Effect of nutritional rickets on dental development in North Indian children: A prospective study

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ABSTRACT
Rickets is a disorder caused by a lack of Vitamin D, calcium or phosphate. It leads to softening and weakening of the bones. Dental manifestation of rickets includes enamel hypoplasia and delayed tooth eruption. The present prospective study was carried out to report various dental anomalies caused due to rickets in children. The present study was carried out on 120 pediatric subjects between the age-group of group 6-18 years of age with history of rickets in infancy as reported by pediatrician. The developmental defect of enamel index was used for recording enamel lesions along with radiographs. Statistical analysis was done using SPSS version 16 (SPSS, Chicago, IL, USA). Enamel hypoplasia was detected in 90 cases, missing teeth were detected in 45 cases, bilaterally missing mandibular second premolars were detected in 30 cases, seven cases were found with bilaterally missing maxillary first premolars, maxillary canines, and mandibular second premolars. Significant dental disturbances, e.g., hypoplasia, missing teeth can occur in children suffering from rickets in infancy. Careful and thorough management of this disorder can be done by conservative dental treatments such as composite sealant restorations and other preventive dental treatments.

Key words: CHypo-calcification, Rickets, Treatment, Vitamin D

INTRODUCTION
Rickets is a softening of bones in children due to deficiency or impaired metabolism of Vitamin D, phosphorus or calcium, potentially leading to fractures and deformity.[1] Clinical symptoms such as born deformity, spinal curvature, craniofibrosis, enlargement of the anterior fontanel, rachitic rosary, and joint swelling are important findings in rickets.[2] 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.[2] The Greek-derived word “rachitis” meaning inflammation of the spine was adopted as the scientific term for rickets, due chiefly to the words similarity in sound.[3]

Rickets is ranked among the top five childhood diseases in developing countries.[4] In Indian subcontinent, both calcium and Vitamin D deficiencies are responsible for rickets. 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.[5-8]

Rickets typically presents at 6-24 months of age.[9] 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 cavities in teeth (dental caries).[10] Deficiency during early childhood can affect permanent teeth and ensuing caries can sometimes lead to tooth loss at a young age in addition to malocclusion and chronic periodontal disease.[10] Light microscopic and ultra-structural examination of teeth of such children reveal abnormalities of dental hard tissues, affecting both enamel and dentin.[11] As the dental manifestations appear much later after eruption of teeth, there are very few longitudinal and/or retrospective studies evaluating the effect of rickets on tooth development, which could be because of lack

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of database at most institutes and prospective follow-up. Hence, this prospective study was carried out to evaluate the effect of rickets in infancy on dentition if any and enumerate the anomalies.

MATERIALS AND METHODS

Study setting
A hospital-based prospective study was carried out for a period of 2 (2010-2012) years in a dental hospital in Ludhiana. The hospital provides dental treatment to all population classes. Ethical clearance was obtained from the hospital authorities.

Study population and sample size
Pediatric patients of both the genders of age-group 6-18 years of age with history of rickets in infancy as reported by pediatrician were examined clinically and diagnostic procedures carried out after parental consent. The age-group of the patients were selected according to the age which was mentioned in the medical records when the patient visited the hospital. The exclusion criteria for the study were patients with history of fluorosis, any syndromes or any congenital skeletal deformities. After scanning the medical records, a total of 120 patients (72 males and 48 females) were selected and enrolled for study after taking informed consent from the parents. Data collection was done on a self-structured format exclusively made for the study [Table 1]. The developmental defect of enamel index was used for recording enamel lesions. The examination of teeth with enamel hypoplasia was conducted using Federation Dentaire Internationale. Buccal and lingual surfaces of each tooth were examined and defects were recorded. To facilitate the recording of defect intensity, contract codes were used. Any anomalies of tooth size, shape, and number were noted and co-related with radiographs.

Statistical analysis
The data were first transferred to a personal computer and uploaded into Microsoft Excel (Microsoft Corporation, USA). Data were statistically analyzed using SPSS package version 16 (SPSS, Chicago, IL, USA) in terms of number and percentages only and no other statistical tests were used.

RESULTS
A total of 120 patients with positive history of rickets in infancy were examined. Dental manifestations due to the effect of rickets on tooth development are depicted in Figure 1. Enamel hypoplasia was detected in 90 (75%) cases [Figure 2], missing teeth were detected in 45 (37.5%) cases [Figure 3], bilaterally missing mandibular second premolars were detected in 30 (25%) cases, seven cases were found with bilaterally missing maxillary first premolars, maxillary canines, and mandibular second premolars (6.25%) and seven cases were detected with bilaterally missing deciduous lateral incisor (6.25%). Other dental findings revealed spontaneous gingival and dental abscesses occurring without history of trauma and caries. Radiographic examinations revealed large pulp chambers, short roots, poorly defined lamina dura, and hypoplastic alveolar ridge in majority of patients.

DISCUSSION
Rickets is a disease of infancy affecting calcium metabolism. It could be acquired or inherited in the form of X-linked disorder. Calcium or Vitamin D deficiency is an acquired cause while inherited form rarely occurs as X-linked.
dominant trait. The most common cause of rickets in India is calcium and Vitamin D deficiency.\[13\]

Of the 120 patients selected for the study, enamel hypoplasia was noticed in 90 patients while missing teeth were present in 45 cases. Enamel hypoplasia has been reported in the previous studies on dental manifestations of rickets as well.\[10,14\] Although these defects are more common in the hereditary form of rickets, the cases with enamel hypoplasia showed a predisposition to those teeth, which were developing at the time of disease.\[15\] Such teeth are more prone to caries because of decreased mineralization. Hence, preventive regimen comprising of periodical examinations, topical fluoride application, application of pit and fissure sealants, and maintenance of good oral hygiene should be performed. 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.

Increased an organic substance might necessitate sodium hypochlorite treatment prior to composite restorations as indicated in the protocol for molar-incisor hypomineralization.\[18\]

Missing teeth were seen in 45 cases, of which mandibular second premolars were most commonly missing teeth followed by maxillary second premolars which have not been routinely observed in previous studies. Nutritional and X-linked - Vitamin D deficiency rickets presents itself at around 4-18 months of age, while calcium deficiency rickets presents after 2 years of age.\[9\] This is the time that corresponds to premolar initiation and calcification. Besides calcium and Vitamin D deficiency, abnormal serum alkaline phosphatase levels which are usually seen in rickets, might hinder the transition from bud to cap stage. Missing teeth require comprehensive evaluation as regards to their space maintenance for replacement or closure of space. This requires regular dental visits to chalk out treatment strategies. Besides the studies reveal high pulp horns increase occurrence of abscesses that necessitate increased professional care to keep these children free of disease.

CONCLUSION

Rickets is a disease that looms over the healthy development of our future generations. This article explores the dental manifestations of rickets, although further research over a larger database and long follow-up is required to assess the pattern of dental manifestations. This paper shows significant dental disturbances, e.g., hypoplasia, missing teeth can occur in children with rickets in infancy. Nutritional rickets and its dental consequences visible later in life have not been elaborately studied. Pediatric dentists can be more aware of the scars of this medically crippling condition and plan treatment more judiciously and conservatively in case of missing teeth. Further research can be done to ascertain the relation between missing teeth and rickets and pattern of missing teeth.

REFERENCES


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