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CASE REPORT

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Vitamin D deficiency and chronological hypoplasia with hypomineralisation: a case report

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Abstract

Patients being reported for vitamin D deficiency (VDD) are increasing, particularly among the children and adolescents. This study aims to manifest the clinical and dental evaluations of a child with VDD, referred to the dental office. A 10-year-old British Asian boy was referred to the paediatric specialist dentistry clinic by the general dentist for dental management. The medical history depicted that the patient was diagnosed with VDD, secondary hyperparathyroidism and delayed growth. Moreover, his mother had the VDD during pregnancy. The patient was breast fed and had rickets in infancy. He was prescribed vitamin D supplements at the age of 16 months. He had received multiple dental treatments under local anaesthesia but with limited cooperation. Clinical examination revealed that the patient had chronological enamel hypoplasia shown as bands at the occlusal third on specific teeth. Suboptimal hygiene with general plaque induced gingivitis, dental caries in permanent and primary teeth, and delayed the teeth eruption. Preventions included appropriate oral hygiene and dietary advice, fluoride varnish application and fissure sealant placement. The treatments included anterior direct composite restoration, posterior composite restoration, stainless steel crowns and extractions. Thorough medical history is essential to understand the underlying causes of dental defects. Early dental intervention can restore the patient appearance and function and prevent further dental damage.

Keywords

Vitamin D deficiency; Hypoplasia; Oral health

1. Introduction

Vitamin D is produced by exposing the skin to ultra-violet B (UVB) radiations from the sun. The sun exposure through a window prevents the stimulation for vitamin D synthesis as the glass absorbs radiations. Secondary sources of vitamin D are the supplements, and foods such as oily fish, beef liver, cod liver oil, shiitake mushrooms, egg yolk and cheese [1].

Vitamin D along with parathyroid hormone (PTH), fibroblast growth factor 23 (FGF23), and calcitonin have role in the children growth. Adequate levels of vitamin D are essential for the musculoskeletal and dental development. Other body systems including muscular, immune, neurologic, respiratory and cardiovascular also require vitamin D [2, 3].

Lower vitamin D levels result in less calcium absorption through intestine and subsequently cause low blood calcium levels. This stimulates PTH secretion from parathyroid gland. Calcium is released from bones and may lead to bone alterations including rickets, osteoporosis or osteomalecia [4]. Moreover, VDD can develop autoimmune conditions such as type I diabetes, inflammatory bowel disease and multiple sclerosis [5]. It may also cause respiratory diseases including respiratory tract infections, tuberculosis and asthma [4]. The studies have associated VDD with hypertension, cancer, musculoskeletal pain, migraine and neuropsychiatric diseases such as schizophrenia, dementia and depression in adults [6]. A recent review reports negative impacts of VDD on soft and hard oral tissues [7]. It increases hypoplasia, hypomineralisation, caries, gingivitis and chronic periodontitis [8].

The dentists must thus be aware of VDD complications to better manage the dental patients. This report provides the details of general and dental manifestations, and the management of a child having VDD, referred to dental clinic.

2. Case description

A 10-year-old British Asian boy was referred to a specialist paediatric dentistry clinic by the dentist for suitable dental management. The patient mainly complained that: “I do not like the look of my front teeth”. Mother was also not happy with the appearance child’s teeth. Family history indicated no such dental condition in the family. The mother was diagnosed with low levels of vitamin D during pregnancy and breast feeding the patient.

The patient was diagnosed for VDD along with secondary hyperparathyroidism and delayed growth. He had delay

and difficulty in walking because of rickets. He was treated through vitamin D supplements from 16 months of age till now.

There were multiple previous dental treatments under local anaesthetic and preventive care at general dental practice but with limited cooperation. Oral hygiene habits included teeth brushing twice a day with 1450 ppm fluoride toothpaste. He was treated with fluoride varnish every 6 months. His diet was cariogenic and included fresh fruit, flavoured yoghurt, sweets and fruit juice.

He lived with parents, two older siblings and grandmother. He was attending primary school and became upset when children teased him about the teeth appearance.

Clinical extra-oral examination indicated symmetrical and convex facial profile. Intra-oral assessment depicted mixed dentition with suboptimal oral hygiene and plaque induced gingivitis. Fig. 1 shows the anterior teeth appearance. There was generalised physiological gingival pigmentation, and hypoplasia affecting the maxillary and mandibular primary and permanent teeth. The hypoplasia appeared as a chronological band at the occlusal third of 16, 55, 11, 21, 26, 56, 36, 75, 32, 31, 41, 42, 85 and 46.

He had class I permanent molar relationships on both sides, and mild crowding of upper and lower anterior segments with increased overbite. Dental caries was seen on 54, 63, 26, 36 and 74. The defective amalgam restorations were tracked on 55, 65, 75 and 84.

An orthopantomogram, and right and left bitewing radiographs were taken for caries diagnosis, pathology and assessing the developing dentition (Fig. 2). All permanent teeth were present except the 18. The dental age of 8 years reflected some dental delay. The radiographs exhibited irregular occlusal enamel and radiolucency on the following teeth; 54: occlusal and distal deep into dentine, 26: occlusal into outer 1/3 of dentine, and 74: extensive occlusal and distal with furcal involvement. Moreover, there were radiopaque restorations on 55, 65, 75 and 84. The lamina dura of the teeth appeared normal. Occlusal opacities with post-eruptive breakdown were noted on 16, 26, 36 and 46.

The differential diagnosis revealed chronological enamel hypoplasia, enamel hypomineralisation and amelogenesis imperfecta (hypoplastic pitted). The definitive diagnosis indicated chronological enamel hypoplasia and hypomineralisation with VDD.

Dental treatment was phased as the initial, intermediate and long-term. The initial phase included prevention, *i.e.*, the oral hygiene and dietary advice, prophylaxis and topical fluoride varnishing. The intermediate phase involved continued preventive care with reinforced oral hygiene, diet, fluoride varnish and fissure sealants (3M Espe Clinpro Sealant) on 16 and 46. Occlusal composite restorations (3M Espe Filtek Z350 XT) were made on 26 and 36, stainless-steel crowns (3M^{TM} Stainless Steel) on 55, 65, 75 and 85, and extractions for 54, 64, 74 and 84. Direct composite restorations were placed on 11, 21 and 63 (Fig. 3). The long-term treatment included the reviewing of patient at three months, six months, and a year after the completion of dental treatment to monitor the developing dentition and provide feasible preventive care.

3. Discussion

Previous studies had shown a link between VDD and multiple orofacial factors. There was an increase of VDD in children and adolescents of UK because of less sunlight exposure particularly in the winter [1]. The infants and adolescents are the vulnerable groups because of fast bone development after birth and during puberty [9]. Breast milk contained relatively low vitamin D levels compared to cow milk [9, 10]. Exclusively breastfed children, particularly those born to vitamin D deficient mothers were vulnerable to develop rickets. Preterm new-borns were at risk of vitamin D insufficiency because of lacking transplacental vitamin D transfer during third trimester and low sun exposure in postpartum hospitals. Moreover, the Asian people from Indian subcontinent required three times the amount of sun as Caucasians, while Africans might need six to ten times [11]. These findings were consistent with the present report where patient was a child living in UK, British Asian in origin, mother had VDD during pregnancy and the patient was breast fed.

It seemed from the clinical photos and radiographs that hypoplasia was mostly affecting the occlusal third of permanent first molars, central incisors, lower lateral incisors and primary second molars. Furthermore, hypomineralisation was observed on all permanent first molars. This fitted with the clinical history of being vitamin D deficient, particularly when those teeth were formed. The earlier and later forming teeth might been saved by the vitamin D supplementation.

The major VDD complications in this patient were rickets, secondary hyperparathyroidism, enamel hypoplasia, hypomineralisation and dental caries. The rates of dietary vitamin D related rickets in UK had increased from 0.34 cases per 100,000 children between 1991–1996 to 3.16 cases between 2007–2011 [12]. A recent case report from India for 8 years old female patient with severe VDD and rickets manifested the following clinical and dental details; prominent forehead, depressed nasal bridge, bowing of legs and hands, widening of ankles and wrists, prominent clavicles, alopecia, sparse eyelash and eyebrows, premature primary teeth loss, delayed permanent teeth eruption and large pulp chambers with short roots [13]. However, none of the previous features were seen in this case as the rickets was treated in infancy.

Severe VDD ($<$10 ng/mL) resulted in the hypocalcemia and hypophosphatemia leading to secondary hyperparathyroidism. PTH enhanced the calcium absorption from intestine, bone turnover and produced 1α,25-dihydroxyvitamin in kidneys. This led to the high calcium levels in serum and low phosphate with disturbance in bone mineralisation which might develop rickets, osteomalacia, renal osteodystrophy and calcifications in extraosseous areas including coronary calcifications. This could have cardiovascular consequences and thus raise the mortality risk [14]. However, the condition could be treated by replenishing vitamin D which returned the PTH levels to normal [15]. Dental manifestations of hyperparathyroidism were the brown tumors, loss of jaw bones density, delayed dental growth and development, tounge or lip paresthesia and changes in facial muscles [16]. In current case, only the delayed dental growth and development was evident.

Skeletal and dental mineralisation processes occurred con-

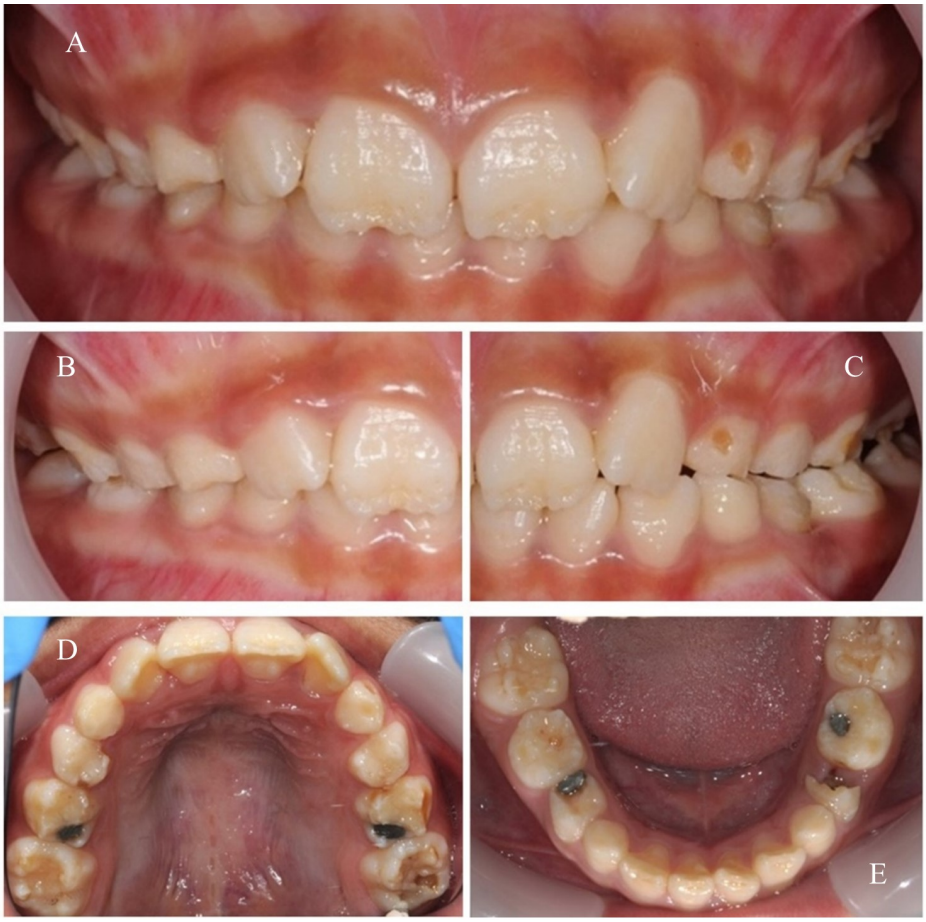

FIGURE 1. Pretreatment intra-oral photographs. (A) frontal view, (B) lateral right view, (C) lateral left view, (D) upper occlusal view, and (E) lower occlusal view.

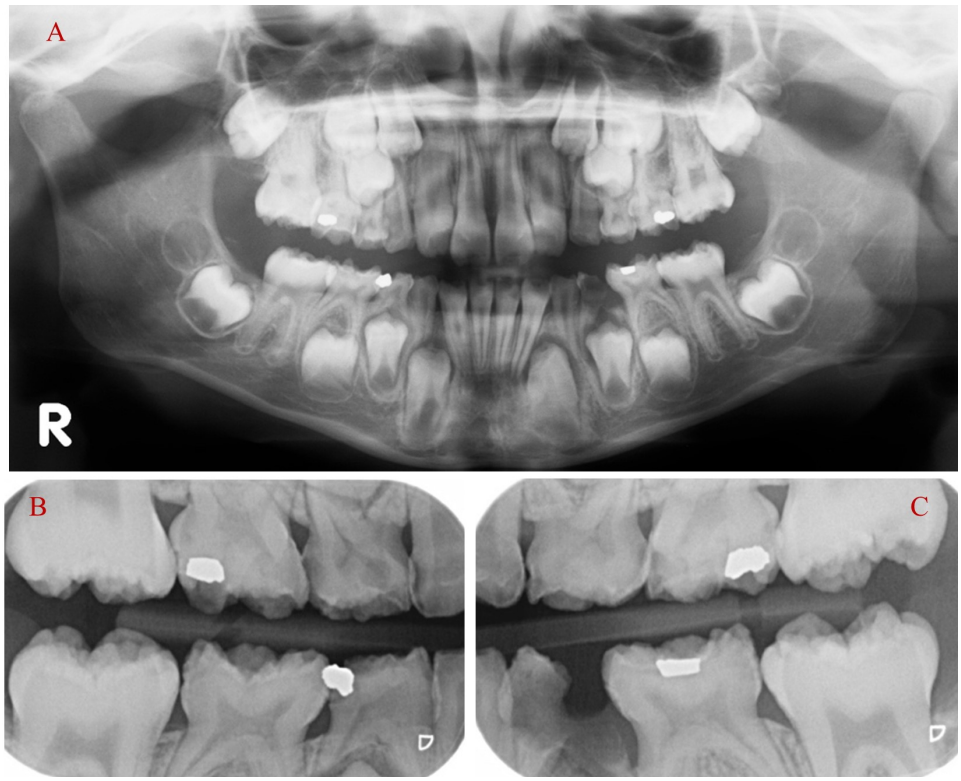

FIGURE 2. Pre-treatment intra-oral radiographs. (A) orthopantomogram radiograph, (B) right bitewing radiograph, and (C) left bitewing radiograph.

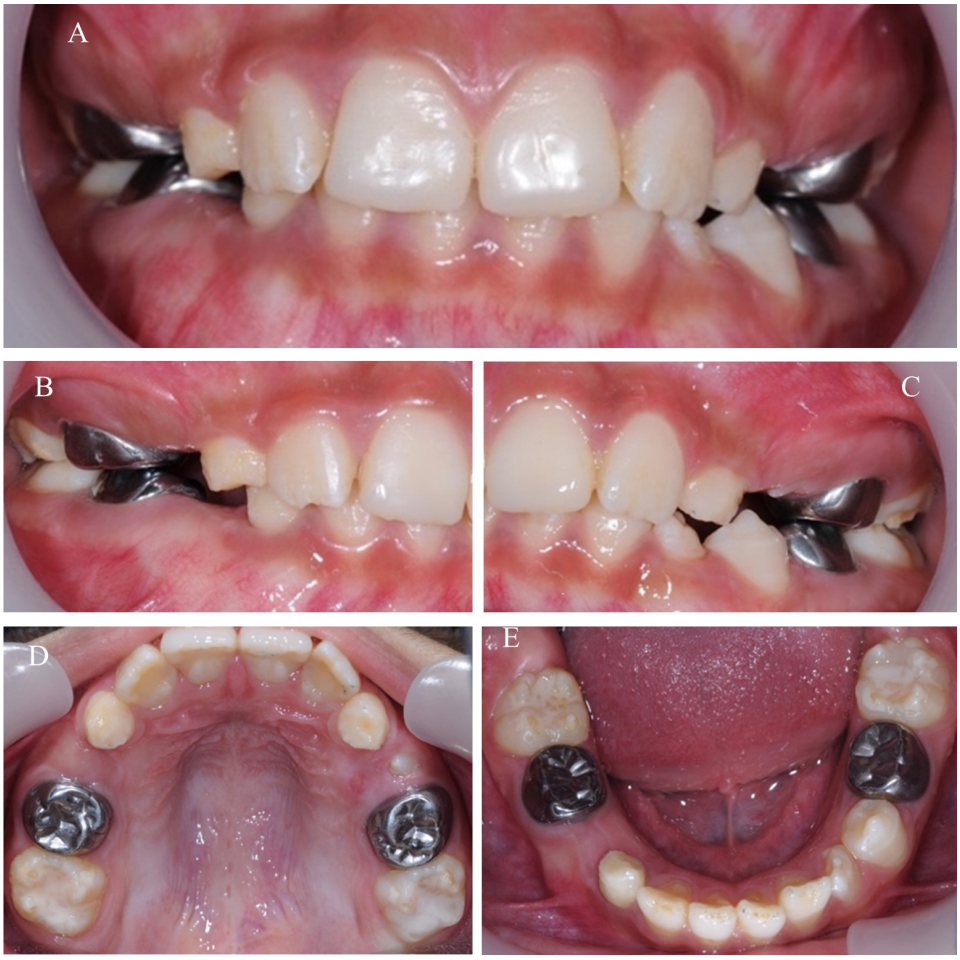

FIGURE 3. Post-treatment intra-oral photographs. (A) frontal view, (B) lateral right view, (C) lateral left view, (D) upper occlusal view, and (E) lower occlusal view.

currently. The interrupted mineral metabolism might lead to teeth and/or bone deformities. No signs of bone abnormalities were seen in this case. Teeth with enamel hypomineralisation and hypoplasia were prone to sensitivity, fracture and dental caries [4]. A large (n = 223) multi-center and cross-sectional study found that the vitamin D insufficiency was associated with caries (adjusted odds ratio (OR) 2.89, 95% Confidence intervals (CI) 1.43–5.86) and gingival bleeding (adjusted OR 2.36, 95% CI 1.10–5.01) [17]. Another case report depicted that a VDD patients had severe periodontitis [18]. In present case, the suboptimal oral hygiene led to generalised plaque induced gingivitis.

Dental caries was an infectious disease linked to the factors such as poor oral hygiene, cariogenic diet and cariogenic bacteria. The irregular dental surfaces in hypoplastic teeth would act as plaque retentive areas where brush cleaning might be difficult and thus increasing the caries risk [8]. This was evident in the current case.

An appropriate dental management including the prevention and treatment was essential to prevent further teeth damage and restore shape, aesthetics and function. The option for anterior teeth treatment in children was the composite restoration. For posterior teeth, the composite restoration or full coverage restorations such as stainless-steel crowns in primary teeth ensured tight marginal seal. If primary teeth had poor prognosis, the extraction might be indicated with or without space maintenance. More options were available to manage the severe breakdown in permanent dentition [8].

The case reports might have limitations such as inability to generate hypothesis or generalise the findings, and not revealing the cause-and-effect relationship. Moreover, there could be higher bias especially during the data collection or interpterion. However, the case reports could be valuable for an in-depth understanding of rare or new conditions. They were also inexpensive and might guide in developing further studies [19].

In current report, the initial dental management by general dentist was to place amalgam restorations on primary molars which were less successful than the composite or stainless-steel crowns. The patient was self-conscious as his permanent anterior teeth were a concern regarding appearance. The dentist rightly referred him for the specialised care and teeth management.

4. Conclusions

Vitamin D deficiency is not an uncommon condition among children and may lead to medical and dental complications. However, early detection and appropriate management can improve the long-term outcomes. This is vital in dental care of patients with poor aesthetics and dental caries.

AVAILABILITY OF DATA AND MATERIALS

The generated and analyzed data during the present report are not publicity available as the consent and the assent that were obtained from the parent of the patient and the patient were for the author only.

AUTHOR CONTRIBUTIONS

AA—contributed to the conception and design of the study. She delivered the dental care for the patient. She drafted the text and figures of this report. In addition, she critically revised and submitted the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

A written informed consent from the mother of the patient and a written assent from the patient were obtained to publish this report.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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