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CASE REPORT

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Molar–incisor malformation (MIM): a case report with a comprehensive review of the literature

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Abstract

Background: Molar–incisor malformation (MIM) is a recently described dental developmental anomaly involving the first permanent molars, maxillary central incisors, and second primary molars. Reported cases remain rare worldwide, with only sporadic reports from China and other Asian populations. The condition is characterized by abnormal root development and distinctive pulp chamber alterations, posing diagnostic and therapeutic challenges in pediatric dental practice. **Case**: We report a case of MIM in a Chinese child presenting with apical periodontitis of a maxillary central incisor. Clinical and radiographic examinations revealed characteristic cervical enamel defects and pulp chamber calcification consistent with MIM, along with genetic findings. Microscopy-assisted root canal treatment was performed, and radiographic follow-up at 9 months demonstrated resolution of apical inflammation and satisfactory clinical outcomes. In addition, a comprehensive review of the literature on MIM published since 2014 is presented. **Conclusions**: This case underscores the importance of early recognition of MIM and careful differential diagnosis to avoid mismanagement. Awareness of its characteristic clinical and radiographic features can facilitate appropriate treatment planning. Further case reports and genetic studies are warranted to elucidate the etiology and optimize management strategies for affected children.

Keywords

Molar–incisor malformation; Dental developmental anomalies; Pulp calcification; Clinical management

1. Introduction

Molar–Incisor Malformation (MIM), also referred to as molar–root incisor malformation (MRIM), is a recently described developmental dental anomaly that primarily affects the first permanent molars, maxillary central incisors, and second primary molars. It was first reported within the past decade [1, 2]. Clinically, MIM typically presents as enamel defects located at the cervical third of the labial surfaces of the maxillary central incisors. Although the first permanent molars and second primary molars often exhibit normal crown morphology on intraoral examination, radiographic findings characteristically reveal marked cervical constriction, narrow and linear pulp chambers, and pronounced underdevelopment of the distal roots. Globally, reports of MIM-affected teeth remain limited. A single-center audit conducted in a pediatric dental unit reported a prevalence of molar–incisor malformation of 0.47% (5 of 1054 children examined), highlighting the rarity of this condition in the pediatric population [3]. The earliest documented cases were published in 2014 [4]. Subsequently, an additional 12 cases were reported, and the condition was formally designated as molar–incisor malformation (MIM) [5]. These authors proposed that the observed root malformations might be associated with central nervous system–related disorders occurring within the first 1–2 years of life. However, approximately 6.9% of reported patients had no identifiable medical history [1]. Due to compromised structural integrity, teeth affected by MIM are susceptible to bacterial infection, which may result in pulpitis, apical periodontitis, premature tooth loss, and subsequent space management problems. At present, the exact etiology of MIM remains unclear, and clinical management strategies are largely determined by the severity of root malformation and symptoms. Comprehensive evaluation is, therefore, essential to formulate individualized treatment plans.

Therefore, this case report describes a rare presentation of MIM involving a permanent maxillary central incisor complicated by periapical periodontitis with acute exacerbation and outlines its clinical management. The uniqueness of this case lies in the successful endodontic treatment of an affected incisor with atypical cervical morphology and comprehensive management of MIM-affected teeth, particularly when tooth preservation is feasible.

2. Case presentation

A 9-year-old girl presented to the Department of Pediatric Dentistry, Stomatology Hospital, Zhejiang University School of Medicine, with a chief complaint of pain and discomfort in the upper left anterior tooth for 3 days. Her medical history was unremarkable, except for a congenital hemangioma on the left leg diagnosed at birth at Shanghai Ninth People's Hospital, which required no surgical or medical intervention (Fig. 1). The mother reported an uneventful pregnancy, with no illnesses, complications, or medication use. Intraoral examination revealed localized narrowing at the middle third of the labial surfaces of teeth 11 and 21 (Fig. 2, the yellow arrows). Tooth 21 exhibited mild tenderness to percussion, no mobility, slight gingival erythema and swelling at the marginal gingiva, bleeding on probing, and a positive response to cold testing. The vestibular sulcus corresponding to tooth 21 was mildly swollen and tender to palpation, with no sinus tract observed on either the buccal or palatal mucosa. Teeth 55, 65, 75, and 85 showed grade II mobility with otherwise normal gingival conditions, and teeth 55 and 85 were carious. Oral hygiene was poor, with abundant soft white deposits adherent to the tooth surfaces. Given the abnormal cervical morphology of the affected incisor, poor oral hygiene and plaque accumulation in the constricted cervical area may have facilitated bacterial ingress, thereby contributing to the development of pulpal inflammation and subsequent periapical pathology. The first permanent molars were fully erupted and exhibited normal crown morphology, texture, and color. Periapical radiographic examination (Fig. 3A) demonstrated wedge-shaped radiolucent defects at the cervical one-third of both the mesial and distal crown surfaces of teeth 11 and 21 (Fig. 3A, the red arrows), along with evident pulpal calcifications within the pulp chambers (Fig. 3A, the yellow arrows). No distinct periapical radiolucency was observed at the apex of tooth 21 on periapical imaging. However, cone-beam computed tomography (CBCT) revealed a small, focal, patchy radiolucent area at the apex of tooth 21 that was not clearly visible on the periapical radiograph (Fig. 3B, the yellow arrow). The apical foramen of tooth 21 appeared morphologically closed, with an estimated apical opening diameter of approximately 0.4 mm (Fig. 3C), consistent with histological expectations for apical closure. No developmental abnormalities of the maxillary alveolar bone were identified. CBCT imaging further demonstrated cluster-like radiopaque masses within the pulp chambers of teeth 11 and 21, consistent with pulpal calcifications (Fig. 3D, the yellow arrows). In addition, the maxillary and mandibular first permanent molars (teeth 16, 26, 36, and 46) showed preserved clinical crown morphology but markedly narrow, straight pulp chambers (Fig. 3E,F, the yellow arrows). The mesial root canal spaces of these molars were relatively enlarged, whereas the distal roots exhibited pronounced underdevelopment with a morphology resembling root resorption (Fig. 3E,F, the red arrows). The second permanent molar tooth germs (teeth 17, 27, 37, and 47) were impacted and positioned adjacent to the distal cervical root regions of the corresponding first permanent molars (Fig. 3E,F). High-density pulp stones were also observed within the pulp chambers of teeth 13 and 23 (Fig. 3G,H, the yellow arrows).

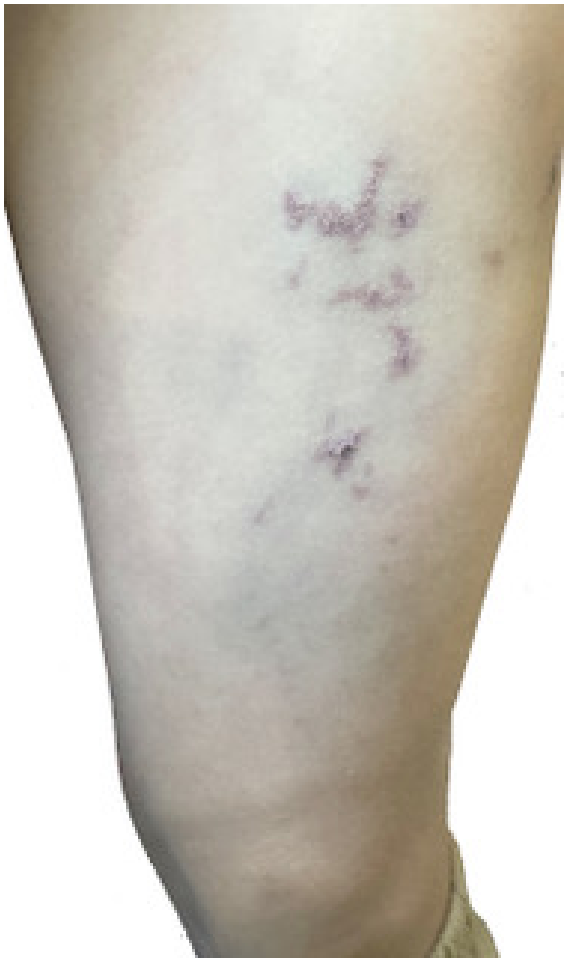

FIGURE 1. Hemangioma of the left lower limb.

The clinical features of this case were consistent with a diagnosis of MIM. Tooth 21 was diagnosed with chronic apical periodontitis with acute exacerbation and managed with nonsurgical root canal therapy. Endodontic treatment of tooth 21 was initiated under local anesthesia using 4% articaine and rubber dam isolation. Under dental operating microscope magnification, endodontic access was established, and copious purulent and bloody exudate was discharged from the pulp chamber, indicating active infection. Several sizeable pulp stones were identified (Fig. 4, the red arrow) and carefully removed using a P5 ultrasonic tip. The tooth exhibited a single canal, which was extirpated, and canal patency was established using K-files. The canal orifice was notably wide, and the working length was determined to be 22.5 mm. Chemomechanical preparation was performed up to size 25/06 using NiTi rotary files, PLEX-2.0 (Orodeka Medical Equipment Co. Ltd, Jining, Shandong, China), with alternating irrigation of 1% sodium hypochlorite and saline. The 1% low-concentration sodium hypochlorite solution was intentionally selected to ensure effective antimicrobial activity while reducing cytotoxicity. Although MIM is associated with abnormal root development, the structural defect in this case was mainly confined to the cervical region rather than the apical area. Under dental operating microscope magnification, the canal walls of tooth 21 were observed to be morphologically intact, indicating a minimal risk of irrigant extrusion. After canal drying with paper points, calcium hydroxide was placed as an

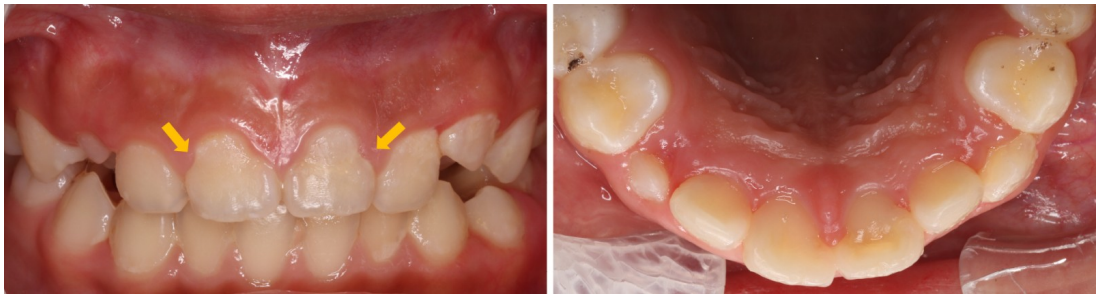

FIGURE 2. Intraoral photographs at first visit. The yellow arrows showing the cervical constriction of teeth 11 and 21.

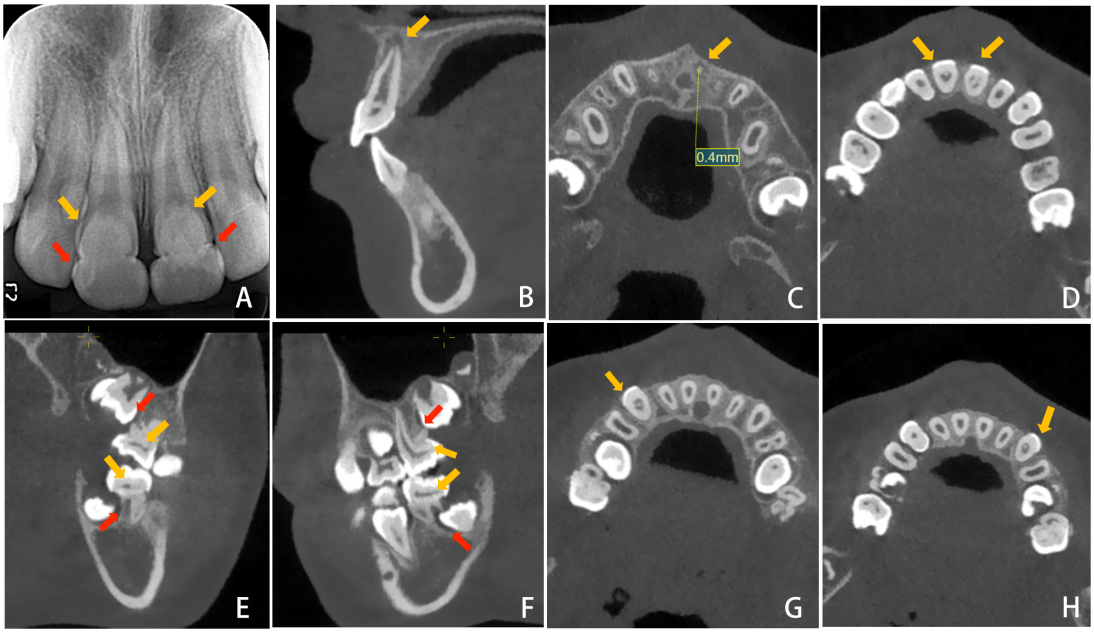

FIGURE 3. Initial radiographs. (A) Periapical radiograph. The yellow arrows showing the pulpal calcifications, and the red arrows showing the wedge-shaped radiolucent defects at the cervical areas of teeth 11 and 21. (B) Periapical radiolucency (the yellow arrow) associated with tooth 21. (C) The apical foramen of tooth 21 showing an estimated opening diameter of approximately 0.4 mm (the yellow arrow). (D) Calcified masses (the yellow arrows) in the pulp chambers of teeth 11, 21. (E,F) Narrow and linear pulp chambers (the yellow arrows) in teeth 16, 26, 36, and 46; incomplete development of the distal roots (the red arrows) of the first permanent molars; teeth 17, 27, 37, and 47 impacted at the distal cervical root area of the first molars. (G,H) Calcified masses (the yellow arrows) in the pulp chambers of teeth 13, 23.

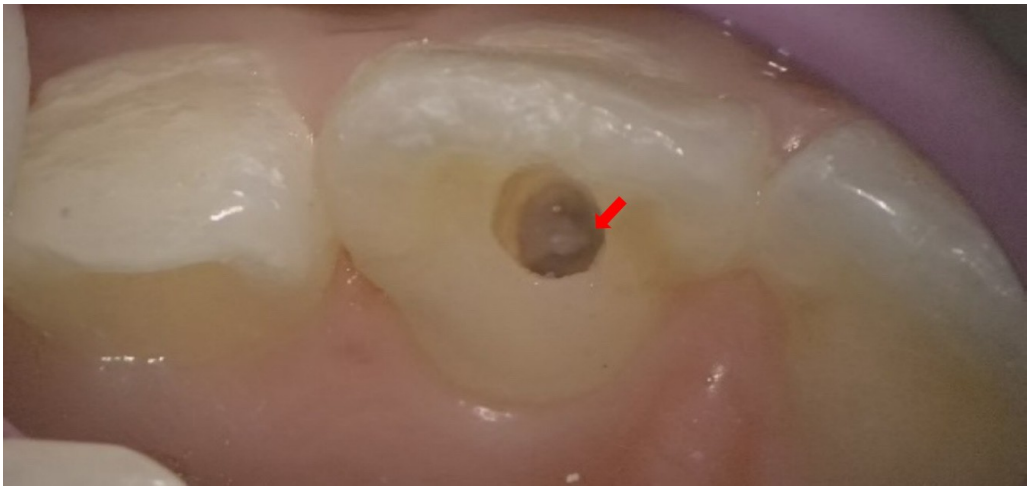

FIGURE 4. Calcified mass (the red arrow) in the pulp chamber of tooth 21 under the microscope.

intracanal medicament, and the access cavity was temporarily sealed with glass ionomer cement. Comprehensive oral hygiene instructions (OHI) were provided to the patient.

At the 3-week follow-up visit, the temporary restoration remained intact. Tooth 21 was asymptomatic, with no tenderness to percussion, no mobility, and healthy gingival tissues. Under microscopic visualization, the temporary restoration was removed, and the canal was re-entered. Canal patency was reconfirmed, and irrigation was repeated using alternating 1% sodium hypochlorite and saline, supplemented with ultrasonic activation. The working length remained unchanged at 22.5 mm. After canal drying, a master cone trial was completed. Final obturation was performed using iRoot SP (Innovative BioCeramix, Vancouver, BC, Canada) in combination with warm vertical condensation of gutta-percha (VDW, Munich, Germany). A postoperative periapical radiograph revealed slight extrusion of iRoot SP beyond the apical foramen but demonstrated a dense and well-adapted root canal filling (Fig. 5). The canal orifice was lined with zinc phosphate cement, followed by enamel etching with 37% phosphoric acid (Etch-37^{TM} wth BAC 0-E5503EBM, Bisco Inc., Schaumburg, IL, USA), application of a universal adhesive (3M, St. Paul, Minnesota, USA), and composite resin restoration (3M, St. Paul, Minnesota, USA). Occlusion was checked and adjusted, and the restoration was polished. Teeth 55 and 85 were left untreated due to their proximity to exfoliation. As the first permanent molars were asymptomatic and exhibited no clinical signs of pathology, they were placed under periodic observation to preserve the child's masticatory function.

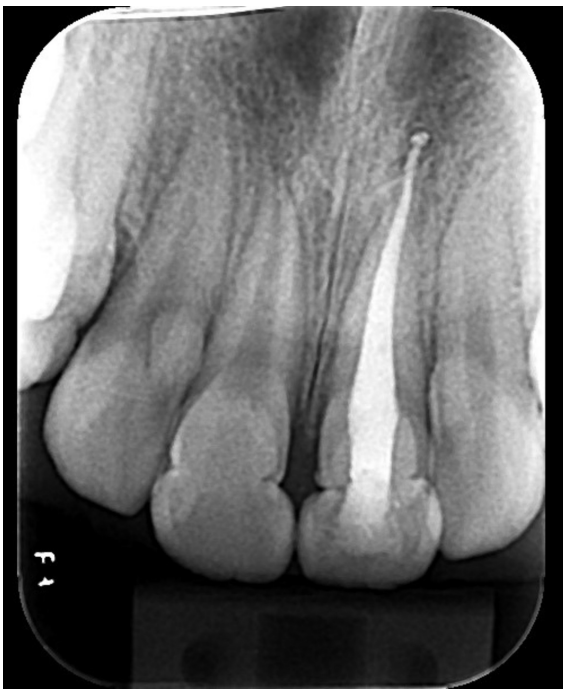

FIGURE 5. Immediate postoperative periapical radiograph after root canal treatment.

At the 10-week follow-up, the patient reported no discomfort. Intraoral examination showed that the restoration on tooth 21 remained intact (Fig. 6, the yellow arrow), with no tenderness to percussion or abnormal mobility; however, plaque and debris accumulation were evident on the cervical region (Fig. 6, the blue arrows). The crowns of teeth 16, 26, 36, and 46 appeared clinically normal (Fig. 6, the red arrows). Periapical radiography of tooth 21 revealed radiopaque filling material within the root canal, with a small amount of sealer visible at the apical region, and no periapical radiolucency was detected (Fig. 7A, the yellow arrow). Panoramic radiography demonstrated no radiographic signs of periapical pathology associated with teeth 16, 26, 36, or 46 (Fig. 7B, the red arrows), and the presence of all four third-molar tooth germs was identifiable (Fig. 7B, the green arrows). The patient was maintained under observation, and meticulous oral hygiene was reinforced.

At the 9-month follow-up, the patient reported no discomfort. Intraoral examination of tooth 21 (Fig. 8) revealed no abnormal clinical findings. The patient's oral hygiene had improved markedly compared with the previous visit. Teeth 13 and 23 were fully erupted and exhibited cervical narrowing at both the mesial and distal aspects of the crowns (Fig. 8, the yellow arrows). Teeth 25 and 35 had erupted as permanent successors, whereas teeth 55 and 85 remained retained at this stage. Panoramic radiography (Fig. 9) demonstrated that tooth 21 remained largely unchanged compared with prior examinations. No radiographic evidence of periapical pathology was identified in teeth 16, 26, 36, or 46. Teeth 55 and 85 were extracted at this visit, and the remaining teeth were maintained under continued clinical and radiographic observation.

The patient's caregiver expressed relief following treatment, noting improvement in dental discomfort and greater confidence in daily oral hygiene management after professional guidance.

A detailed clinical timeline of clinical findings and interventions is summarized in Table 1.

3. Discussion

3.1 Clinical characteristics of MIM

Molar–incisor malformation (MIM) represents a distinct developmental dental anomaly with characteristic morphological features that differ from conventional dental malformations. Clinically, affected first permanent molars typically exhibit normal crown contours, whereas radiographic examination reveals markedly short, thin, or underdeveloped roots. In the incisor region, characteristic findings include cervical enamel defects at the cervical third of the crown and pulpal calcifications or pulp stones. MIM most often presents bilaterally and predominantly involves the first permanent molars (maxillary and mandibular), second primary molars, and central incisors.

Epidemiological data from MIM cases reported between 2014 and 2019 indicate that 98.9% of patients exhibited involvement of the first permanent molars, 39.1% of the second primary molars, 35.6% of the central incisors, and 6.9% of the canines, with a mean patient age of approximately 9 years [1]. In a retrospective study of 38 patients [6], nearly all individuals demonstrated involvement of all four permanent first molars, with only three exceptions. Maxillary central incisors were affected in 17 patients (44.7%), mandibular central incisors in 2 patients, and maxillary canines in 4 pa-

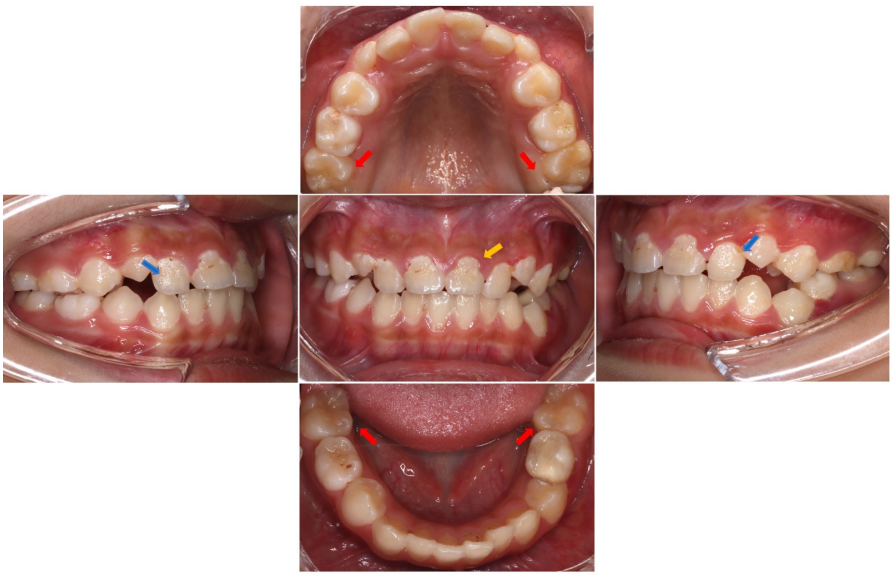

FIGURE 6. Intraoral photographs at the 10-week follow-up. The yellow arrow showing the intact restoration of 21, the red arrows showing the normal crowns of the first permanent molars, and the blue arrows showing plaque and debris accumulation.

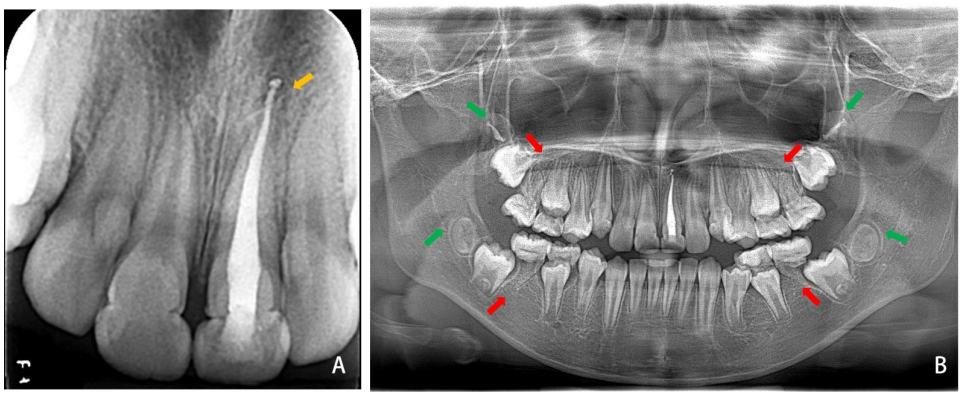

FIGURE 7. Radiographic findings at the 10-week follow-up. (A) Periapical radiograph. The yellow arrow showing no periapical radiolucency. (B) Panoramic radiograph. The red arrows showing no radiographic signs of periapical pathology with teeth 16, 26, 36, and 46, and the green arrows showing the third-molar tooth germs.

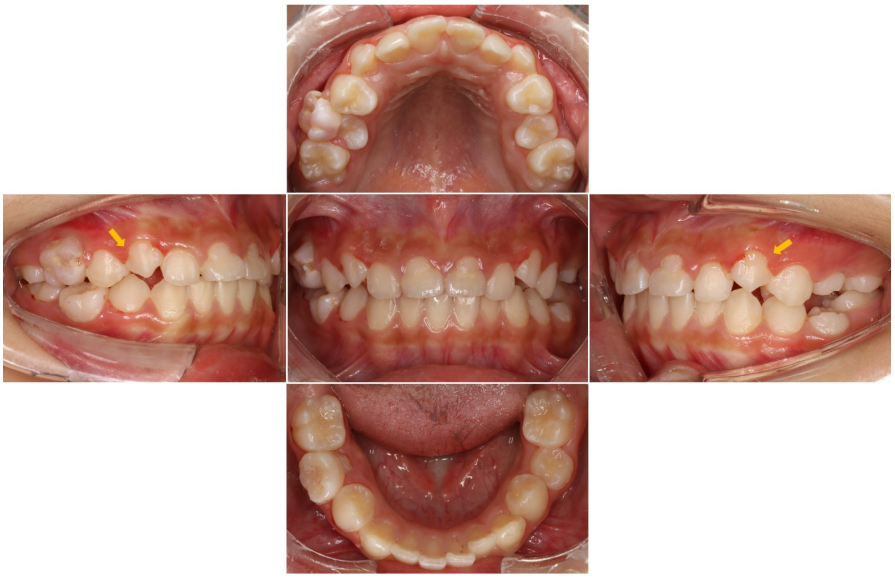

FIGURE 8. Intraoral photographs at the 9-month follow-up. The yellow arrows showing the cervical narrowing of teeth 13 and 23.

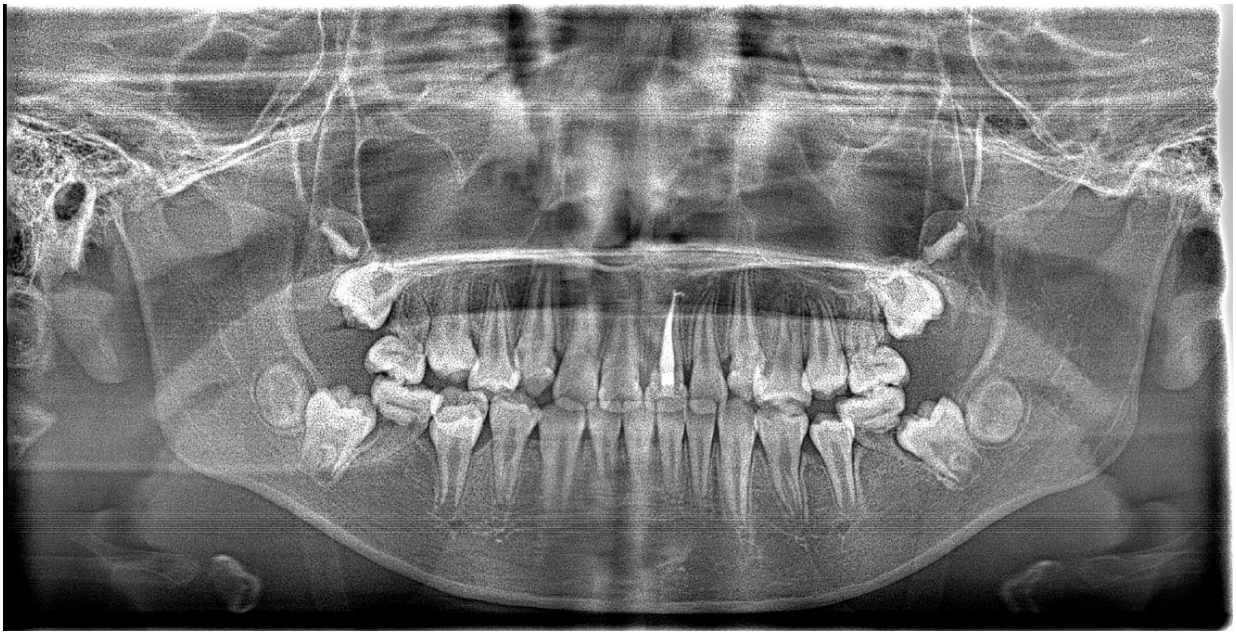

FIGURE 9. Panaramic radiograph at the 9-month follow-up.

TABLE 1. Detailed clinical timeline of the case.

Date	Key clinical findings	Interventions/Management
16 January 2025 (Initial visit)	Pain and discomfort in tooth 21 for 3 days; slight percussion tenderness; mild gingival inflammation; poor oral hygiene. Wedge-shaped cervical defects and pulpal calcifications in teeth 11 and 21. Abnormal root morphology of first permanent molars consistent with MIM. Poor oral hygiene. Periapical radiolucency at the apex of tooth 21; narrow pulp chambers and dysplastic roots of four first molars.	Diagnosis of molar–incisor malformation (MIM). Tooth 21 diagnosed with chronic apical periodontitis with acute exacerbation. Decision to perform root canal therapy of tooth 21.
16 January 2025 (Treatment initiation)	Purulent and bloody exudate released from the pulp chamber of tooth 21; pulp stones present.	Root canal therapy initiated and pulp stones removed ultrasonically. Canal prepared to size 25/06; calcium hydroxide placed; temporary restoration applied. First molars under observation. OHI.
06 February 2025 (3-week visit)	Tooth 21 asymptomatic; no percussion tenderness or mobility.	Final root canal obturation using iRoot SP and warm vertical condensation. Composite resin restoration completed.
18 April 2025 (10-week follow-up)	No symptoms; restoration intact; debris noted at the cervical area. Poor oral hygiene. No periapical radiolucency detected at tooth 21; no radiographic signs of periapical pathology in the first permanent molars.	Continued observation and reinforcement of oral hygiene.
02 November 2025 (9-month follow-up)	Tooth 21 clinically stable; oral hygiene was markedly improved. Stable radiographic appearance of tooth 21 compared with previous examinations; no evidence of periapical pathology in teeth 16, 26, 36, or 46; presence of four third-molar tooth germs on the panoramic radiograph.	Primary teeth 55 and 85 extracted. Long-term observation continued.

OHI: oral hygiene instructions.

tients (10.5%), including mandibular canine involvement in 2 cases (5.3%). Additionally, MIM-related changes were observed in the deciduous molars of 20 patients (52.6%). In the present case of the 9-year-old Chinese female patient, with four first molars involved, the characteristic features of MIM were clearly demonstrated, including cervical constriction of the maxillary central incisors and canines, extensive pulpal calcifications, and markedly underdeveloped roots of the first permanent molars, which were well documented by CBCT imaging.

3.2 Proposed etiology of MIM

The etiology of MIM appears to differ from that of previously described hereditary or environmentally induced dental anomalies. Current evidence suggests that MIM is not primarily driven by classical Mendelian inheritance, and several studies have proposed an association between MIM and systemic conditions affecting the brain and central nervous system during early childhood (approximately 1–2 years of age). In a case series of 12 affected children, 10 had documented medical histories of meningitis, spina bifida, epilepsy, hydrocephalus, or idiopathic encephalopathy occurring within this developmental window [5]. Similarly, an analysis of all reported MIM cases since 2019 identified prematurity (17.2%), meningitis (12.6%), early antibiotic exposure (9.2%), spina bifida (8.0%), stroke (6.9%), hydrocephalus (5.7%), and PHACE Syndrome (posterior fossa brain malformations, hemangiomas, arterial anomalies, cardiac defects and coarctation of the aorta, and eye abnormalities) (4.6%) as the most frequently reported comorbidities. However, approximately 6.9% of patients had no significant medical history [1]. A recent five-case series further supported a non-genetic etiology of MIM, as affected children presented with heterogeneous systemic conditions—including pelvicaliectasis with nephrological follow-up, hemolytic uremic syndrome, severe neonatal hypertension requiring neonatal intensive care unit (NICU) admission, and multisystem disorders such as Williams syndrome—while no familial aggregation was identified [7]. Based on these observations, it is hypothesized—though not conclusively demonstrated—that vascular impairment or viral infection or systemic diseases during early development may interfere with root formation, leading to the characteristic dental phenotype.

In the present case, a detailed review of the patient's medical history did not reveal any documented systemic diseases involving the brain or central nervous system during the critical period of 1–2 years after birth. Several prenatal and perinatal conditions were, however, identified, including maternal exposure to a freshly painted latex environment during early pregnancy, severe hyperemesis throughout gestation, and maternal calcium deficiency. In addition, the patient was diagnosed with a large hemangioma on the left leg at birth, which did not require surgical or pharmacological intervention. These factors are reported here as case-specific observations. Importantly, there is currently no direct evidence supporting a causal relationship between these prenatal or perinatal exposures and the development of MIM, and similar exposures are known to occur in many individuals who do not develop this condition [8].

To explore a potential genetic contribution to MIM in this patient, whole-exome sequencing was performed on peripheral blood as an exploratory analysis. Variant filtering was conducted using predefined criteria, including a minor allele frequency $\leq$0.05 across six population databases (Genome Aggregation Database (gnomAD) exome_ALL, gnomAD_exome_EAS, gnomAD_genome_ALL, gnomAD_genome_EAS, gnomAD3_genome_AF, and gnomAD3_genome_AF_EAS) or previously reported pathogenicity in ClinVar or the Human Gene Mutation Database (HGMD). Using these criteria, more than 10,000 candidate variants were initially identified (**Supplementary Table 1**). When the analysis was further restricted to genes associated with the phenotype of "central nervous system abnormalities", three candidate genes—*centrosomal protein 85 like* (*CEP85L*), *Abelson murine leukemia viral oncogene homolog 1* (*ABL1*), and *DEP domain containing 5* (*DEPDC5*)—remained (**Supplementary Table 2**). However, due to the absence of family-based segregation analysis, as the parents were temporarily unwilling to undergo genetic testing, and the lack of functional validation experiments, the pathogenic relevance of these variants could not be determined. Therefore, no definitive causative mutation or genetic etiology could be established in this case. Further studies incorporating family-based sequencing and functional analyses will be required to clarify whether genetic factors contribute to the pathogenesis of MIM in a subset of patients.

Recent studies have also suggested a potential association between MIM and ciliary dysfunction resulting from mutations in the *T-complex testis expressed 1 domain 2* (*TCTEX1D2*) gene during tooth development in a subset of patients [9]. Taken together, existing evidence indicates that MIM is likely a heterogeneous condition, potentially involving interactions between early-life systemic disturbances and genetic susceptibility. However, at present, these relationships remain largely associative, and the precise pathogenic mechanisms underlying MIM require further investigation.

3.3 Pathogenesis of MIM and pulpal calcifications

The formation of calcified masses within the pulp chambers of MIM-affected teeth has been attributed to early developmental disturbances during crown and root formation. It has been proposed that early injury to the vascular plexus at the base of the dental papilla disrupts normal tissue homeostasis, allowing serum or tissue fluid to infiltrate the follicular interstitial space and subsequently precipitate calcified spherules. These spherules may coalesce to form an ectopic mineralized structure known as the cervical mineralized diaphragm (CMD), which interferes with normal dentin mineralization and root morphogenesis by acting as a physical barrier at the crown–root junction [4]. Micro-computed tomography and histological studies of exfoliated MIM-affected molars have consistently demonstrated that the CMD exhibits a radiodensity intermediate between enamel and dentin and is primarily composed of hydroxyapatite-rich, amorphous hard tissue [10, 11]. Histological analyses further revealed that the pulpal floor of MIM-affected molars is composed of hetero-

geneous hard tissues, including dentin-, bone-, and cartilage-like components with abundant vascularization, suggesting inappropriate differentiation of apical pulp and dental follicle cells during tooth development [10–12]. At the dentin–pulp interface, irregular crystal-like deposits and reduced dentinal tubule density have been observed, supporting the notion that some of these calcified structures arise from passive mineral precipitation, rather than regulated cellular processes. Collectively, these findings indicate that CMD formation and associated pulpal calcifications create structural vulnerability at the crown–root junction, impair normal root morphogenesis, and increase susceptibility to pulpal and periapical pathology. In the present case, extensive pulpal calcifications were identified not only in the affected maxillary central incisor, but also in other teeth, supporting the hypothesis that aberrant cervical hard tissue formation may compromise pulpal integrity and predispose MIM-affected teeth—particularly tooth 21—to subsequent pulpal and periapical disease.

3.4 Differential diagnosis of MIM

In clinical practice, early recognition and diagnosis of MIM remain challenging, but are essential for appropriate management. Several pediatric dental conditions—including molar–incisor hypomineralization (MIH), amelogenesis imperfecta, and hereditary dentin dysplasia—may present with overlapping clinical or structural features, underscoring the need for careful differential diagnosis. MIH is a qualitative enamel defect of systemic origin, affecting at least one of the four first permanent molars and frequently involving the incisors. Clinically, it is characterized by demarcated enamel opacities larger than 1 mm in diameter, with altered translucency and color ranging from creamy white to yellow or brown. These lesions typically occur on the smooth buccal or lingual surfaces of first permanent molars and incisors [13, 14]. In contrast, MIM involves the same tooth groups, but is distinguished by normal coronal enamel mineralization. Affected incisors typically exhibit cervical constriction accompanied by intrapulpal calcifications, while the first permanent molars show markedly shortened, malformed, or absent roots. Despite superficial similarities in tooth distribution, these distinctive structural features—particularly the combination of normal enamel with severe root dysplasia in first molars—are critical for differentiating MIM from MIH.

Amelogenesis imperfecta (AI) is a group of hereditary enamel disorders and is typically classified into four major types: hypoplastic, hypocalcified, hypomaturation, and taurodontic forms [15]. Affected teeth typically exhibit generalized enamel defects, including surface pitting or depressions, with discoloration ranging from white to yellow-brown. The enamel may be of normal thickness or reduced and is often soft and hypersensitive. Both primary and permanent dentitions are usually involved, affecting multiple tooth types throughout the dentition [16]. Radiographically, the enamel in AI demonstrates radiopacity similar to or slightly lower than that of dentin. In contrast, MIM can be readily distinguished from AI by its localized pattern of involvement, preservation of coronal enamel mineralization, and characteristic radiographic findings, particularly the presence of cervical constriction and severe root malformations.

Dentin dysplasia (DD) is an autosomal dominant hereditary disorder of dentin formation and is clinically classified into type I and type II. Type I DD, also referred to as radicular dentin dysplasia, primarily affects root development, whereas type II DD, or coronal dentin dysplasia, predominantly involves the crowns [17]. Type I DD should be carefully differentiated from MIM. Clinically, affected teeth typically present with normal crown morphology and color, but exhibit increased mobility and a tendency toward premature exfoliation. Radiographically, type I DD is characterized by short, blunt, and malformed roots, with complete obliteration of the pulp chambers and crescent-shaped pulp remnants parallel to the dentino-enamel junction [18]. Although the short roots and obliterated pulp chambers observed in type I DD may resemble features of MIM, DD can be distinguished by its hereditary nature and its generalized involvement of the entire dentition, in contrast to the characteristic predilection of MIM for the first permanent molars and incisors.

In this case, normal enamel mineralization, characteristic cervical constriction, pulp calcifications, and severe root underdevelopment of the first permanent molars allowed differentiation of MIM from conditions such as MIH, AI, and DD.

3.5 Management considerations for affected teeth

Management strategies for MIM differ between affected incisors and first permanent molars. Maxillary central incisors with cervical constriction not only compromise esthetics, but also carry an increased risk of pulp infection and traumatic fracture with pulp exposure. Asymptomatic incisors may be preserved with appropriate coronal restorations, whereas teeth presenting with pulpitis or apical periodontitis may require endodontic treatment or, in severe cases, extraction [19]. In the present case, apical periodontitis of tooth 21 was likely associated with plaque accumulation and soft debris retention at the malformed cervical region, where abnormal structural interconnections with CMD dentin may facilitate bacterial ingress. The inflammatory process subsequently involved the pulp tissue and progressed to the periapical region. Given that maxillary central incisors usually have a single and relatively straight root canal, successful root canal therapy is often achievable. Considering the near-complete morphological closure of the apical foramen of tooth 21 and the relatively mild cervical enamel defect, nonsurgical root canal therapy was selected. Strict oral hygiene measures and regular follow-up were emphasized, and a definitive full-coverage restoration is recommended once craniofacial growth is complete. At the 9-month follow-up, tooth 21 remained asymptomatic with stable clinical and radiographic findings, indicating a favorable outcome. Consistent with this observation, among 17 reported MIM patients with incisor involvement, two experienced incisor loss, whereas four successfully retained their incisors following endodontic or restorative management [6].

For first permanent molars without clinical symptoms, an initial conservative approach with careful observation and periodic follow-up may be appropriate. In symptomatic cases, attempts at root canal therapy have been reported to preserve

affected molars. A retrospective study of 38 MIM patients documented successful endodontic preservation of 10 first permanent molars [6]. In another case report, root canal treatment of bilateral mandibular first molars resulted in a favorable outcome on one side, while treatment failed on the contralateral molar due to severely calcified and non-negotiable canals [20]. Nevertheless, extraction has frequently been advocated for symptomatic MIM-affected first permanent molars when performed at approximately 8–9 years of age, to facilitate spontaneous mesial migration of the second permanent molar, particularly in patients with developing third molars [21–23]. This preference largely reflects the challenges posed by abnormal root morphology and complex canal configurations, which substantially reduce the predictability and success of endodontic treatment.

In the present case, management was individualized based on clinical findings. Root canal therapy was performed for the symptomatic maxillary central incisor with a nearly closed apex, while asymptomatic first permanent molars were managed conservatively to maintain occlusal stability and masticatory function. Extraction of first permanent molars may be considered if symptoms develop or if eruption of the second molars is compromised.

This case report has several strengths. To our knowledge, this is the first reported case of molar–incisor malformation in a Chinese pediatric patient. In addition, genetic testing was performed to explore potential pathogenic variants, providing preliminary molecular insights into this rare condition. Comprehensive clinical management was implemented, including microscopic endodontic treatment, CBCT-based assessment, oral hygiene intervention, and long-term monitoring of both affected and at-risk teeth, emphasizing a whole-dentition management approach. Nevertheless, several limitations should be acknowledged. The follow-up period was limited to nine months, and longer observation is necessary to evaluate long-term outcomes, particularly for the first permanent molars. Moreover, family-based genetic analysis could not be completed because the patient's parents declined further genetic testing, limiting definitive conclusions regarding the genetic etiology of MIM in this case.

Building on these considerations, this case provides several clinically relevant lessons. First, MIM should be considered in young patients presenting with cervical constriction of incisors and unexplained pulpal or periapical pathology, even in the absence of obvious enamel hypomineralization. Second, poor oral hygiene may exacerbate the vulnerability of malformed cervical structures, facilitating microbial ingress and subsequent pulpal involvement. Third, affected incisors with relatively simple canal anatomy and near apical closure may be successfully managed with conventional root canal therapy. Finally, asymptomatic first permanent molars may be managed conservatively with regular monitoring, while extraction should be reserved for cases with severe symptoms or unfavorable root morphology. Collectively, this case underscores the importance of early recognition, individualized treatment planning, and long-term surveillance in the management of MIM.

4. Conclusions

In conclusion, the etiology of MIM remains incompletely understood, which limits effective prevention strategies. Clinically, MIM-affected teeth are prone to impaction, premature tooth loss, space loss, spontaneous pain, and esthetic concerns, posing significant challenges for diagnosis and management. Awareness of the characteristic clinical and radiographic features of MIM is, therefore, essential to avoid misdiagnosis. Early identification, regular follow-up, meticulous oral hygiene supervision, and timely, individualized intervention are critical to optimizing long-term outcomes in affected children.

AVAILABILITY OF DATA AND MATERIALS

All data supporting the findings of this case report are included in the submitted article and its supplementary materials. The whole-exome sequencing results are provided in **Supplementary Tables 1 and 2**. Raw sequencing data are not publicly available due to patient privacy considerations but may be made available from the corresponding author (QS) upon reasonable request.

AUTHOR CONTRIBUTIONS

QS—made substantial contributions to the conception and design of the case report, served as the primary clinician responsible for the patient's treatment, and participated in drafting the manuscript. XXL—was responsible for data acquisition, organization, and interpretation, prepared all figures and tables, coordinated manuscript submission, and led the revision and language editing process. YTZ—drafted the initial version of the manuscript, conducted an extensive literature search and critical review, and contributed substantially to the organization and scientific content of the manuscript. ZFW—provided senior academic supervision, participated in the discussion of the treatment strategy, guided the scientific framing and structure of the manuscript, advised on journal selection, and critically revised the manuscript for important intellectual content. All authors contributed to editorial revisions, read, and approved the final version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Since this is a case report and does not involve research or clinical trials, the Medical Ethics Committee of Stomatology Hospital of Zhejiang University School of Medicine has exempted it from the requirement of ethical approval. This case report was conducted in accordance with the principles of the Declaration of Helsinki. All clinical procedures were performed as part of routine dental care, and written informed consent for treatment was obtained from the patient's legal guardian.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at https://oss.jocpd.com/files/article/2072931983342616576/attachment/Supplementary%20material.xlsx.

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