (5/5) | Pulp obstruction (18/20) | Cervical constriction (38/38) |
| Cervical constriction (11/17) | Wedge-shaped crown defect (2/5) | Cervical constriction (15/20) | Pulp obstruction (37/38) |
| Split root (5/17) | Slender root (2/5) | Short root (14/20) | Slender/spiky root (35/38) |
| Calcified canal or pulp stone (3/17) | Dilacerated root (1/5) | Slender/spiky root (8/20) | Long root (34/38) |
| Slender root (2/17) | Undeveloped root (4/20) | Short root (34/38) | |
Figure 4. (A) Panoramic radiograph and intraoral periapical radiographs of Patient 10 at the age of 7 years. All permanent first molars exhibit normal crown contour, constricted pulp chambers, and indistinct underdeveloped roots. Also, all deciduous second molars show short, slender, and underdeveloped roots. (B) Panoramic radiograph taken 4 years later. Note the periapical cyst of right mandibular first molar, which is not seen in the past image.
The awareness and diagnosis of MIM are essential due to its related complications and prognosis. MIM-affected teeth easily develop periapical and periodontal lesions despite the lack of causative factors such as dental caries or calculi. Concerning endodontic lesions, in the current study, 18 teeth (52.9%) with endodontic lesions were extracted during the follow-up period, and ten teeth (29.4%) retained after proper root canal treatment. The root canal treatment of MIM is hard due to various conditions that include cervical mineralized diaphragm of pulp, many accessory canals, and a lack of periodontal support. During the follow-up period, less than half of patients lost one or more permanent teeth affected by MIM. Treatments such as implant installation or orthodontics should be considered after tooth loss. A careful review of the patient’s history, especially in the neonatal period, and awareness of the clinical and radiological features are essential for proper diagnosis.

There were some limitations in our study. The main limitation was the lack of histologic correlation of MIM-affected teeth. The second one was the lack of the three-dimensional (3D) evaluation, using cone-beam CT or multidetector CT images. We could not analyze the CT data because few subjects had undertaken CT images. However, we expect that more information about the morphologic malformation of MIM-affected teeth could be harvested when using the 3D images in further studies. The third one was that the present study only used the limited sample of specific population who had visited our hospital for dental care. Further investigations are required, including general population with a larger sample size. Also, detailed medical histories, including specific treatment and used drugs in the neonatal period, are needed for getting close to the etiology of MIM development.

5. Conclusion

MIM may cause early loss or severe inflammatory condition of the affected teeth and the surrounding region, and dentists should be aware of its clinico-radiological features. Early diagnosis with appropriate treatment will lead to the favorable prognosis for patients, especially young and adolescent individuals.

**Author contributions**

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