ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 13, Issue, 01, pp. 15674-15677, January, 2021

DOI: https://doi.org/10.24941/ijcr.40626.01.2021

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE REPORT

CONGENITAL RICKETS AND DENTAL CARIES- A CASE REPORT

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ARTICLE INFO

Article History:
Received 06th October, 2020
Received in revised form
08th November, 2020
Accepted 11th December, 2020
Published online 30th January, 2021

Key Words:

Rampant caries, Rickets.

ABSTRACT

Rickets is a disorder caused by a lack of Vitamin D, calcium or phosphate. 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. Dental manifestation of rickets includes enamel hypoplasia and delayed tooth eruption. Careful and thorough management of this disorder can be done by conservative dental treatments such as composite sealant restorations and other preventive dental treatments. The aim of present article is to report a case of rickets in a 7 year old boy, describing dental findings and to raise awareness on characteristics of this disorder.

Citation: ***Dr. Jigna S Shah and Mahalaxmi Panda.*** "Congenital rickets and dental caries- A case report", *International Journal of Current Research*, 13, (01), *15674-15677*.

INTRODUCTION

Rickets is a disease of calcium metabolism which occurs when infants or children obtain insufficient vitamin D. Rickets is ranked among the top five childhood diseases in developing countries. The most common cause of rickets in India is calcium and vitamin D deficiency. A number of factors have been indicated as being responsible for a high prevalence of Vitamin D deficiency and rickets including religious customs, atmospheric pollution, increased skin pigmentation, vegetarian diets, and maternal Vitamin D deficiency (Virat Galhotra, 2015). Clinical symptoms such as born deformity, spinal curvature, craniotabes, enlargement of the anterior fontanel, rachitic rosary, and joint swelling are important findings in rickets. The specific X-ray findings including a cupping, flaring, and fraying of metaphysis; and the elevation of the level of serum alkaline phosphatase are essential for the diagnosis of rickets (Anil Kumar Nagarajappa, 2015). Vitamin D not only regulates calcium and bone metabolism but also reduces the risk of malignancies, auto immune, cardiovascular and infectious diseases. Its deficiency causes mineralization defects in teeth, leading to poorly mineralized and hypoplastic dentin consisting of calcospherites rather than properly mineralized dentin (Anil Kumar Nagarajappa, 2015). Harris and Sullivan (1960) reported first dental characteristics of rickets.[2] Rickets typically presents at 6-24 months of age. Since, this is critical time period of development of teeth, the dental manifestation include enamel hypoplasia, delayed formation of teeth, and increased the incidence of caries in teeth.

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Deficiency during early childhood can affect permanent teeth and can sometimes lead to tooth loss at a young age in addition to malocclusion and chronic periodontal disease. Light microscopic and ultra-structural examination of teeth reveals abnormalities of dental hard tissues, affecting both enamel and dentin.[1] Here we report the oral manifestations of nutritional rickets in a male child of 7 years with rampant caries.

CASE REPORT

A 7 year old boy reported to oral medicine department with dark brown deposits with cavitations on multiple teeth since 4 years. Patient was normal and healthy uptonthe age of 10 months. Then the patient suffered with various complaints like fever and bone pain for which the parents consulted innumerable physicians and subsequently several medications were tried but with no response. Hence patient was advised for varied blood investigations that revealed reduced vit D levels 10ng/ml, calcium levels were 5.3 mg/dl and alkaline phosphatase level 4945 IU/L. Serum phosphate 3.5 mg/dl, blood urea and creatinine levels were within normal range. Radiological investigations therein revealed deformity with bow legs [Figure 1], enlargement of proximal ends of radius and ulna at wrist joint, knock-knee [Figure 2], change in chest shape [Figure 3]. The patient was diagnosed ascongenital rickets with peripheral myopathy and since then he was under the treatment for the same with vitamin D_3 injections (12 lac IU) intramuscularly and other supportive therpay. There was no history of any renal manifestations, costal and spinal abnormalities. Past history revealed normal eruption of primary teeth but later they showed multiple carious lesions. Family history was non-contributory.

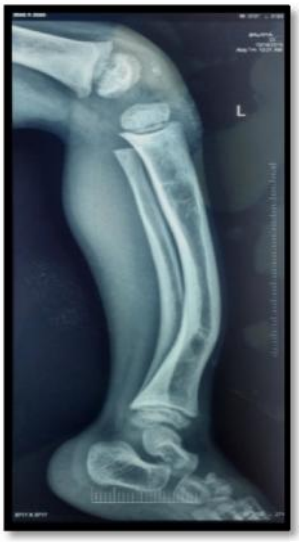

Figure 1A. Shows bowing of tibia and fibula

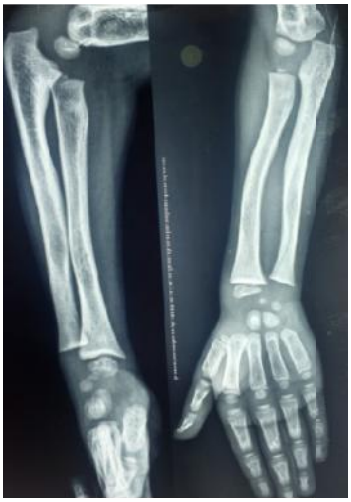

Figure 1B. Shows bowing of radius and ulna

Figure 2. Shows knock knee appearance of legs

When patient visited us he was completely treated for all his systemic manifestations and was under maintenance therapy of vitamin D_3 1000 U sachet once a week and syrup 10 ml BD calcium supplements. His present reports showed vit D levels were improved to 20ng/ml, calcium levels were 9.6 mg/dl and alkaline phosphatise level is 719 IU/L post treatment. On clinical examination, patient appeared pale, lean and presented with short stature, deformed limbs and shape of chest. Intraoral examination revealed generalized erthamatous marginal gingiva.

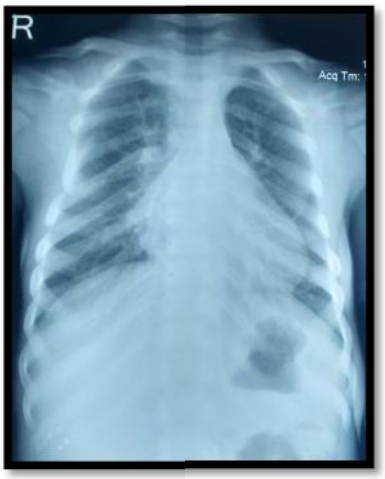

Figure 3. Shows change in chest shape

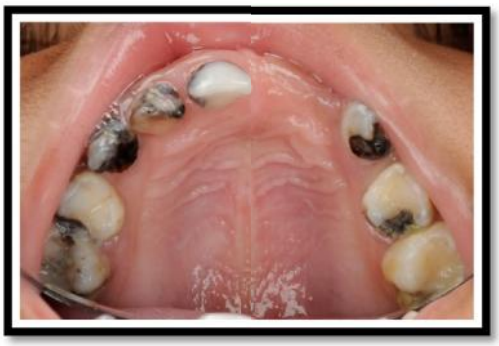

Figure 4.

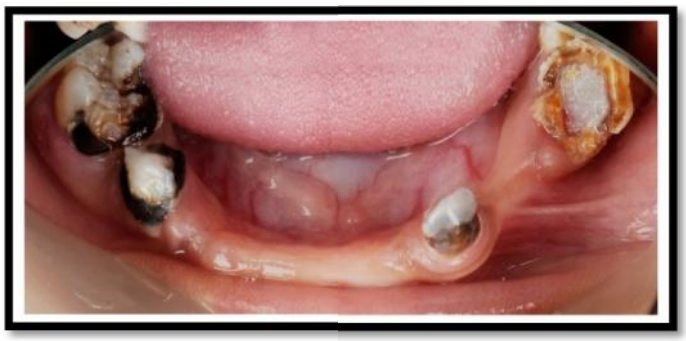

Figure 5.

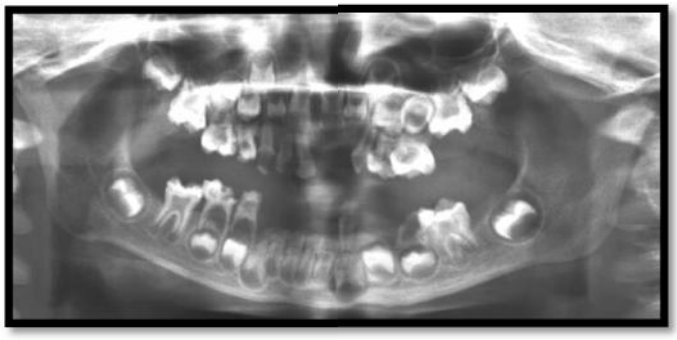

Figure 6.

Hard tissue examination revealed missing 61, 62, 71, 72, 74, 81, 82 with generalized yellowish staining with multiple severe caries affecting all sufaces of teeth, nopenetration of probe in enamel was noticed. Grade III mobility was elicited with 63,73. We were unable to perform periapical radiography due tohigh non-compliance of the patient. [Figure 4 & 5]. Orthopantamogram revealed generalized reducedradiodensity of enamel and dentin with enlarged pulp chambers, in uneruptedmaxillary and mandibular permanent incisors

follicles. [Figure 6] On the basis of patient'shistory, investigation reports, current clinical and radiographic findings, a diagnosis of vitamin Ddeficiency rickets was made. A differential diagnosis of amelogenesisimperfecta and enamelhypoplasia secondary to calcium deficiency were made. Patient was then reffered for extraction of mobile teeth followed by space maintainence till the eruption of succedenous teeth and root canal treatment followed by stainless steel crowns for hypoplastic, carious teeth.

DISCUSSION

Vitamin D is an essential prohormone that on activation by successive hydroxylationsin liver and kidney, binds to the nuclear vitamin D receptor (VDR) and triggers pathways regulating calcium homeostasis, cell proliferation, and cell differentiation. Low, moderate, and extreme deficiencies are defined as 25-hydroxyvitamin D levels 50 nmol/L, 25 nmol/L, and 12.5 nmol/L, respectively and results in impaired bone mineralization and ossification, leading to rickets, osteomalacia, and osteoporosis.[3,4] The current adequate intake value for vitamin D for infants up to 12 months of age is 400 IU.[5,6] The prevalence of vitamin D deficiency is 50-90% in the Indian subcontinent, 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, rachitic rosary, genuvalgum with enlargements of proximity of wrist joint. The diagnosis of rickets is made upon complete physical and dental examination, confirmed by biochemical findings inclusive of hypocalcemia, low-circulating 25(OH)D, hypophosphatemia, elevated serum alkaline phosphatase and hyperparathyroidism (Weisberg *et al.* 2004) with anteroposterior radiographs of rapidly growing skeletal areas as was seen in our case.[1,5] The probable reasons of the wide spread vitamin D deficiency in Indians could be because of low dietary vitamin D intake, high fiber and phytate intake that depletes vitamin D levels, reduced exposure to sunlight, pollution or reduced exposure of skin to sun light because of cultural and traditional habits like "burkha" or "parda".[7] Commonly, a dietary source of vitamin D for vegetarians is milk, provided milk has been fortified with vitamin D. Milk is rarely fortified with vitamin D in India. The vitamin D content of unfortified milk is very low (2 IU/100 mL). Another concern in India is the rampant dilution and/or adulteration of milk and milk products. Low dietary intake of calcium in conjunction with vitamin D insufficiency is associated with secondary hyperparathyroidism. Indian diet has high phytate content that chelates calcium and iron, and reduce intestinal absorption. Vitamin D is degraded at temperatures above 200 °C. Its thermal stability is inversely related to temperature and time. High prevalence of lactose intolerance in India is a major deterrent pertaining milk consumption, further lowering intake of calcium and vitamin D in these individuals.[8]

The only source of vitamin D available to the fetus is that which is derived from the mother, and the vitamin freely crosses the placenta, particularly during the second half of pregnancy.[6] In addition to diet, deprivation of fresh air, exercise and sunlight, profoundly alters the metabolism of the child and produces aberration in the growth not only of the bones and teeth, but in probably every tissue of the body.[9]This is probable cause of disease in our patient. Its deficiency during gestation affects primary teeth, whereas during early childhood it affects permanent teeth. Both enamel and dentin are affected in nutritional deficiency anemia.[3] In rickets caries is exceedingly common, and hypoplasia, or defective calcification of the enamel, is well marked.[10-11] A hypoplastic condition of the teeth is characterized to the naked eye by a defective formation of the enamel and frequently stunted growth of the teeth. The defect usually extends from the cutting edge and may, in severe cases, involve the whole crown as seen in our case. Usually the depth of the defect is greater in the enamel of incisors and is almost pathognomonic of rickets. Rickets is the only condition which interferes with the deposition of calcium over this prolonged period.[11]Enamel hypoplasia showed a predisposition to those teeth, which were developing at the time of disease most common in primary teeth, and was, the chief factor in bringing about premature decay. Such teeth are more prone to caries because of decreased mineralization.[7]Caries is universal and extensive so that it completely masked the hypoplasia. Naturally the lower teeth, which lie in the well of the mouth, will suffer more seriously from deleterious influences which surround the teeth and decay out of all proportion to the others, due to flattening and grinding of molars and their earlier eruption as seen in our patient. It is to be attributed rather to the main part of the enamel of the crown having been laid down in the first two years of life, when rickety conditions are operative.Also there might be increased the incidence of structural developmental defects (lamellae) that may lead to faster ingress of bacteria leading to an early pulpal involvement and abnormal serum alkaline phosphatase levels might hinder the transition from bud to cap stage.[1]

The chronological onset ofcrown mineralization is tooth-groupdependent. Consequently, systemicdiseases or intoxications at a givenperiod can affect various teeth differently.In our case tooth lesions were more extensive and mostly affected the whole crown surface of decidous central incisors and first molars. Crown mineralization of primary teeth occurs from gestation to 12 months of age. Given that the crown of the primary second molars mineralizes from the sixth month in utero to 10 to 12 months after birth, this deficiency may have been already present during gestation.[3]Vitamin D deficient children have been found to have retarded eruption. 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.[1]In our case, orthopantomogrm showed well formed and normal radiodensity of permanent premolars and 2nd molars that were formed in post treatment phase. Management includes providing dietary and commercial vitamin D and calcium supplements. 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, topical fluoride applications, pit and fissure sealants, maintenance of good oral hygiene, extraction of teeth with periradicular abscesses and space maintenance for replacement or closure of space.[1]Vitamin D is the drug of choice with calcium supplements which helps to reduce the dosage of vitamin D related compounds required for treatment. Diet analysis and investigation to exclude causes other than nutrition should be undertaken. Treatment continues until there is radiographic evidence of healing and a return to normalplasma

concentrations of calcium, phosphorus, and alkaline phosphatase.[1]

Conclusion

Rickets can adversely affect dental development and delay in commencement of medicaltreatment may lead to permanent deficit in dental development. Enamel defects increase the incidence of dental caries. Thedentist as well as the pediatrician should be made aware of its features so that early interventioncan prevent subsequent serious and more invasive dental procedures. Ideally, a first dental visit at 12 months ofage enables parents to gauge theimportance of oral health, feeding, and annual dental checkups. If parents failto bring their children at that youngage, a first visit at age 3 is essential; allprimary teeth should have erupted bythen. A checkup at 6 to 7 years of ageallows the dentist to check for enamelhypoplasia or caries on permanentfirst molars and incisors.

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