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CASE REPORT

# Needless rickets: alertness

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**Keywords:**
Child,
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## Abstract

**Objectives:** The present study aims to highlight the resurgence of deficient rickets and the importance of early treatment to prevent disabling sequelae. **Case Report:** Child aged 11 months, 9 months corrected, born by caesarean section due to specific hypertensive disease of pregnancy and fetal centralization, premature 30 weeks, weight 1,065 grams. He received exclusive breast milk up to six months of age, with good weight gain. Evolves with primary malnutrition after introducing complementary diet with low dietary acceptance, during a pediatric consultation it was found developmental delay secondary to the state of malnutrition characterizing deficient rickets, facing the growth curves using the World Health Organization (WHO) graphs, with weight and length below Z-score - 3 for age. Radiological evidence showed generalized bone demineralization, with widening of the extremities and distal femoral metaphysis and cup-shaped proximal tibia. He was admitted to a hospital in the Federal District for seven days where he received diet by nasogastric tube and oral, at the end of hospitalization evolved with appetite return and good weight gain, maintains outpatient follow-up on continuous use of vitamin D and A, and ferrous sulfate. What stands out here is the importance of early diagnosis, as well as instituting mechanisms and behaviors that contribute to treatment adherence.

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## INTRODUCTION

Rickets and osteomalacia are diseases characterized by a defect in bone mineralization. Osteomalacia occurs due to a defect in the mineralization of the bone matrix, it presents in adulthood and is one of the causes of low bone mineral density; Rickets is the mineralization defect of the growth cartilages in children and presents with growth retardation and skeletal deformities[1].

The delay in bone mineralization, characteristic of rickets, is due to anomalies in the homeostatic mechanisms that control the concentrations of calcium and phosphorus ions in extracellular fluids, which are essential for mineralization. Several homeostatic mechanisms intervene to maintain normal levels of these ions, the main one linked to vitamin D metabolism. Several etiologies can lead to rickets, such as primary disorders of calcium and phosphorus metabolism, liver and kidney diseases, deficiency of vitamin D precursors , the latter being called deficiency rickets[2].

With a high prevalence until three decades ago, rickets remains a public health problem in developed countries, where it has reappeared in the last decade due to changes in lifestyle and eating habits[3,4].

Epidemiological data are scarce because this disease was almost extinct at the end of the 20th century. Despite concerns about the persistence and potential increase in the incidence of rickets deficiency in developed countries, its current incidence is unknown[5].

## CASE REPORT

Child with 11 months of chronological age, 9 months of corrected age, born by cesarean section, due to specific hypertensive disease of pregnancy and fetal centralization, premature at 30 weeks, weight 1065 grams. She received exclusive breast milk until six months of age, with good weight gain. It evolves with primary malnutrition after the introduction of complementary feeding with low acceptance of the diet, during a pediatric consultation, a delay in development secondary to the state of malnutrition was observed, characterizing deficiency rickets, in view of the growth curves using the graphs of the World Health Organization (WHO) with weight and length below the Z score - 3 for age. Radiological evidence showed generalized bone demineralization, with cup-shaped enlargement of the extremities and metaphyses of the distal femur and proximal tibia. Serum phosphorus: 2.2 mg/ dL (reference value (RV): 4 - 7). He was admitted to a hospital in the Federal District for seven days where he received a diet by nasogastric and oral tube, at the end of hospitalization he evolved with a return of appetite and good weight gain, he maintains outpatient follow-up in continuous use of vitamin D and A, in addition to ferrous sulfate.

## DISCUSSION

Vitamin D deficiency is a major cause of both rickets and osteomalacia. This vitamin is normally synthesized in human skin exposed to ultraviolet B (UVB) rays and is transformed in the liver into 25-hydroxyvitamin D, which is the most abundant form of the vitamin and is measured as its sufficiency marker. In the kidney, under stricter control, the most active form of this vitamin, 1,25-dihydroxyvitamin D, is synthesized. One of the main causes of rickets is hypophosphatemia , which can occur due to tubular loss of phosphate secondary to excess parathyroid hormone. (PTH) or not. This change may occur as part of Fanconi syndrome , in which there is a proximal tubular defect, with multiple myeloma as the most common cause in adults[6]. Another cause is primary phosphorus-losing tubulopathies , which can be inherited or acquired[7].

The most common causes of hypocalcemic rickets are vitamin D deficiency or resistance to its action, while hypophosphatemic rickets is most commonly caused by renal phosphate loss. The causes of rickets can also be divided into deficiency (comprising inadequate sun exposure or inadequate intake of vitamin D, calcium or phosphorus), vitamin D dependent (type I being secondary to a genetic defect that decreases renal hydroxylation of the vitamin D and type II secondary to a genetic defect in the vitamin D receptor) and vitamin D resistant (due to renal phosphate loss)[1].

The evidence collected in the clinical history of the patient in question, in addition to the laboratory data, elucidate consistent evidence that the cause of the rickets of the patient in question is lacking. It was not possible to measure vitamin D.

Weight and height delay and bone irregularities in patients with rickets are due to prolonged hypophosphatemia , as it is known that maintaining intra and extracellular phosphate levels within a narrow range is important for several biological processes, including energy metabolism, skeletal development and bone integrity. In addition, phosphorus deficiency can compromise chondrocyte maintenance, causing blockage of bone neoformation, resulting in growth delay and rickets[8]. What stands out in this case is that the diagnosis was made before the radiological changes described in the literature occurred in most cases.

Radiologically, rickets is characterized by enlarged epiphyses and metaphyses, “cup-shaped”, with irregular mineralization lines, without defined contours and delay in maturation. In the other regions, signs of osteomalacia are observed, with generalized osteopenia, curving of the long bones, varus or valgus in the lower limbs, pseudo-fractures (Looser ‘s zones) which are more frequent in the femoral neck, shoulder blade and pubis, fractures, deformities in the rib cage and spine: biconcave vertebrae, kyphoscoliosis, marked lordosis. Fluctuations in disease severity during growth result in the appearance of radiodense lines , parallel to the metaphysis. In vitamin D-dependent cases with secondary hyperparathyroidism, there

are areas of subperiosteal resorption and cysts[9]. In the case in question, the radiological alteration was characterized by the brush border, metaphyseal enlargement, low mineralization of the cup-shaped secondary ossification nucleus in the distal femur and proximal tibia (Fig. 1).

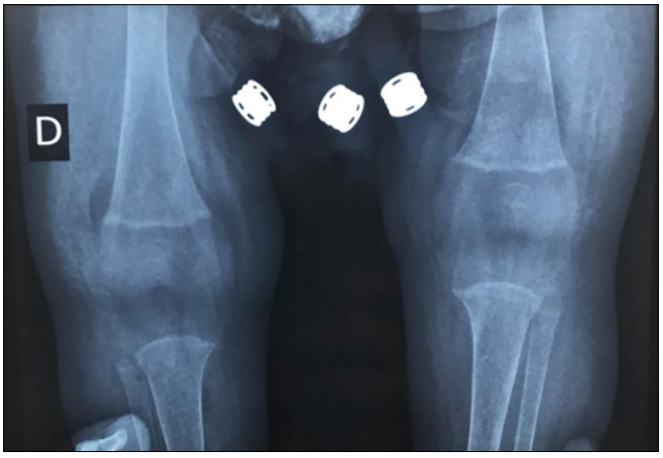


**Figure 1.** Brush border, metaphyseal enlargement , cup-shaped in distal femur and.

## CONCLUSION

Given the evidence of the case described here, it can be concluded that early diagnosis is essential to reduce the likelihood of sequelae resulting from mineral deficiencies. Adherence to drug treatment, as well as the establishment of healthy eating habits are extremely important in the prevention and treatment related to this preventable cause of morbidity and mortality.

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