Bilateral root absence of permanent first molars: literature review and case report

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Abstract

Aim This case report and literature review aim to elucidate the rare occurrence of bilateral root absence in permanent first molars, emphasizing its clinical implications, diagnostic challenges, management, and possible association with systemic conditions. A unique case involving a 13-year-old male diagnosed with atypical hemolytic uremic syndrome (aHUS) is presented to illustrate these points.

Methods A comprehensive review of existing literature was conducted to explore the etiology, diagnosis, and management strategies for root absence. In addition, the detailed case study of the patient with aHUS and root absence of all four permanent first molars complements the review, providing insights into systemic impacts on dental development.

Results The review highlights systemic diseases, including aHUS, as significant etiological factors contributing to dental anomalies such as root absence. The presented case underscores the importance of a thorough differential diagnosis and consistent monitoring, especially in patients with systemic conditions.

Conclusion This report emphasizes the need for a multidisciplinary approach in diagnosing and managing dental anomalies associated with systemic diseases. Further research is recommended to understand the systemic influences on dental root development and improve patient outcomes.

KEYWORDS Root absence, aHUS, Dental anomalies.

Introduction

Bilateral root absence of permanent first molars is a rare dental anomaly with significant implications for dental function, stability, and overall oral health. This condition, marked by either incomplete root formation (root hypoplasia) or the pathological loss of previously developed roots (root resorption), poses diagnostic and therapeutic challenges [Kun, 2007]. Root hypoplasia refers to a developmental defect where the roots fail to fully form, leading to shortened or absent roots, which can compromise the tooth's longevity and resistance to normal occlusal forces [Tewari et al., 2010]. In contrast, root resorption is a progressive loss of root structure after development, often triggered by systemic conditions, trauma, or orthodontic treatment [Nasehi, Nazhari & Mohtasam, 2015; Khojastepour, Bronoosh & Azar, 2010]. This condition can manifest in any tooth, but it holds particular clinical significance when it affects permanent first molars due to their crucial role in occlusal function and alignment. Understanding the underlying causes of root absence, such as genetic predispositions, local inflammatory responses, or systemic influences, is crucial for appropriate management. Although epidemiological data on bilateral root absence of permanent first molars are scarce due to the rarity of this anomaly, studies estimate that true root developmental anomalies—such as root hypoplasia or agenesis—occur in less than 1% of the general population, often associated with syndromic or systemic conditions [Luder, 2015; Liang et al., 2003]. Atypical hemolytic uremic syndrome (aHUS) is an ultra-rare, complement-mediated thrombotic microangiopathy with an estimated incidence of 0.23–0.42 cases per million population per year in children [Loirat & Frémeaux-Bacchi, 2011; Raina et al., 2019]. It is most often diagnosed in infancy or early childhood and has been associated with multi-organ damage, including renal impairment and inflammatory complications, both of which may affect dental development. The focus of this paper is to comprehensively present the existing literature on root absence, discuss contemporary scientific theories concerning its etiology, and provide an updated review of the literature. Additionally, this paper highlights a unique case of bilateral root absence affecting all four permanent first molars in a 13-year-old Caucasian male. This patient, who has a medical history of aHUS, was referred to our Department for further evaluation and management. The exploration of this case, alongside a review of the literature, aims to enhance the understanding of root absence's underlying mechanisms and implications for dental treatment planning.

Literature Review

Timing and Morphogenesis

Root formation, also known as radiculogenesis or rhizagenesis, is the development of the root pulpo-dentinary organ, closely associated with cementogenesis, the outline of the dentoalveolar ligament, and the formation of the alveolar bone. After crown formation, in the advanced bell stage, the enamel organ exhibits a bilayered Hertwig's epithelial root sheath (HERS), which grows apically to determine the number, shape, and size of the roots [Kumar, 2015; Miletich et al., 2011]. Abnormalities in the size and shape of the tooth arise from disturbances during the morphodifferentiation (cap-bell) stage of tooth development. Morphogenesis is regulated by enamel knot formation during the bud stage, and if disrupted, it can lead to improper cusp formation or the arrest of tooth formation. The root shape is determined after crown formation during the advanced bell stage, with HERS playing a pivotal role in the process [Kumar, 2015; Miletich et al., 2011]. Root development is regulated by the interactions between HERS, the basement membrane, mesenchymal papilla, and the dental follicle. HERS originates from the cervical loop, where the external and internal enamel epithelium meet to form a double epithelial layer. HERS has an annular structure surrounded by a basal membrane that separates it from the pulp and follicular mesenchyma. As HERS grows in a centripetal direction, it forms rings from which the roots are identified, and the number of roots correlates with the number

of epithelial "tongues" produced. For example, molars with two roots have two epithelial tongues that fuse to form two rings, leading to the formation of two roots. These two epithelial layers remain attached and progress apically, shaping the future dental roots. Root elongation and tissue formation are related to the coordinated proliferation of sheath epithelial cells and surrounding mesenchymal cells [Luan, Ito & Diekwisch, 2006]. Root dentin forms in parallel with the apical proliferation of HERS. The latter induces odontoblastic differentiation, and pulp parenchyma cells near the anchor fibrils differentiate into odontoblasts, which produce predentin that mineralises to form dentin. However, the outer dental epithelium of HERS does not differentiate into ameloblasts like the crown. As the basement membrane degrades, HERS disintegrates, and follicular cells near the root dentin differentiate into cementoblasts, which synthesise and deposit the cement matrix. As root development progresses, the HERS gradually shrinks, reducing the size of the root tube and allowing for the formation of the root apex [Rhrich & Aghoutan, 2020]. Root formation is a slow process. For the first permanent molar, root formation continues until about 9–10 years of age. In permanent teeth, root development takes longer and requires more time than crown development [Rhrich & Aghoutan, 2020]. The basic processes of root formation (Figure 1) include: (1) the development of HERS associated with the transition from crown to root development, (2) apical growth of HERS associated with root elongation, (3) induction of odontoblast differentiation and radicular dentinogenesis, (4) disintegration of HERS and initiation of cementogenesis, and (5) formation of acellular and cellular cementum [Luder, 2015]. The development of the root of the permanent first molar begins after crown morphogenesis is completed, with root formation initiated by the development of HERS from the cervical loop. This process starts in the early postnatal stages and continues as the tooth erupts into the oral cavity. A special process confined to the development of multirooted teeth is the formation of bifurcation or trifurcation. Tongue-shaped epithelial projections from the cervical loop, which remain inactive during crown formation, proliferate and unite when the root trunk divides, forming a continuous bridge. These epithelial bridges induce odontoblast differentiation and dentin production at the pulp cavity floor, similar to HERS. However, recent studies, such as Kim et al. (2015), indicate that odontoblast differentiation and dentin formation in the furcation area critically depend on osterix, suggesting that specific regulatory mechanisms might control furcation formation [Kim et al., 2015].

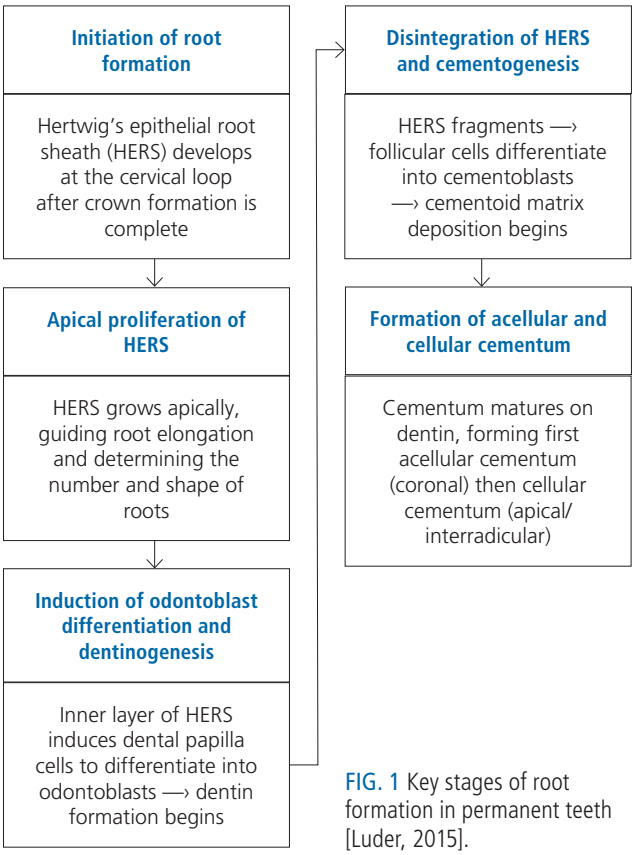

FIG. 1 Key stages of root formation in permanent teeth [Luder, 2015].

Molecular Factors and Genes Involved in Root Development

The root development process in teeth is orchestrated by a complex network of signaling cascades involving various growth factors and transcription factors. Molecular insight into odontogenesis shows the active involvement of signaling families, including fibroblast growth factor (FGF), transforming growth factor-β (TGF-β), Wingless-related integration site (Wnt), and Sonic Hedgehog (Shh), which regulate the expression of transcription factors such as Msx, Pax, and Runx families. FGF8 from the lateral oral epithelium and BMP4 from the medial oral epithelium differentially regulate Dlx1, Dlx2, Barx1, Msx1, and Msx2, forming the "Odontogenic homeobox code" for tooth identity. In tooth root development, Pax9 and Barx1 interact with Msx1 to regulate BMP4 protein secretion. These interactions are crucial for the development of multicusped teeth. Failures in these interactions can result in tooth development arrest at the bud stage due to insufficient levels of BMP and enamel knot signaling [Kumar, 2015; Miletich et al., 2011]. Bone Morphogenetic Proteins (BMPs) and Epidermal Growth Factor (EGF) are essential for influencing cellular processes such as division, differentiation, and apoptosis, which are critical for tooth development. Insulin-like Growth Factor (IGF) and Fibroblast Growth Factor (FGF) also significantly contribute, with IGF promoting cellular proliferation within HERS, which is essential for root elongation, while FGFs facilitate critical epithelial-mesenchymal interactions necessary for tooth morphogenesis. Key transcription factors such as Msx1, Msx2, and Runx-2, along with Sonic Hedgehog (Shh), regulate the transcriptional activities required for the differentiation of odontoblasts and cementoblasts, impacting the mineralisation of dentin and cementum. Additionally, enamel proteins secreted by HGH cells, along with follistatin and activin A, interact with BMP signaling to guide the differentiation and functional behaviors of cells involved in tooth root development [Heikinheimo et al., 1998; Lan et al., 2014]. This orchestrated signaling ensures the precise development and functional maturation of the tooth root [Dassule et al., 2000; Yamashiro, Tummers and Thesleff, 2007; Koyama et al., 2001; Nakao et al., 2009].

Etiology of Root Absence

Short and/or misshapen roots are most often due to hard tissue resorption. Such secondary abnormalities, which usually affect single teeth or small groups of teeth, are frequently a consequence of dento-periodontal traumas, local periodontal inflammation, or orthodontic tooth movement involving excessive forces [Tronstad, 1988]. Irrespective of the cause, root resorption constitutes an inflammatory reaction and, therefore, its consequences are not typically considered a malformation. The most common true human root malformations can be subdivided into disorders of root development alone and disorders of root

Root resorption		Root hypoplasia		
Internal	**External**	**Genetic predispositions**	**Systemic diseases**	**Environmental exposures**
Chronic inflammation or pulpal infection	Inflammatory conditions (Periapical or periodontal inflammation)	Hereditary conditions: - dentin dysplasia - amelogenesis imperfecta - ectodermal dysplasia Specific Gene Mutations	Congenital and Systemic Disorders: - congenital syphilis - hypoparathyroidism	Traumatic injuries to primary teeth
Orthodontic movement	Mechanical stimulation (Orthodontic therapy, Occlusal stress)		Nutritional Deficiencies: - a deficiency of Vitamin D (rickets) - Insufficient levels of calcium (hypocalcemia)	Dental fluorosis during tooth development
Herpes zoster	Luxation injuries (Dental Trauma)		Endocrine disorders: - hypothyroidism	Radiation and Chemotherapy in pediatric cancer patients
Idiopathic factors	Neoplastic conditions (Tumors). Impacted and supernumerary teeth			
	Transplantation and reimplantation			
	Endocrine disturbances and systemic conditions: hypoparathyroidism - hyperparathyroidism - hypocalcemia - Gaucher's disease - Paget's disease - hypophosphatemia - hyperphosphatemia - Stevens–Johnson syndrome - odontodysplasia - dentin dysplasia - dentinogenesis imperfecta - Langerhans cell histocytosis - Goltz syndrome - Papillon-Lefevre syndrome - anachoresis - Turner syndrome - dietary habits			

TABLE 1 The possible causes of root absence according to the literature [Nasehi, Nazhari & Mohtasam, 2015; Khojastepour, Bronoosh & Azar, 2010].

development associated with general tooth dysplasia.

Root hypoplasia is influenced by various factors, including genetic predispositions, environmental exposures, and systemic diseases. Beyond well-known systemic, genetic, and local inflammatory causes, recent evidence suggests that metabolic conditions such as obesity may also influence the prevalence of dental anomalies, possibly through altered endocrine signaling and inflammatory pathways [Simsek et al., 2019].

Table 1 shows the etiological factors that can cause the absence of tooth root development or lead to its resorption while Figure 2 schematically differentiates root resorption and root hypoplasia.

Case report

A 13-year-old male patient presented to the Department of Paediatric and Preventive Dentistry, University of Zagreb with the primary concern of persistent second deciduous molars lacking permanent successors. The patient's medical history is notable for aHUS, diagnosed at one year of age. The initial presentation included fever, anemia, and thrombocytopenia. The condition progressed to chronic renal failure, which subsequently led to the development of arterial hypertension. Genetic analysis revealed a mutation in the C3 complement component gene, as well as polymorphisms in regulatory genes. Initial management included plasmapheresis, corticosteroids, and hemodialysis. The patient was later transitioned to continuous eculizumab therapy, which was replaced by ravulizumab at the age of 12. Additionally, at four years of age, the patient underwent a three-month course of inhaled corticosteroid therapy with fluticasone for transient symptomatic obstructive bronchitis. Laboratory tests, including a complete blood cell count and assessments of electrolytes, calcium, phosphorus, and alkaline phosphatase levels, all returned to normal values. The patient had no history of dental treatment, trauma, or oral health issues. His oral hygiene was satisfactory,

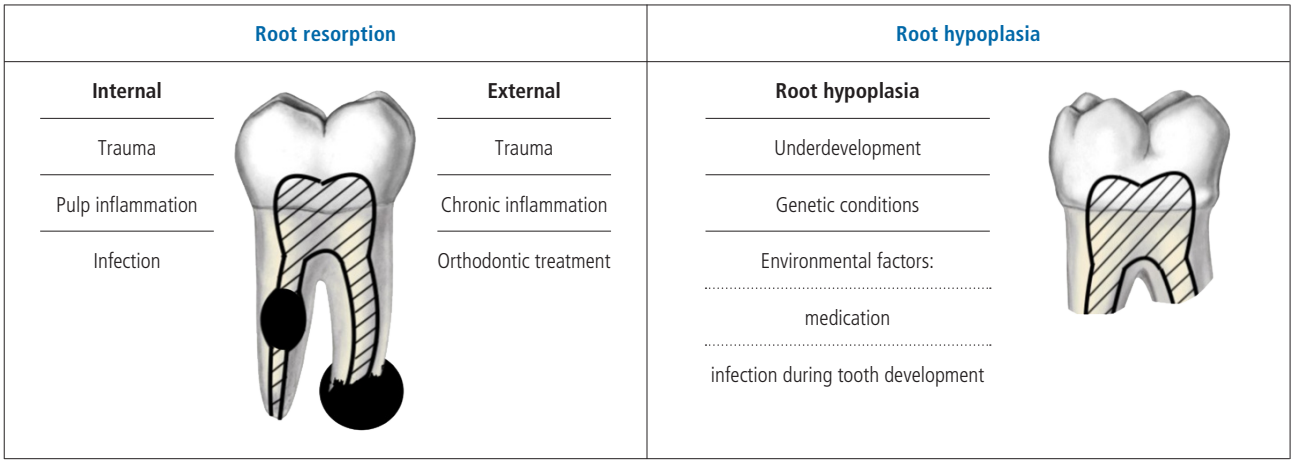

FIG. 2 Schematic presentation of root resorption and root hypoplasia.

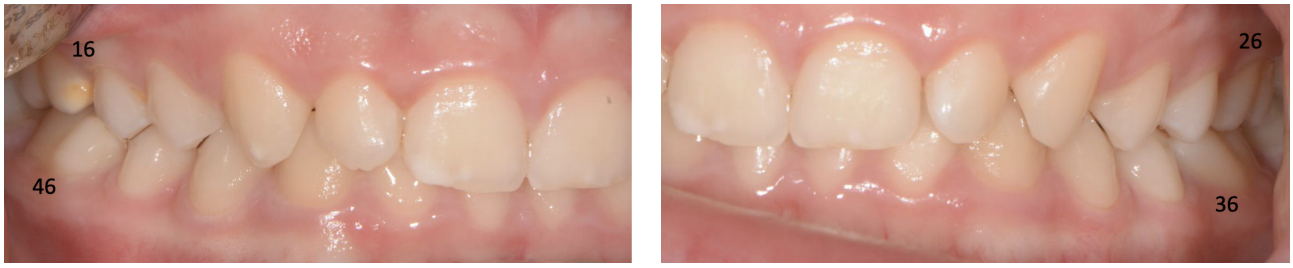

FIG. 3 Intraoral findings.

with no caries, parafunctional habits such as bruxism, wear facets, or premature contacts detected. There was also no family history of early primary tooth loss, abnormal root resorption, or spontaneous loss of permanent teeth. Additionally, the patient had no history of tooth sensitivity to temperature changes, spontaneous pain, or pain during chewing. Upon clinical examination, the gingival tissues around the affected teeth appeared normal in both color and texture. Clinical and dental evaluations showed healthy soft tissues with no supra- or subgingival calculus or abnormal pocketing. There was no history of orthodontic treatment, and occlusion was normal, with a Class I molar and canine relationship, normal overjet, and overbite. No occlusal interferences or tooth mobility were observed. The affected teeth responded normally to electrical and heat pulp testing. Both percussion and palpation yielded unremarkable results. Based on the patient's history, clinical examination, and radiographic findings, a diagnosis of multiple idiopathic apical root resorption was made. The patient was advised to maintain proper oral hygiene and periodic follow-ups were recommended. All teeth had vital pulps, with no signs of periodontal or periapical inflammation. The resorption was detected incidentally on a panoramic radiograph, with the patient being completely asymptomatic. No local etiologic factors were identified, and the teeth and periodontium appeared clinically normal. Extraoral examination did not reveal any significant abnormalities. However, intraoral examination showed the absence of deciduous teeth and the presence of fully erupted permanent first molars, as shown in Figure 3. Diagnostic imaging, particularly an orthopantomogram (OPG), revealed atypical root morphology in the first permanent molars (teeth 16, 26, 36, and 46). These teeth had normal-sized crowns but displayed abnormal root development, as depicted in Figure 4. This condition, involving root hypoplasia and/or resorption, is relatively rare. The OPG also indicated incomplete root formation in the distal roots of teeth 36 and 46, as well as the palatal root of tooth 16. For tooth 26, root development was not clearly visible, likely due to the overlap with the maxillary sinus. The second lower molars were nearly fully developed, while the upper second molars showed wide pulp chambers, indicating ongoing root development. The follicles of the upper third molars and the left lower third molar were also observed. Notably, the lower right first molar had a Class I composite restoration, and remnants of fissure sealant were present in the central and lingual fissures of the lower left first molar. The upper molars exhibited some pigmentation but had no cavities or restorations. Aside from demineralisation pigmentation on the incisors and canines, there were no other pathological findings in the hard dental tissues. All teeth were vital with no abnormal mobility. Since no restorative or orthodontic treatment was necessary, further radiographic imaging was not scheduled, in accordance with the ALARA (As Low As Reasonably Achievable) principle. Given that all teeth remained vital, it was recommended that the patient undergo periodic dental monitoring to preserve tooth structure and function for as long as possible. The focus was placed on regular check-ups and preventive care to prevent potential complications. If increased tooth mobility occurred, extraction and age-appropriate prosthetic rehabilitation could be considered. As the patient was clinically asymptomatic, his parents declined further PA radiographs to assess any progress as well as further histological or bacteriological analysis.

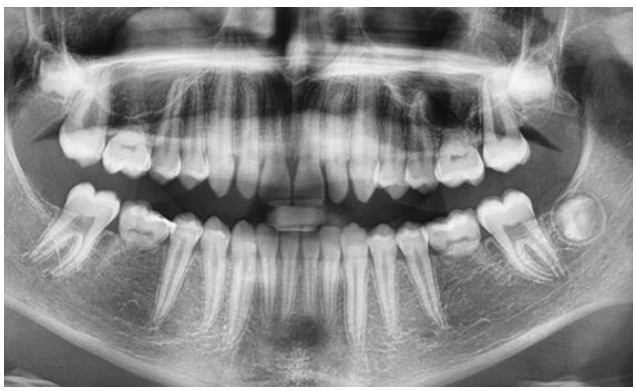

FIG. 4 Panoramic radiograph showing the root abasence of permanent first molars.

Discussion

In the field of dental research, variations in root anatomy and their developmental anomalies have garnered significant attention. For instance, numerous studies have emphasised the prevalence and characteristics of additional roots in permanent molars [Chandra et al., 2011; Garg et al., 2010]. Additionally, the process of root resorption, as explored in earlier study [Al-Junaid & Acharya, 2018], highlights conditions that may mimic or exacerbate root absence in permanent first molars. Proper differentiation between these conditions is essential for accurate diagnosis and treatment planning. Idiopathic root resorption (IRR) presents with a wide range of clinical features, typically affecting a single tooth or a localised region in the dentition. Stafne and Slocumb [1944] evaluated 179 cases of IRR and found that in most instances, only a single tooth was involved, with more than one tooth being affected in only 19 of the cases [Stafne & Slocumb, 1944]. However, in our case, all four permanent first molars are affected, with complete root absence in all involved teeth, marking a distinct and more extensive presentation than the typical isolated or localised forms reported in the literature. Liang et al. reviewed multiple idiopathic root resorption (MIRR) cases and noted that most instances were asymptomatic and

discovered incidentally during routine radiographic examinations [Liang et al., 2019]. However, some patients reported cold sensitivity, loss of restorations, tooth mobility, and tenderness in surrounding tissues. In contrast, our patient does not exhibit any signs of mobility, tenderness, or sensitivity in the affected teeth, further differentiating this case from the typical MIRR presentation. Localised root resorption is typically confined to one to three posterior teeth, while multiple tooth involvement usually affects more than three teeth in a symmetric pattern. In many cases of multiple tooth resorption, the process progresses, eventually leading to the loss of teeth [Liang et al., 2019]. However, in our patient, while there is extensive bilateral root absence affecting all four permanent first molars, the teeth remain vital, with no mobility or tenderness, suggesting a more stable and non-progressive condition. Molar Incisor Malformation (MIM) is typically identified by the presence of underdeveloped, short, narrow, spiky roots, primarily affecting the permanent first molars and sometimes the permanent central incisors [Kim et al., 2019]. In addition to these features, MIM can lead to significant complications such as tooth devitalisation, abscess formation, and eventual bone loss, making early diagnosis with radiographic imaging crucial for preventing further pathology. However, in our case, the presentation differs from the typical characteristics of MIM. While the teeth in our case display the root abnormalities typically associated with MIM, such as shortened and underdeveloped roots, the key distinction is that the teeth remain vital, with no signs of pulp necrosis, abscess formation, or associated bone loss. Furthermore, the patient does not exhibit any signs of pathology typically associated with MIM, such as pulp obstruction or the need for endodontic treatment. This suggests that, despite the presence of abnormal root development, the teeth have retained their vitality and function without the complications commonly seen in MIM. Some reports have linked root hypoplasia to traumatic incidents affecting primary dentition, which may lead to partial or complete arrest of root formation in permanent teeth [Andreasen et al., 2018]. However, our patient's medical history does not indicate any trauma to the primary dentition, making this an unlikely cause. Furthermore, it would be highly unusual for trauma to affect all four primary second molars symmetrically, further distancing our case from this etiology. Additionally, Andreasen et al. discuss how certain traumatic events during primary dentition may cause a temporary arrest in root formation in permanent teeth, which can lead to root resorption and abnormalities [Andreasen et al., 2018]. While this might explain some isolated cases of root resorption, our patient does not exhibit any occlusal anomalies, aesthetic issues, or functional impairment, which are commonly associated with root resorption resulting from trauma. The lack of such abnormalities in our case further supports the need for a different diagnosis. Our case presents a unique manifestation of multiple roots affected, involving bilateral complete absence of all four permanent first molars without trauma, mobility, or functional impairment. This diverges from the typical presentations in the literature, which often involve trauma, localised resorption, or progressive deterioration. This highlights the necessity for further investigation and a more tailored differential diagnosis, potentially indicating a distinct subtype of idiopathic root resorption or root hypoplasia. Developmental anomalies affecting molar morphology, such as molar-incisor malformation [de Fátima Vieira Fernanda Gabriela et al., 2020], show potential parallels to bilateral root hypoplasia. Understanding the relationship between these anomalies and systemic diseases is critical for a comprehensive approach to diagnosing and managing such conditions. For instance, systemic diseases like aHUS can have a profound impact on dental development. Given that aHUS was diagnosed in the patient at the age of one year, coinciding with the cap stage of the first molar tooth formation [Thesleff & Tummers, 2008], it is plausible that tooth root hypoplasia may be a complication associated with the disease. The pathophysiology of aHUS involves uncontrolled activation of the alternative complement pathway, leading to endothelial cell injury in microvessels. This injury results in the formation of platelet-rich microthrombi, which narrow and occlude the microvasculature. Consequently, this process causes destruction of circulating erythrocytes, depletion of platelets, and ischemia of the affected tissues [Hofer et al., 2014]. Although the cap stage of permanent tooth formation primarily focuses on crown development rather than root formation, early features of root development are still present [Li, Parada & Chai, 2017]. Microischemia at this stage can impair the vascular supply to the developing tooth tissues, potentially leading to root hypoplasia due to inadequate delivery of nutrients and oxygen. Additionally, aHUS is a systemic condition marked by significant inflammation resulting from the release of the complement components C3a and C5a due to defects in complement regulation [Raina et al., 2019]. These small anaphylatoxins bind to specific receptors (C3aR and C5aR), activating immune cells and contributing to the pathogenesis of various inflammatory and autoimmune diseases [Peng et al., 2009]. Their actions promote systemic inflammation, which adversely affects multiple physiological processes, including dental health. This inflammation increases the risk of microbial translocation in the oral cavity, exacerbating local immune responses. Consequently, it can disrupt tissue development and delay the healing process [Sobieszczański, 2023]. Other systemic conditions, such as hyperparathyroidism, hypoparathyroidism, hypophosphatemia, hyperphosphatemia, hypocalcemia, Gaucher's disease, Paget's disease, Stevens–Johnson syndrome, Goltz syndrome, Papillon-Lefevre syndrome, anachoresis, and Turner syndrome, have also been identified as factors that may contribute to root resorption rather than dental hypoplasia [Nasehi, Nazhari & Mohtasam, 2015; Khojastepour, Bronoosh & Azar, 2010]. Moreover, tooth developmental anomalies have also been described in paediatric patients with severe systemic immune conditions, such as severe combined immunodeficiency and juvenile myelomonocytic leukemia, where systemic inflammation and therapy may disrupt odontogenesis [Cossellu et al., 2013]. Additionally, certain rare genetic disorders, such as Robinow syndrome, have been linked to craniofacial and dental anomalies, including root malformations, highlighting the broad phenotypic spectrum of syndromic dental defects [Eronat et al., 2009]. Hyperparathyroidism is a recognised consequence of aHUS, particularly following severe acute renal failure or prolonged chronic kidney disease [Loirat & Frémeaux-Bacchi, 2011]. Renal impairment leads to electrolyte imbalances, such as hyponatremia, hyperkalemia, hypocalcemia, and hyperphosphatemia, which can trigger secondary hyperparathyroidism. This condition causes the body to resorb mineralised bone, including dental structures, in an effort to restore equilibrium [Yuen, Ananthakrishnan & Campbell, 2016]. As a result, dental development may be affected, leading to altered eruption patterns, dental abnormalities, demineralisation, and potentially partial root resorption [Davis, 2015]. Chronic kidney disease can disrupt various stages of tooth development, causing anomalies in tooth number, shape, enamel quality, or root morphology [Velan & Sheller, 2021]. These disruptions occur because teeth are highly sensitive to the bioavailability of calcium and phosphate ions during mineralisation, and any disturbance in these levels can lead to developmental defects [Lucas & Roberts, 2005].

Additionally, fever during the first years of a child's life was significantly associated with a higher odds of MIH [Fatturi et al., 2019; Bagattoni et al., 2022], which can be attributed to the demineralised lesions of the teeth noticed in this case. Another potential factor that may have disrupted the normal root formation of the first molars in our patient is a 3-month course of inhaled corticosteroids administered to alleviate symptoms of obstructive bronchitis. Although reports on this issue are limited, some authors have noted the loss of tooth structure in young children undergoing similar treatment for asthma [Wogelius et al.,2010]. Corticosteroid therapy is known to suppress osteoblast formation and activity, leading to decreased bone formation. A comparable effect on ameloblasts may be plausible [Pawlicki et al.,1992] and could help explain our observations regarding root formation, particularly as the timing coincides with the early stages of root development in first molars. Considering all of the above, we are inclined to believe that the process in question is multifactorial, leading to root hypoplasia of the first permanent molars, rather than any of the previously mentioned potential differential diagnostic entities. Permanent first molars serve as long-term indicators of prenatal, perinatal, and postnatal health. In cases like the one presented in this study, genetic and molecular analyses could offer deeper insights into the underlying causes of the condition in future research. Additionally, maternal health during pregnancy could be examined as part of this analysis. Careful clinical examination of these efficient and cost-effective diagnostic tools, could aid in the prevention, early detection, and effective treatment of various dental pathologies.

Conclusion

This case underscores the critical importance of thorough dental and medical history assessment in diagnosing and managing dental anomalies. The findings highlight a possible association between systemic conditions, such as aHUS, and bilateral root absence. Clinicians should be aware of the potential dental implications of early childhood illnesses and consider these factors in their diagnostic and management approaches. Further investigation into the links between systemic diseases and dental anomalies is crucial for advancing dental care and outcomes for affected patients.

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Ethical approval

The Ethical Committee of the School of Dental Medicine gave permission for publishing.

Patient consent

Written patient consent was obtained from the parent/ legal guardian.

Authors' contribution

L. Šimunović and B. Špiljak equally contributed to the manuscript.

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