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A RARE CASE REPORT OF RICKETS

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ABSTRACT

One of the major micronutrient deficiencies, which affects the growing bone in children is *Rickets*. Derived from the word 'rickets' Rickets means 'twisted', which refers to the bony deformities or 'bowlegs', the characteristic feature of rickets. Nutritional rickets (NR) is still the most common form of growing bone disease despite the efforts of health care providers to reduce the incidence of the disease.[1] It is mainly a nutritional deficiency of Vitamin D and is rarely seen as secondary to dietary deficiency of Calcium or Phosphorus, due to the inefficient absorption of Calcium and Phosphorus. Vitamin D deficiency predominates in high-latitude countries in at-risk groups (dark skin, reduced sun exposure, infants, and pregnant and lactating women) but is emerging in some tropical countries due to sun avoidance behavior. Calcium deficiency predominates in tropical countries, especially in the malnourished population.[2] The prevalence of the disease has increased in both developed and developing countries. African, Middle Eastern, and Asian countries have a wide prevalence rate of 10% to 70%. About 85% of the Indian population is vitamin D deficient despite abundant sunlight.[3] Here is a case of Rickets with peculiar features.

Keywords: Rickets, Vitamin D deficiency, Developmental delay, Soft-skull, Genu Valgum.

INTRODUCTION

Vitamin D deficiency is the leading cause of nutritional rickets in children and thus remains a major cause of morbidity. This is seen both in developing and developed countries. Rickets was first described in association with skeletal deformities by Glisson during the middle of the 17th century in London.[4] This is considered an old disease that has affected children globally for ages. Nutritional rickets was once considered virtually eradicated by fortification of milk or direct administration of Vitamin D in developed countries. Recently it has been found that children who are exclusively breastfed are more prone to nutritional rickets as it's a poor source of Vitamin D. Vitamin D deficiency is not just related to poor dietary intake and insufficient exposure to sunlight, but malabsorption syndromes, anti-convulsant drugs, and chronic liver disease.

In the whole process of deficiency of Vitamin D, the clinical features dominate the bony manifestations, this is because the deficiency of Vitamin D leads to increased levels of Parathormone which causes concomitant loss of phosphate from the kidney leading to low serum phosphate levels. This phosphate plays a major role in the enchondral calcification of the cartilaginous growth plate of the bones. Enchondral calcification is the process where the chondrocytes in the growth plate hypertrophy and undergo apoptosis followed by mineralization. Phosphate is responsible for the apoptosis of the chondrocytes, thus with low serum phosphate, apoptosis is hampered, and hypertrophy of the growth plate continues which leads to characteristic swellings at the growth plates. Mineralization also gets affected and thus there is bending of the weight-bearing bones.[5]

CASE REPORT:

A 7-year-old boy, firstborn to third-degree consanguineously married parents, was brought by his father with difficulty in walking since 5 years, cough and cold for three days. History of present illness states that he had a cold starting with sneezing in the morning times and had teary eyes while sneezing. The cough was mild, dry, and more during the night. History reveals that the child had foot deformity (Congenital talipes equinovarus) since birth for which a Ponseti cast was done. Later on, it was noticed by parents that the child had difficulty walking since one year of age, he had generalized weakness and overall growth was not normal. Birth history includes, the baby was full-term, born through normal vaginal delivery, had delayed cry, admitted to NICU for 3 days. The baby had no episodes of seizures and birth weight was3.5kg. The mother died while delivering the second baby, who is a 4-year-old boy, alive and healthy now. Father didn't remember much of the developmental milestones. Immunization and family history was normal.

On examination child was presented with a soft skull (like a ping-pong ball), short stature, Rachitic rosary, Harrison's sulcus, Pigeon Chest, widening of the bilateral wrist joint, Knock knees (Genu valgum), Kyphoscoliosis, mild waddling gait, and muscle power 3/5.

His weight was 14.4 kg, height was 92cm, and head circumference was 48 cm. From the above examination, the patient was diagnosed with rickets.

DISCUSSION

In the present case, it was observed that the manifestations had been for a long and no treatment was taken in time, which led to the above condition of the patient. The inadequate intake of vitamin D and insufficient exposure to sunlight might be one of the prominent causes. As per the IAP growth Charts, as the Height-for-age is less than the 3rd percentile, the child is stunted and even his Weight-for-age is less than the 3rd percentile. In rickets, vascularization, chondrocyte apoptosis, and mineralization of the cartilage matrix surrounding the apoptotic chondrocytes are impaired. The delayed chondrocyte apoptosis results in the accumulation of hypertrophic chondrocytes in the growth plate, which further leads to a loss in their columnar arrangement and, therefore, disorganization of the growth plate. The same is evident in this case as hypertrophied costochondral junctions, swelling at the end of long bones.[8]Rickets associated with vitamin D deficiency usually occur between 6 months and 2 years of age, while rickets associated with very low calcium

status usually occurs in older children, when breast-feeding stops. In this case, as told by the informant the child had difficulty walking since one year of age, thus this could be considered under rickets associated with vitamin D deficiency between 6 months and 2 years of age.[9] When examined clinically the case presented with softening or thinning of the skull bones (craniotabes), which was examined by applying pressure to the skull, there was an inward collapse typically followed by a snapping back after removing pressure. (like ping-pong ball).[10]Other signs included swelling of the wrist (see Fig. 5,7), knee, and ankle (see Fig. 1,2,3) and, as a result of weight-bearing, legs deformities such as bowing of the arms, knock-knees(genu-valgum) (see Fig.2), difficulty in walking were seen. . Other signs of rickets such as swelling of the costochondral joints of the ribs (rachitic rosary, see Fig. 4,6), deformity of the soft rib cage, Kyphoscoliosis were also noticed. Non-osseous features included poor linear growth, delayed motor development, lethargy delayed tooth eruption and poor-quality tooth enamel, and predisposition to respiratory infections during infancy as told by the informant. However, detailed laboratory analysis and examination were needed. Mother died 4 years back and thus even this would have impacted the overall health of the child Vitamin D deficiency remains one of the major causes of the poor growth of kids around the globe. Thus, there's a need to intensify the programs related to Vitamin D supplementation. As recently during the Covid pandemic, it has been seen that Vitamin D does play important role in boosting immunity as well.

CONCLUSION

Vitamin D deficiency is a known and one of the important cause of rickets. It affects the children around the globe and even in topical countries where the sunlight is abundant.[9] Thus, identification of the Vitamin D deficiency on time and proper management is required. Timely intervention helps in correcting the bone metabolism which prevents damage to bones.[10] As Vitamin D also boosts immunity, it improves the overall health of the child by preventing infections and thus improves the quality of life.[11]

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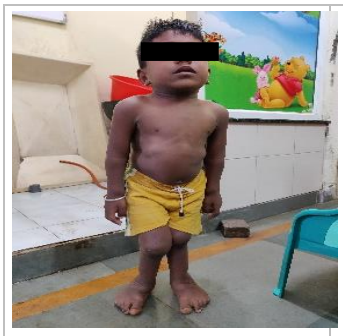
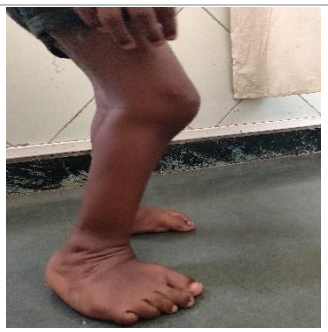
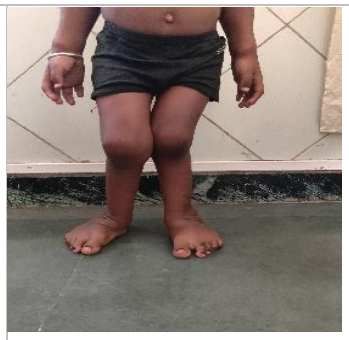
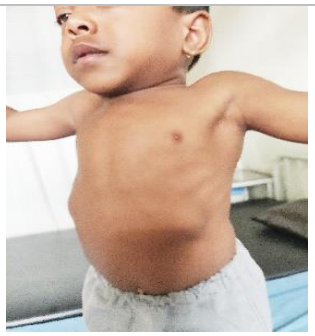
Pictures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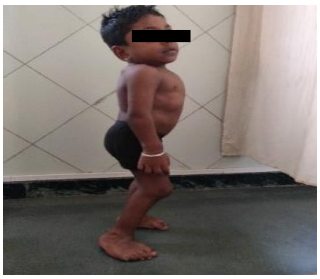	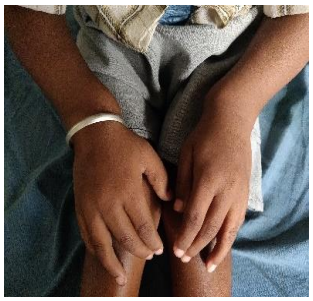	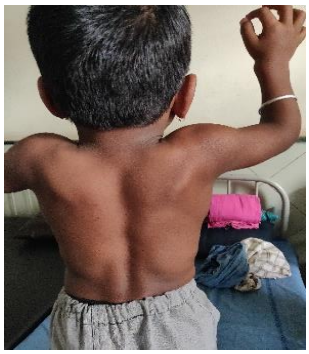	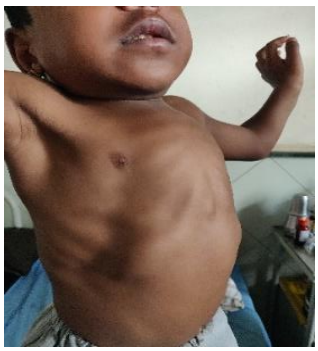
Figure 5	Figure 6	Figure 7	Figure 8
Figure 9	Figure 10		

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