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Case Report

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Rickets-A Case Report

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ABSTRACT: Rickets is a disease of growing bones caused by unmineralized matrix at the growth plates in children only before fusion of the epiphyses. Because growth plate cartilage and osteoid continue to expand but mineralization is inadequate, the growth plate thickens. On examination there was box shaped head, closed fontanelles and cranial sutures, bowed legs and waddling gait. On investigation, Serum calcium decreased, serum phosphate decreased, serum Vitamin D decreased, serum alkaline phosphatase increased and normal serum creatinine & SGPT.X-Ray of the knee, ankle and wrists show splaying, cupping and fraying of the metaphysis of the long bones with widening of the growth plates resulting slight bowing of the femur and tibia. The patient was treated with Vitamin D. Stoss therapy of Vit D 400,000 IU was given by intramuscular route in 4 divided doses over 1day. This is followed by Vit D 600 IU daily and oral calcium 500mg daily for 3 months. Orthopedic BRACE was advised after radiological healing of bone.

Keywords: Rickets, Vitamin D, Calcium, Bow Legs.

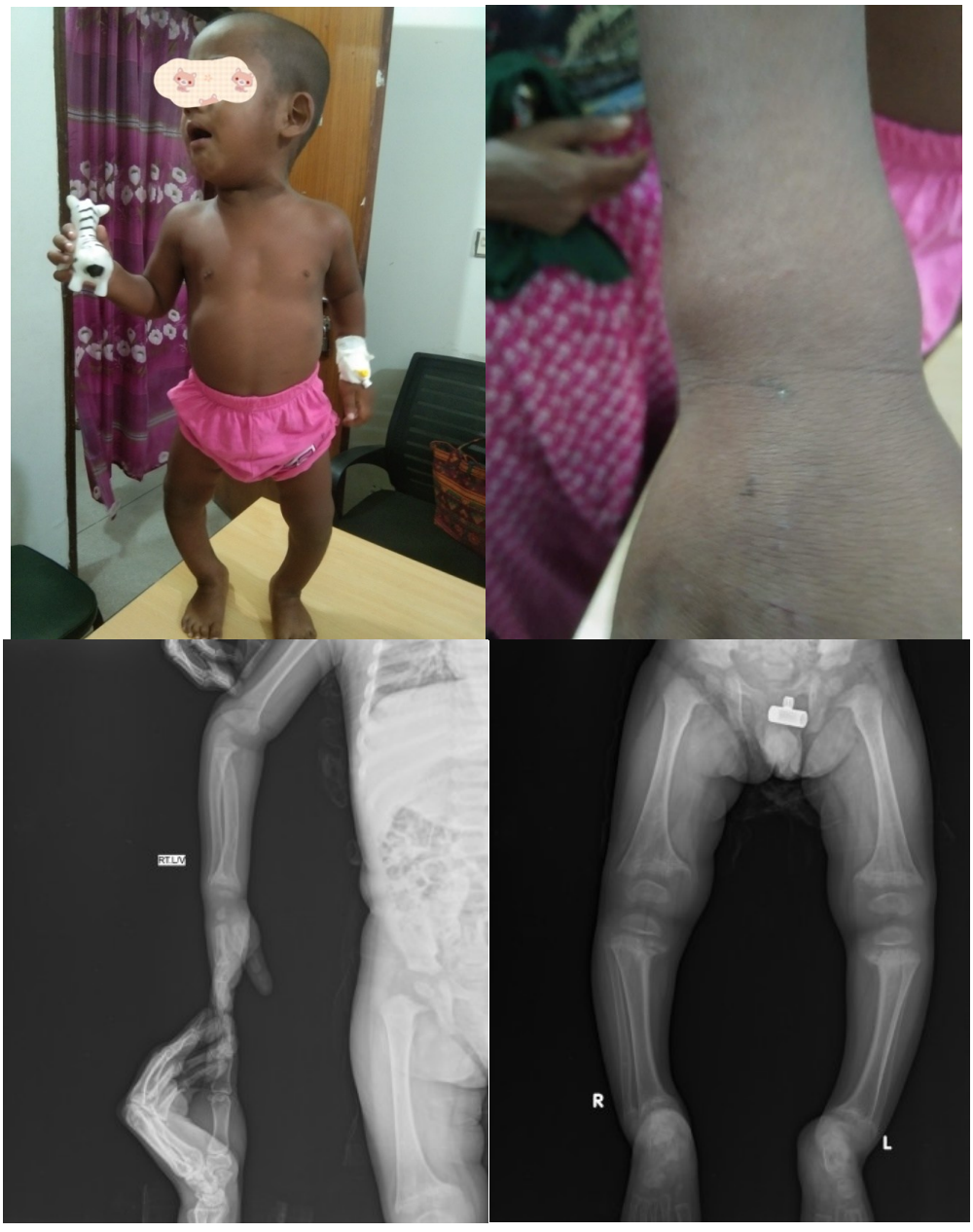
INTRODUCTION

Rickets are a disease of growing bone caused by unmineralized matrix at the growth plates in children only before fusion of the epiphyses.[1] Nutritional Rickets are caused by deficiency of Vit D, calcium and phosphate, leading to softening and weakening of bones. Factors associated with the pathogenesis of rickets include maternal Vit D deficiency, prematurity, lack of sufficient sunlight exposure, darkly pigmented skin, less intake of dairy products and vegetarian diet containing high fiber and phytate.[2, 3] Though it is rare, there are also many genetic causes of rickets. Affected children typically present at the age of 18 months with delayed motor development, hypotonia, and short stature and they have knock knees or bowed legs.[3] We present a case of rickets in a 2 year 7 month old boy along with a brief review of the case to build awareness.

CASE REPORT

Rahmat Ali, a 2 year and 7 month old completely immunized boy of non-consanguineous parents of poor socioeconomic family, born at term by LUCS at private clinic, hailing from Shaluka, Naogaon Sadar, Naogaon attended at OPD, of 250 bed general hospital, naogaon with the complaints of bending of lower extremities for the last 8 months and difficulty in walking. The informant mother noticed that her boy's leg gradually became bent with walking difficulty during the last 8 months. There is no history of fever or joint pain. On query, mother informed that the child's grandfather also had leg deformity, but her older son and husband has no such

problem. Rahmat was given exclusive breast feeding for 1st 2months and thereafter diluted formula milk for 3months. Then the child was given rice powder, sugar, suji, sagu and only rice and salt. The child did not eat meat, egg yolk, vegetables but occasionally fish. There was no history of taking any offending drug. The boy played at open environment with his peers. Mother's height, weight and body built was average. Rahmat had history of admission at naogaon sadar hospital 3 times during the last 2 years due to diarrhea and respiratory infections. Developmental milestones were age appropriate. On examination the baby was mildly pale, non-icteric, not cyanosed, non-edematous, BCG scar mark present. There is a box shaped head, closed fontanelles and cranial sutures. Her vital signs are within normal limits. Anthropometry measurement showed head circumference 48 cm (-0.83) SD, weight 10.7kg, height 78 cm and WHZ (+0.55 SD). The locomotor system examination revels widening of wrist and ankle joints, bowing of the lower extremities, bulk& tone of the muscles-normal, deep tendon reflex-normal with flexor planter reflex. There is a waddling gait. Higher psychic function could not be evaluated. Cranial nerves examination found normal with intact sensation. All other systems reveal no abnormalities.



Investigation

Haemoblogin 10.7gm/dl, ESR 30 mm 1stHour, TC 8000/cu.mm, S.Alkaline phosphatase 2560U/L, S.Calcium 9.2mg/dl, S. Phosphate 2.85mg/dl, S.Vit D(D_2 + D_3) 13.90 ng/ml , X-Ray of the knee, ankle and wrists joints showed splaying, cupping and fraying of the metaphysis of the long bones with widening of the growth plates resulting mild bowing of the femur and tibia. So, the child is diagnosed as Rickets. Stoss therapy of Vitamin D 400, 000 IU intramuscularly over 1day in 4 divided doses was given. Thereafter, oral Vitamin D 600 IU daily and oral calcium 500mg daily for 3 months was prescribed with advised for follow up visit.

DISCUSSION

Rickets, a disease of growing bone, occurs in children only before fusion of the epiphysis and is due to unmineralized matrix at the growth plates. It was rampant in northern Europe and the United States during the early years of the 20th century. It is a significant public health problem in developing countries also.[4] It primarily affects children under the age of five, a critical period for bone growth and mineralization. In severe cases, rickets leads to skeletal deformities, delayed growth, and, if untreated, permanent disability or even death.[5] The peak age of rickets is 3-18 months.[6] National rickets survey in Bangladesh, 2008 found that prevalence of rickets in Bangladesh is 0.99%.[7] Nutritional rickets has emerged as a public health problem in Bangladesh during the past two decades, with up to 8% of children being clinically affected in some areas[8]. In Pakistan, literature highlighted the prevalence of rickets as 2.25% among children.[9] Data among children under 16 years of age from UK between 2015 to 2017 revealed that yearly incidence of rickets was 0.48 per 100, 000 children.[10] There are many causes of rickets, including vitamin D disorders, calcium deficiency, phosphorus deficiency, and distal renal tubular acidosis. Though it is rare, genetic mutation also plays an important role in the pathogenesis of rickets, e.g., Vit D dependent rickets type1A &IB, Vit D dependent rickets type 2A & 2B, X-Linked hypophostemic rickets, autosomal dominant hypophosphatemic rickets (ADHR), autosomal recessive hypophosphatemic rickets (ARHR) type 1 type 2, hereditary hypophosphatemic rickets with hypercalcuria (HHRH).[4] Despite ample sunlight in many tropical regions including sub-Saharan Africa, nutritional rickets remains a significant public health challenges. In Ethiopia and similar low-resource settings, factors such as poor dietary intake, insufficient sunlight exposure, and inadequate health care contributes to the persistence of rickets.[6] In one study, it was suggested that low dietary calcium intake was important factor in young children while adolescent children Vit D deficiency was mainly responsible for developing rickets.[11] Factors that have been shown to be important in the pathogenesis of nutritional rickets at younger age include maternal Vit D deficiency, living in temperate climate, lack of sun exposure and darkly pigmented skin.[2] Some investigators have suggested that nutritional rickets seen in sunny areas may be caused by calcium deficiency rather than Vit D deficiency and that calcium supplementation alone may be effective for treatment.[12] In our country there is plenty of sunlight throughout the year and therefore the high incidence of rickets is unexpected. The exact etiology of nutritional rickets could not be found due to lack of investigation facilities in our country.[8] Most common presentation of rickets are bowed legs, rachitic rosary, frontal bossing of the skull, widened wrist and ankle joints, poor growth, delayed motor development, recurrent lower respiratory infection, chronic diarrhea and fits.[13] Investigation includes estimation of S.calcium, phosphate, Vit D level, Alkaline phosphatase and parathyroid hormone(PTH) . Depending upon the causes of rickets serum calcium may be normal or low, serum phosphate low, s.alkaline phosphatase increased, serum PTH may be normal or elevated, 25(OH) D low or normal, 1, 25$(OH)_2$ low, normal or high. X-Ray of the wrist, knee and ankle joints show splaying, cupping and fraying of the metaphysis of long bones and widening of the growth plates. Children with nutritional vitamin D deficiency should receive vitamin D and adequate nutritional intake of calcium and phosphorus. There are 2 strategies for administration of vitamin D. With stoss therapy, vitamin D (300, 000-600, 000IU) is administered orally (preferred) or intramuscularly as 2-4 doses over 1 day(vitamin D_3 is preferred to D_2 because of longer half-life of D_3).The alternative strategy is daily vitamin D with a minimum dose of 2, 000 IU/day for a minimum of 3 mo.Either strategy should be followed by daily vitamin D intake of 400 IU/day if <1 yr old or 600 IU/day if >1 yr old. It isimportant to ensure that children receive adequate dietary calcium (minimum of 500 mg/day) and phosphorus; this dietary intake is usually provided by milk formula, and other dairy products, although calcium supplements may be needed in some patients. Rarely,

patients benefit from orthopedic intervention for leg deformities.[4]

Prognosis

Most children with nutritional vitamin D deficiency have an excellent response to treatment, with radiologic healing occurring within a few months. Laboratory test results should also normalize rapidly. Many of the bone malformations improve dramatically, but children with severe disease can have permanent deformities and short stature. Rarely, patients benefit from orthopedic intervention for leg deformities, although this is generally not done until the metabolic bone disease has healed, there is clear evidence that the deformity will not self-resolve, and the deformity is causing functional problems.

Prevention

Most cases of nutritional rickets can be prevented by universal administration of 400 IU of vitamin D to infants <1 yr old. Older children with risk factors for inadequate intake should receive 600 IU/day. Vitamin D may be administered as a component of a multivitamin or as a vitamin D supplement.

CONCLUSION

Nutritional rickets is a preventable bone disease, occurs due to lack of Vitamin D, calcium and phosphorus. So, adequate sunlight exposure, along with ensuring enough dietary calcium and phosphorus intake can prevent most of the cases of rickets. Furthermore, early recognition of signs-symptoms of rickets and timely intervention can minimize its long-term deadly consequences.

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