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# INADEQUATE SUNLIGHT: CAUSATIVE FOR RICKETS IN COVID PANDEMIC

Pediatrics

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## ABSTRACT

Vitamin D-dependent rickets is a disorder of bone development that leads to softening and weakening of the bones (rickets). It is characterised by early-onset rickets due to the inability to maintain adequate concentrations of active forms of vitamin D or a failure to respond fully to activate vitamin D. There are two types of vitamin D dependent rickets (VDDR) that cause rickets in children. Vitamin D dependent rickets type 1 is caused by an inborn error of vitamin D metabolism and vitamin D dependent rickets type 2 is caused by a defect in vitamin D receptor .Here, we present to you a case of 3 years of female child who presented with signs of vitamin D deficiency.

## KEYWORDS

Vitamin D Dependent, Inborn Error Of Metabolism, Receptor Defect.

## INTRODUCTION

Rickets is a condition that results in weak or soft bones in children. Symptoms include bowing of legs, stunted growth, bone pain, large forehead, and trouble sleeping. Complications may include bone fractures, muscle spasms, an abnormally curved spine, or intellectual disability. The most common cause of rickets is a vitamin D deficiency. This can result from eating a diet without enough vitamin D, dark skin, too little sun exposure, exclusive breastfeeding without vitamin D supplementation, celiac disease, and certain genetic conditions. Other factors may include not enough calcium or phosphorus.[4][5] The underlying mechanism involves insufficient calcification of the growth plate.[1,2]

The recognition that genetic defects in vitamin D activation or responsiveness could cause rickets evolved from detailed clinical and biochemical studies that identified the critical role that vitamin D played in ensuring normal bone and mineral metabolism. Historically, rickets has been considered a manifestation of poor socioeconomic status and a diet that failed to provide adequate amounts of vitamin D and/or calcium [3,4]. The primary cause of vitamin D deficiency usually involves interplay of nutritional inadequacy and lack of sunlight exposure with overlapping contributions by cultural, environmental, and genetic factors[6]

Both skeletal and extra skeletal manifestations are present in patients with nutritional rickets. Skeletal symptoms include swollen wrists and ankles, delayed tooth eruption, leg deformity, rachitic rosary, frontal bossing, craniotabes, and bone pain.[5,7] On the other hand, extra skeletal findings include muscle weakness and hypocalcemic seizures.[7] Nutritional/medical history, biochemical testing, and radiographs are used to diagnose nutritional rickets. Along with low level of 25 (OH) vitamin D, laboratory findings are also helpful in its diagnosis as well as in differentiating it from other causes of rickets [7]

The prevalence of VDDR was approximately 30% two decades ago; however, in the present time, it has reduced to 0.8%. The clinical presentations include weak bones causing bone pain, delayed growth, short stature, and bowed legs due to impaired weight bearing capacity of weakened legs. The syndrome usually presents with these symptoms and is also associated with alopecia in severe cases.[8] In VDDR, the serum calcium levels are either normal or low along with elevated alkaline phosphatase level and low serum phosphorus levels. The diagnostic hallmark of VDDR type II is the increased serum levels of 1, 25-dihydroxyvitamin D3 due to the mutations of VDR gene. The mutations alter the absorption of calcium and phosphate resulting in hypocalcemia and hypophosphatemia, leading to VDDR type II and deranged vitamin D activity in other organs.[9]

Vitamin D is essential for bone growth, mineralization, and absorption of calcium and phosphate, which is deficient in rickets.[10] Vitamin D is obtained by dietary intestinal absorption and through the skin and is available as vitamin D2 and vitamin D3. Initially, in the liver, vitamin D is hydroxylated to 25-hydroxyvitamin D3 (25-OHD3). Later, 25-OHD3 bound to the vitamin-binding protein is transported to the kidney, where it undergoes hydroxylation to form the hormonally active metabolite, 1,25-dihydroxyvitamin D3 (1,25-OH2D3).[11] The tissue receptors for vitamin D metabolites are localized in various organs, including kidney, intestine, pancreas, parathyroid gland, muscle, pituitary, skin, and bones and 1,25-OH2D3 binds specifically to a receptor in the nuclei to stimulate calcium transport and also controls the expression of target genes mediated through the nuclear VDR.[12] The treatment of nutritional vitamin D deficiency with cholecalciferol consists of an intensive phase followed by a maintenance phase.

## CASE REPORT

A 3 years old girl child presented to our department with complaints of difficulty in walking first observed at the age of 2.5 years . On clinical examination, Central nervous system examination was normal but child had typical features of vitamin D dependent rickets . Head to toe examination revealed frontal bossing , rachitic rosary , wrist widening , genu valgum and double malleolus . Detailed work up and relevant investigations were done . CBC showed haemoglobin of 12.3g/dl , WBC of 5200/ul and platelet count of 3L/mm3. Calcium was 9.1mg/dl(Normal 8.8-10.2) , potassium was 4.3mmol/l (Normal 3.5-5.5) , phosphorus was 2mg/dl (Normal 2.7-4.5) which was lower than the normal range, alkaline phosphatase was 2556.7 U/L (Normal 64-306) which was significantly raised and CK-MB was 27 U/L (Normal 0-24). Vitamin D levels were done which was extremely low with a value of 3ng/ml . Xray of wrist was done which showed classical signs of rickets which are widening of epiphyseal ends with increased space between epiphysis and metaphysis, cupping , fraying ,splaying at ends of long bones (Radius and Ulna) . After confirming the diagnosis of rickets, Child was given mega dose of vitamin D and calcium following which child was advised regular follow up . After one month of treatment , repeat x-ray wrist was done which showed white line of calcification which is an indicator of healing rickets and vitamin D levels were >150ng/ml. There was clinical improvement as well in

form of reduced widening of rickets and reduced genu valgum. Hence , the child was started on maintenance dose of vitamin D and calcium and was asked for regular follow up.

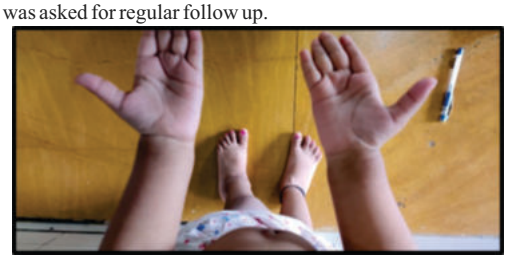

**Figure-1 Image Showing Widening Of Wrists At Time Of Presentation**

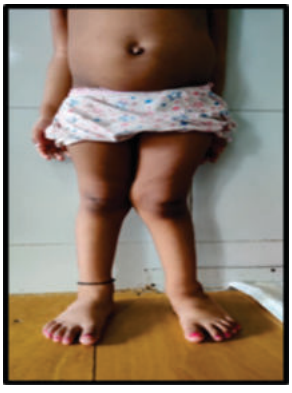

**Figure-2 Image Showing Genu Valgum At The Time Of Presentation**

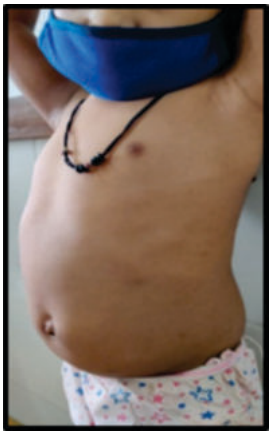

**Figure-3 Image Showing Rachitic Rosary At The Time Of Presentation**

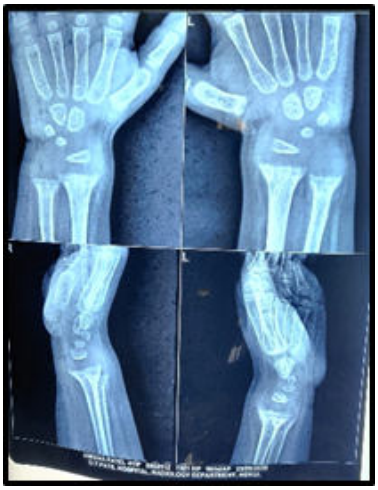

**Figure-4 X-ray Of Wrist Showing Widening Of Epiphyseal Ends With Increased Space Between Epiphysis And Metaphysis, Cupping, Fraying, Splaying At Ends Of Long Bones (radius And Ulna)**

**Figure-5 Image Showing Reduced Widening Of Wrist Post Vitamin D Treatment**

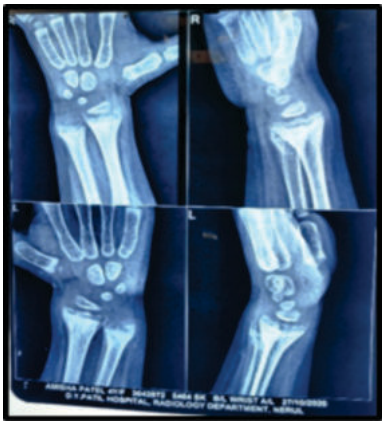

**Figure-6 Image Showing X-ray Wrist With White Line Of Calcification Post Vitamin-d Treatment**

## DISCUSSION:-

Child was classical case of Vitamin D dependant Rickets diagnosed at the time of presentation. We clinically diagnosed our patient as rickets based on history of no exposure to Sunlight during COVID times and associated clinical features supporting the diagnosis. Also laboratory evaluation helped in diagnosing the case. After nutritional cause of rickets, exposure to sunlight plays an important role in causing rickets in school going children.

Due to lockdown during COVD times, patient was not able to go out and play causing almost no exposure to sunlight, hence Rickets. In the present case patient had typical features of vitamin D dependent rickets which improved after administration of megadose of vitamin D and calcium thus establishing the diagnosis of vitamin D dependent rickets.

## CONCLUSION:

Rickets is a disorder of growing school going children that arises from defective mineralization of the growth plate. Nutritional rickets is a preventable disease by maintaining an adequate intake of vitamin D through both dietary and sunlight exposure.

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