



CASE REPORT

GRADE II DENTAL MANIFESTATIONS IN RICKETS: A CASE REPORT

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Abstract

Rickets is a disease that occurs during childhood due to failure of growing bone to mineralize. Proper bone formation requires vitamin D and any disturbance in its production, absorption or metabolism is paramount in the development of rickets. Vitamin D deficiency has emerged as a significant public health problem throughout the world, even in the Indian context in spite of wide availability of sunlight. One of the most important oral alterations elicited is the recurrent formation of spontaneous dental abscess in the absence of caries or trauma, as dentition is highly susceptible due to structural defects in enamel or dentin, so bacteria can invade easily from oral cavity to dental pulp. The aim of present article is to report a case of rickets in a 14 year old girl, describing dental findings and to raise awareness on characteristics of this disorder.

Key words: Calcium, Dental abscess, Rickets, Vitamin D.

INTRODUCTION

Vitamin D not only regulate calcium and bone metabolism but also reduce the risk of malignancies, auto immune, cardiovascular and infectious diseases.^[1] Its deficiency causes mineralization defects in teeth, leading to poorly mineralized and hypoplastic dentin consisting of calcospherites rather than properly mineralized dentin. This mineralization defect together with hypophosphatemia may affect dental development and maturity.^[2] Rickets is attributed to enlarged coronal pulp spaces, high pulp horns extending beyond dentino enamel junction, grossly defective dentine in the form of dentinal clefts and microcleavage allowing ingress of micro-organisms to pulp, leading to formation of characteristic spontaneous periapical abscess without dental caries or trauma. The enamel is relatively thin, hypocalcified or hypoplastic.^[3] Early diagnosis is essential to minimize morbidity.^[4]



CASE REPORT

A 14-year-old female (Figure 1) presented with yellowish dark brown deposits on all teeth since four years. Medical history revealed deformity with both legs (Bow legs), enlargement of proximal ends of radius and ulna at wrist joint, knock-knee (genu valgum) at the age of 4 years and was medically diagnosed as rickets. Since 7 years of age she was under the treatment for the same. There was no history of any change in chest shape or renal manifestations, costal and spinal abnormalities but recurrent respiratory infections was present. Family history was non-contributory. Patient was reported with deficient secondary sexual characters development. Patient presented with short stature, appeared pale, lean with 28 kg weight, 129.3 cm height and reported anorexia. Later on she was advised to consume a balanced diet by a dietician. Currently, patient advised to take following medications as mentioned in reports: Calcitriol Sachet (vitamin D), Tab Calcinex (calcium), Syrup I-up (iron), Zincovit (multivitamin) supplements. When patient visited us she was completely treated for all her systemic manifestations and was under maintenance therapy. Her reports revealed that at the age of 7 years her vit D levels were 10ng/ml which improved to 20ng/ml, calcium levels were 6.5 mg/dl which improved to 11.2 mg/dl post treatment. Alkaline phosphatase levels remained elevated to 231.2 IU/L even post treatment and blood urea and creatinine levels were within normal range. Patient did not provide us with her pre and post treatment chest and bone radiographs. Intraoral examination revealed generalized grade II gingival enlargement with inflamed papillary and marginal gingivae which was erythematous, edematous and soggy in consistency, with class II gingival recession, spontaneous bleeding on probing with grade III stains and calculus (Figure 2). Hard tissue examination revealed missing 17,27,35,37,45,47 with over retained 75. Generalized chalky white opaque patches were observed on all surfaces of all teeth with generalized yellowish staining with pitting deformity (Figure 3), however no penetration of probe in enamel was noticed. Grade I mobility was elicited with 31,32,33,41,42,43 with Angle's class I malocclusion. We were unable to perform periapical radiography due to high non-compliance of the patient. Orthopantomogram revealed generalized reduced radiodensity of enamel and dentin with enlarged pulp chambers, irregular periapical radiolucencies involving most of the teeth suggested of multiple dental abscesses in absence of caries (Figure 4,5), congenitally missing teeth buds with 17,35,37,47, unerupted permanent teeth follicles with 27,38,45,48, overretained 75 at occulsal level (Figure 5,6). On the basis of patient's history, investigation reports, current clinical and radiographic findings, a diagnosis of Vit D deficiency Rickets was made. A differential diagnosis of Amelogenesis imperfecta and Enamel hypoplasia secondary to calcium deficiency were made. Patient was referred to department of Pedodontics and Endocrinologist for further management.

DISCUSSION

The prevalence of vitamin D deficiency is 50-90% in the Indian subcontinent,^[1] due to dietary or sun light exposure deficiency. Signs and symptoms of rickets often appear at about 8-10 months of age and manifests as lateral bowing of legs, frontal bossing, enlargement of costochondral junctions,^[5] rachitic rosary, genu valgum with enlargements of proximity of wrist joint^[6]. The diagnosis of rickets is made upon complete physical and dental examination, confirmed by laboratory examination including serum calcium, phosphorus, alkaline



phosphatase, blood urea and creatinine and 25 hydroxy vitamin D levels (Table 1) and with anteroposterior radiographs of rapidly growing skeletal areas.^[4]

Harris and Sullivan (1960) reported first dental characteristics of rickets. It is responsible for defective teeth mineralization leading to dysplastic dentin with a non-calcified matrix with tubular defects.^[7] As enamel and dentin formation occur between 4 months in utero and 11 months of age, defects in primary dentition can usually not be prevented. However permanent teeth form after birth and their development could possibly be improved by medication started soon after birth. Abnormal dental development and dentin formation may persist despite therapy. The sequence of abscesses formation usually appear to follow eruption sequence.^[4,8] Melissa A et al conducted a study in 14 patients and found that open bite (Angle's class II malocclusion, 85.7%) and enamel hypoplasia (14.3%) were most common dental findings in rickets patients.^[9]

Seow and Latham (1986) reported spectrum of dental manifestations ranking from mild to severe, based on number of abscesses and radiographic appearance of teeth.

Grade I: presents minimum or lack of dental manifestation.

Grade II: moderate pulp enlargement with few dental abscesses.

Grade III: extremely large pulp chambers and multiple dental abscesses.^[7]

Thus, the case we have presented here includes almost all the dental manifestations of rickets, suggesting Grade II variety.

Management includes providing dietary and commercial vitamin D and calcium supplements.^[1]

The main management strategy for the dental manifestations is the prevention of dental abscesses through prophylactic pulp therapy, pulpotomy, pulpectomy, coverage of molar teeth with stainless steel and restorations with composite and resin cements,^[10] topical fluoride applications, pit and fissure sealants, maintenance of good oral hygiene, extraction of teeth with periradicular abscesses and eventual restoration with implants.^[3]

CONCLUSION

Rickets can adversely affect dental development and delay in commencement of medical treatment may lead to permanent deficit in dental development. Effective strategies for prevention of dental abscesses should be employed to suit each individual patient's needs. The dentist as well as the pediatrician should be made aware of its features so that early intervention can prevent subsequent serious and more invasive dental procedures.

Table 1 Vitamin D Status in Relation to 25 (OH) D Levels^[1]

Vitamin D status	Levels
US IOM classification	
Severe deficiency	<5 ng/mL
Deficiency	<15 ng/mL
Sufficiency	>20 ng/mL
Risk of toxicity	>50 ng/mL
US Endocrine Society classification	
Deficiency	<20 ng/mL
Insufficiency	21-29 ng/mL
Sufficiency	>30 ng/mL
Toxicity	>150 ng/mL



Figure 1- 14 years old female patient



Figure 2- Gingival enlargement and inflammation with calculus deposits



Figure 3- Severe Enamel Hypoplasia with pitting, Intrinsic and Extrinsic stains



Figure 4 -Multiple periapical abscess without caries



Figure 5- Overretained 75



Figure 6- Unerupted teeth buds with 27,38,45,48, congenitally missing teeth buds with 17,35,37,47

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