ORAL MANIFESTATION AND MANAGEMENT IN VITAMIN D-DEPENDENT RICKETS TYPE I: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Background: *Vitamin D-dependent rickets type I (VDDR-I) is an autosomal recessive disorder caused by the inactivating mutations in the 25-hydroxy vitaminD-1α-hydroxylase genes. Clinical features in individuals with VDDR-I include growth retardation, generalized weakness, and an increased risk of pathologic fractures. Oral manifestations and their treatment associated with this disorder are limited, with only three case reports and one case series reported to date regarding the oral findings in VDDR-I patients.*

Objectives: *To increase awareness of VDDR-I, highlighting its characteristic features, oral manifestations, and treatment protocols. Methodology: An 8-year-old male Saudi patient diagnosed with VDDR-I presented in the Pediatric Dentistry Clinic. The oral & radiological findings and their treatment protocol were documented.*

Results: *The dental examination of the patient revealed marked enamel hypoplasia in the permanent maxillary incisors, slightly enlarged pulp chambers in the molars, congenitally missing mandibular second premolar, and the presence of pulp stones in all the mandibular anterior teeth.*

Conclusion: *Mild to severe enamel hypoplasia can be managed with esthetic reconstruction using composite build-ups and ceramic veneers.*

Keywords: *Vitamin-D dependent rickets-I, oral manifestation, management, enamel hypoplasia.*

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INTRODUCTION

Hereditary rickets, a rare and complex genetic disorder, always interest researchers and physicians due to its complex interplay of genes and hormones responsible for growth and development. There are two different groups of vitamin-D-deficient hereditary rickets. The first group comprises of Vitamin D-Dependent Rickets Type I (VDDR-I) and Vitamin D-Dependent Rickets Type 2 (VDDR-II). The VDDR-I, also termed as Pseudo-Vitamin D Deficiency Rickets, is an autosomal disorder attributed to mutations within the CYP27B1 gene.[1] This specific gene encodes the enzyme accountable for converting vitamin D into its active state, Calcitriol. Individuals with VDDR-I cannot produce sufficient Calcitriol, leading to calcium and phosphorus deficiency in their bodies. VDDR-II, also termed as Hypocalcemia Vitamin D-Resistant Rickets is caused by mutations in the Vitamin D Receptor (VDR) gene.[2] Individuals with VDDR-II produce normal Cortisol, but their body tissues do not respond properly to vitamin D absorption.[3] The second group of genetic rickets, Hypophosphatemia Vitamin-D Resistant Rickets (HVDRR), are X-linked and are associated with mutations in the PHEX gene.[4]

VDDR-I is the rarest form of autosomal recessive disease, accounting for only 0.4 cases per 10,000 rickets patients.[5] This condition is predominant among Asian and Middle Eastern populations.[3] It typically manifests in the first few years of life and is generally less severe than VDDR-II. Ten variants of VDDR-I have been identified, with little or no clinical difference.[6] The autosomal rickets' most prominent clinical findings include short stature, delayed growth, muscular weakness, lethargy, and pathologic fractures.[5-7] Radiographic analysis reveals features such as osteopenia, bowed legs, and fractures. Blood and urine examination findings suggest hypophosphatemia, elevated alkaline phosphatase, and hypocalcemia. Characteristic laboratory features are hypocalcemia, increased serum concentrations of parathyroid hormone (PTH), and low or undetectable serum concentrations of $1,25(OH)_2D$ despite normal or increased concentrations of 25OHD.[5-7] The pathophysiology of VDDR-I is schematically explained in Figure 1.

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There is a scarcity of reported cases detailing the oral manifestations of Vitamin-D Dependent Rickets Type I (VDDR-I), and only few studies have discussed the oral findings of VDDR-II.[6] In contrast, the oral findings associated with HVDRR are well documented in the literature. Studies on HVDRR have shown anterior open bite with marked enamel and dentinal defects or thinning. The enamel in these patients is often hypoplastic and can appear clinically like pitted enamel hypoplasia.[5,8,9] Radiographic findings demonstrate large pulp chambers, short roots, a hypoplastic alveolar ridge, and poorly defined lamina dura.[10] Enlarged pulp horns and thin enamel increase the risk of pulpal exposure, leading to multiple abscesses and radiolucency. Histopathological examination of extracted teeth from HVDRR patients has revealed calcified interglobular dentinal masses and fissures extending from the enamel to the dentin-enamel junction.[7,11-13] It is worth noting that there may be similarities between the oral manifestations of VDDR-I and the other two hereditary vitamin D-Dependent Rickets variants due to their common genetic involvement in calcium metabolism.

This case report documents oral manifestations of VDDR-I in an 8-year-old male patient who presented at the Department of Pediatric Dentistry, King Saud University College of Dentistry, Riyadh. The patient underwent esthetic reconstruction in the Pediatric Dentistry Department. In addition to describing the case, this report presents differential diagnoses, review of similar past case reports, and explores various treatment modalities for the oral complications associated with VDDR-I.

Case Presentation

An 8-year-old male patient previously diagnosed with VDDR-I reported to the Pediatric Dentistry Clinics at King Saud University College of Dentistry seeking esthetic rehabilitation. The patient had short stature and bowed legs. The patient lacked confidence and rarely smiled due to the aesthetic issues. He was child from a consanguineous marriage and the oldest sibling. Past medical history revealed that at the age of 8 months, the patient was hospitalized for ten days due to recurrent infections. Blood examination during this period revealed that the calcium level was 0.5mg/dl; following that, the patient was given calcium supplements. However, no calcium level improvement was observed after taking calcium supplements for ten months. Further investigations, including blood, urine, and genetic testing, confirmed the diagnosis of VDDR-I. The tests were also conducted on the patient's parents and siblings; none reported any metabolic disorders. The patient was being treated with oral calcitriol supplement (0.5ml/day) and vitamin D (1000IU/day) Systemic examinations of the respiratory, nervous, and cardiovascular systems revealed no abnormalities.

Clinical examination revealed delayed growth milestones and teeth eruptions. The patient's vital signs were stable, with a height measuring 104 cm and a weight of 38 kg. Intraoral examination further reported a normal mouth opening with competent lips, mild enamel hypoplasia, and ankyloglossia. The patient had a caries-free mixed dentition, mild gingivitis, and plaque accumulation on anterior teeth. Additionally, the patient exhibited a severe gag reflex and hyper salivation. A well-demarcated enamel hypoplasia was observed on the occlusal and incisal surfaces of permanent incisors and permanent first molars (Figure 2). The patient also had dental crowding with a class I malocclusion. An Orthopantomograph (OPG) revealed hypoplastic enamel, wider pulp chambers in permanent molars, shortened roots, and developmental absence of lower left second premolar (Figure 3). The OPG also revealed maxillary anterior teeth crowding (Figure 4A and B) and pulp stones in the permanent anterior mandibular incisors (Figures 4C and 4D).

Treatment plan

Esthetic rehabilitation was planned for the patient and the treatment plan was explained to the child's mother, and an informed consent was obtained to proceed with the treatment, along with permission for disclosure and publishing rights of this case report. The treatment was divided into two phases. In the first phase, esthetic restoration of the maxillary incisors was performed using composite restorations. The teeth shade was selected using the VITA (VITA Zahnfabik, USA) shade guide. All necessary protocols were followed, and the treatment was carried out under rubber dam isolation to prevent contamination or mishaps during the procedure. After isolating the teeth, plaque, and debris were removed, and minimal preparation was

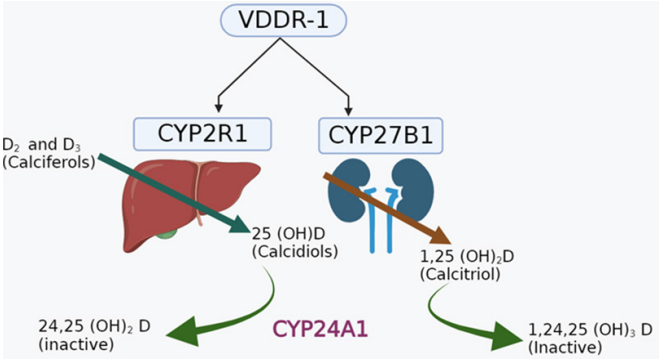
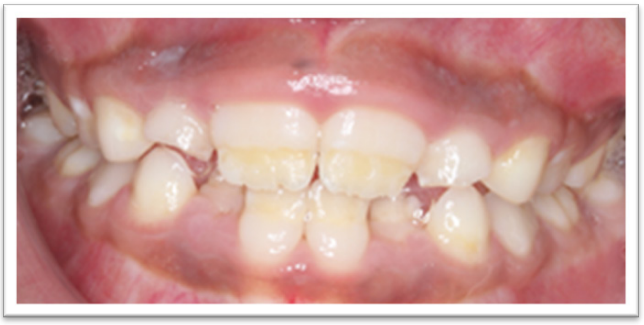


Fig 1: Pathogenesis of VDDR-I



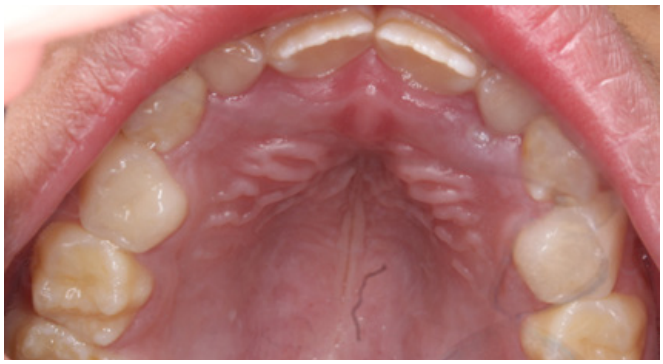


Fig 2: Mild Enamel Hypoplasia on the incisor and molar surfaces

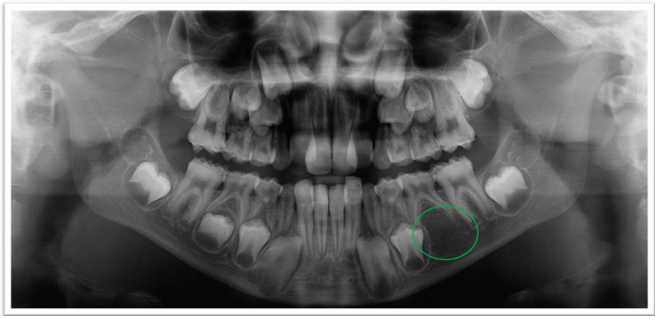


Fig 3: Orthopantomograph of the VDDR-I patient showing missing left mandibular second premolar.

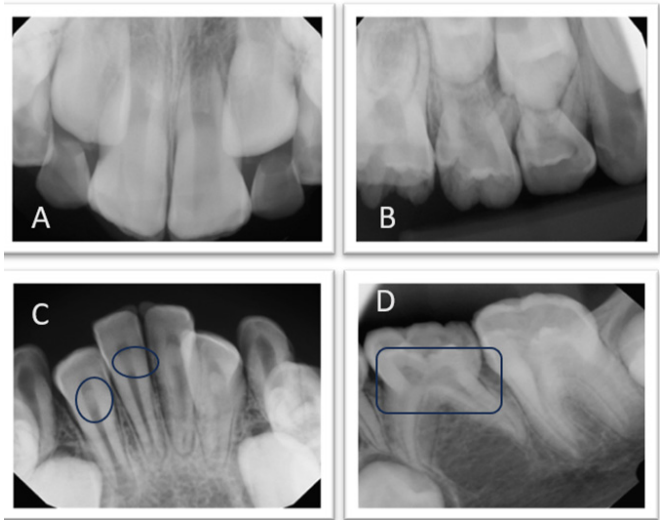


Fig 4: Radiographic findings: A and B: Maxillary anterior and posterior teeth showing dental crowding; C: Mandibular anterior teeth showing pulp stones; D: Posterior teeth with slightly enlarged pulp chamber and short roots.

performed using a diamond bur. The teeth were then etched for 15 seconds and rinsed. A bonding agent (3M ESPE Adper) was applied, followed by curing the anterior teeth using a light-emitting diode (LED) unit for 20 seconds. Shade A3 (Vita Classical, Dentaltix, Germany) was used for the maxillary incisors. Finally, polishing was completed using a Soflex 3M polishing

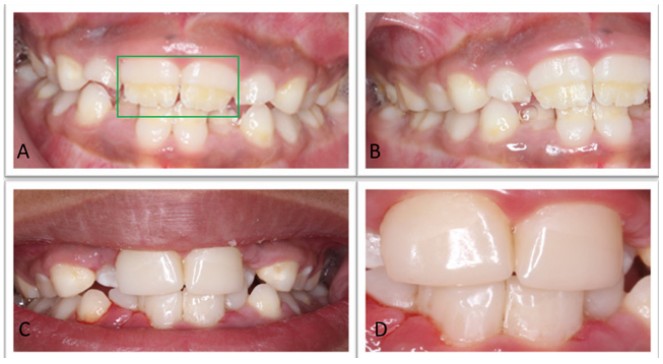


Fig 5: A and B: Presenting mild enamel hypoplasia; C and D: Post-treatment pictures.

kit (Figure 4 C and D).

The patient was scheduled for a follow-up appointment one week after the initial treatment to proceed with the second phase of treatment. During this visit, the patient expressed satisfaction with the esthetic modifications. Figure 5A and 5 B represent pre- and six-months post-operative treatment photographs. In the second phase of treatment, coronal coverage was provided for the maxillary first permanent molars on both sides to prevent enamel breakdown. Tropical application of fluoride varnish (Duraphat, Colgate-Palmolive, GmbH, Hamburg, Germany) was administered to reduce the risk of caries and pulp pathosis. The patient also received instructions on maintaining oral hygiene. The patient was then referred for orthodontic treatment. When the patient returned for a follow-up visit after six months (Figure 5C and D), it was observed that the patient had maintained good oral hygiene.

Literature review

The literature search was performed on the PubMed database through using MeSH terms "Vitamin-D Dependent Rickets Type 1" OR "Vitamin-D Dependent Rickets" OR "VDDR I" AND "Oral or Dental Manifestations" AND "Treatment" AND "Case series" AND "case report". Reference chasing and manual searches were also performed to include all the relevant articles. The initial search retrieved 76 articles. All the included case reports and case series were in the indexed peer-reviewed journal and in English language. After screening the titles and abstracts, 10 case reports were extracted for Vitamin-D-Dependent Type-I and Type-II Rickets. One article was found during reference chasing. Finally, three case reports and one case series reporting oral/dental manifestations and their treatment for VDDR-I were included in this review. Table 1 illustrates the dental findings of the cases reported.

DISCUSSION

This case report presents the oral manifestations and treatment modalities in a patient with genetically confirmed VDDR-I, a rare autosomal rickets caused by genetic variations on chromosome #12. The hydrolase

gene responsible for Vitamin-D activation is located on the long arm of chromosome #12, specifically in region 1, band 12q12-14.[4,5] In VDDR-I, disruptions in this chain lead to a deficiency in the production of $1,25(OH)_2D$. While there are numerous cases reported regarding the general symptoms of this autosomal disease, only three case reports and one case series have been found in the literature that include oral manifestations of VDDR-I patients.

The literature review shows that the dental phenotype of VDDR-I patient is rarely mentioned than the other types of rickets.[11,12,14] Research on VDDR-I has primarily focused on random clinical cases and various phenotypes of 1-alpha-hydroxylase enzymes.[2,15] Consequently, there remains a scarcity of oral/dental manifestations and their treatment modalities in VDDR-I patients. In the present case, the diagnosis of rickets was confirmed by classical signs of skeletal growth (bowed legs), including low serum calcium and $1,25(OH)_2D$ levels, along with elevated PTH. The patient presented with dental issues, including enamel hypoplasia, delayed tooth eruption, multiple pulp stones, and congenitally missing teeth. However, after six months of dental treatment, patients' oral health improved. Early oral health interventions, such as restoration of carious teeth, stainless steel metal crowns to protect first molars, and administering regular fluoride treatment for enamel remineralization and caries prevention, effectively preserve oral health in VDDR-I patients.[12,16]

In cases reported in the literature and in this case, enamel hypoplasia of varying degree was observed, affecting incisors and molars, ranging from mild to severe. In contrast, premolars and second permanent molars remained unaffected in all cases. These variations in enamel condition can be attributed to the distinct developmental stages of teeth from gestational age to maturity, each requiring calcium and phosphate for enamel formation. In VDDR patients, the deficiency of these essential minerals disrupts crown formation and leads to enamel hypoplasia.[6,13,17] The development of permanent incisors, canines, and first molars commences during the first year of growth, with crown formation completed by ages 5 to 7 years of age.[1,10] Literature suggests that VDDR-I is typically diagnosed within the first few years of life; at this point, medication to improve their mineral levels is initiated.[9,18] As a result, varying degrees of discoloration are predominantly observed in the incisors and on the occlusal surface of the first molar. Enamel hypoplasia is a typical dental condition of VDDR-1 and is not reported in other vitamin-D-associated autosomal disorders. However, enamel hypoplasia is also reported in nutritional rickets, but the significance is different from VDDR-I. Overall destruction of complete enamel surface is reported in nutritional rickets,[11] while in VDDR-I patients, only the occlusal and incisal surface of permanent teeth is affected.

Dental tissue mineralization, unlike other bones, does not undergo remodeling.[7,8] As a result, the distribution of enamel hypoplasia is a permanent marker for diagnosing low levels of calcium and phosphate, which are crucial for dental tissue mineralization.[6,17] In nutritional rickets and VDDR-I cases, low levels of calcitriol and phosphorus are commonly recorded, leading to varying degrees of enamel hypoplasia. However, in other forms of autosomal rickets, patients may experience hypophosphatemia, but their calcium levels typically remain within normal limits, and enamel hypoplasia is not as prevalent.[7,17] These findings suggest that enamel hypoplasia primarily occurs due to hypocalcemia and hypophosphatemia. Additionally, the exact causal factor for enamel hypoplasia in VDDR-I remains unknown. Further research is needed to establish a conclusive association of the minerals mentioned above with enamel hypoplasia.

Prominent radiographic findings associated with VDDR-I patients include thinning of dental enamel and enlarged pulp chambers, leading to taurodontism.[14,16,19,20] The characteristic features of this condition are apical displacement of the pulp chamber and the absence of constriction at the cementoenamel junction, which can result in pulpitis and periapical abscess.[1,18] In all the reported cases in the literature review, the periapical abscess was frequently observed, particularly in the mandibular first permanent molar.[14,16,19,20] However, in this case, while enlarged pulp chambers and shortened roots were observed, none of the teeth exhibited pulpitis. Moreover, the patient had multiple pulp stones and congenitally missing tooth in this case, which was not reported in other cases. These findings could be attributed to differences in the genetic types affected among the various VDDR-I cases.

Previous studies have suggested that early intervention of rickets can prevent dental anomalies.[4,5,18,19,21] For instance, patients with nutritional rickets on calcium supplement have less chance of developing caries and enamel hypoplasia. Similarly, in patients with HVDRR, dental defects can be prevented by adequate vitamin and calcium supplements.[4] In the present case, the patient was on oral supplements since the age of 2 years. At the age of eight, when he visited the dental hospital, serum calcium was at normal levels, and the remission of rickets was measured. However, newly erupted permanent incisors appeared brown to yellow, exhibiting enamel hypoplasia. These findings suggest that calcium and vitamin supplements could not reduce the chances of enamel defects in VDDR-I patients. Hence, the esthetic reconstruction, such as the composite buildup and ceramic veneers, is the line of treatment.

There has been significantly less research on oral manifestations and defects associated with autosomal recessive rickets than other clinical conditions. Moreover, no dental records have been reported on VDDR-I cases in Saudi Arabia. This could be due to the early interventions of developmental anomalies are performed in general pediatric and endocrinal clinics, and not in dental clinics. Thus, oral defects related to

TABLE 1: CASE REPORTS AND CASE SERIES ON ORAL MANIFESTATIONS OF VDDR-I

Sr. No	Study	Cases	Age of diagnosis	Serum levels	Clinical findings	Radiographic findings	Dental Treatment
1.	Zambrano et al. 2003,14 Venezuela	1	37 months	Alkaline phosphatase: 1035 U/L. Serum parathormone: 72 pg/ml. Vitamin-D (OH): Normal. Vitamin-D 1,2, 5 (OH): 21 pg/ml.	At the age of 10 years, hypoplastic enamel of all permanent teeth (occlusal and interproximal surfaces). Class II dental malocclusion, generalized marginal gingivitis.	Quadrangular pulp chambers with short roots.	Cosmetic reconstruction with composite restorations. Fluoride application.Metal crown placement on a mandibular first molars.Extraction of the first premolars for orthodontic treatment.
2.	Ito et al. 2014,20 Australia	1	6 months	Parathormone: high 1,25 (OH)2 low	At the age of 3 years, multiple caries and hypersalivation	Large pulp chambers Short roots	Caries, excavation, and restoration were done. Fluoride application was done.
3.	Gjørup et al. 2016,19 Denmark	10 cases (Eight females two males)	Range: 7 months to 2.4 years	Findings were not provided for all the patients.	Dental agenesis in one patient. Taurodontism was seen in 4 patients. Hypoplastic enamel was seen in all the patients	In five patients, endodontic fillings were recorded. In two-child patients' caries, filling was present without pulp treatment. In three patients' periapical radiolucency was seen. Multiple abscesses were reported in two patients.All the included patients had an enlarged pulp chamber with short roots.	Maxillary first molar extractions were done in four patients. A stainless-steel crown was given to one patient. Enamel hypoplasia was common in all ten patients, and composite buildup was provided to all of them. All the patients had undergone extraction due to orthodontic treatment. Well-maintained and caries-free teeth were present in all three adult patients.
4.	Lui et al. 2017,16, China	1 case	3 years	1,25 (OH)2D- 5.82 pg/mL Alkaline phosphatase-800 U/L Parathyroid hormone-388.3 pg/mL Calcium-1.29 mmol/L Phosphorus-1.49 mmol/L	At the age of 10, Enamel hypoplasia with all the permanent teeth. Caries with tooth #15,#16, #26, #27,#34,#46 Dental Malocclusion class II Amelogenesis imperfecta	Delayed development of permanent tooth germs. Thin dentin and large pulp chamber	Composite buildup was provided. A metal crown was given. Caries removal and restorations were done.

these autosomal disorders remained ignored. Moreover, our findings show that early interventions provided to patients with VDDR-I can reduce the chances of further health related issues, suggesting collaborative therapeutic measures involving pediatric dentists, psychologists, and general physicians.

CONCLUSION

This is the first case report of VDDR-I in Saudi Arabia focusing on oral manifestations and their treatment modalities. Early dental treatments and long-term follow-up improves esthetics and general dental health in VDDR-I patients. Mild to severe enamel hypoplasia can be managed with esthetic reconstruction using composite build-ups and ceramic veneers.

Clinical Significance

Patients with VDDR-I need to be managed through multi-disciplinary approach including a team of healthcare professionals, including dentists to manage their dental and overall health. The management often includes vitamin D and calcium supplementation, dental treatments to address enamel hypoplasia, caries, and orthodontic intervention for malocclusion.

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