Renal Rickets, A Severe Form in Children

Sunil Holikar, Kiran Bhaisare and Lawate BB
Department of Pediatrics, MIMSR Medical College, India

Abstract

Worldwide, rickets is the most common form of metabolic bone disease in children. Vitamin D deficiency is the main cause of rickets, though nutritional deficiency of calcium and phosphorous generates the same clinical picture. Many cases are due to poor vitamin D intake or calcium deficient diets and can be corrected by administration of calcium and vitamin D. However, some cases are refractory to vitamin D therapy and are related to renal defects. These include rickets of Renal Tubular Acidosis (RTA), Hypophosphatemic rickets, and Vitamin D Dependent Rickets (VDDR). The latter is due to impaired action of 1α-hydroxylase in renal tubule. These varieties need proper diagnosis and specific treatment.

Case Report

A 15 years male child born of non-consanguineous marriage brought in OPD with complaint of pain in abdomen since 2 days, fever since 2 days and breathlessness since 2 days. Pain was in Rt hypochondriac & supra pubic region, patient had history of similar episodes since the age of 2 years every 3-4 months. At the age of 2 years patient was diagnosed having posterior urethral valve with absent? Ectopic right kidney & left kidney having hydronephrosis. Patient was operated for posterior urethral valve at age of 2 ½ years, he was able to walk at the age of 18 months parents noticed there was thinning of the lower & upper limbs & he had difficulty in walking gradually they found that the lower & upper limbs were bowing, abdomen was lax ,there was bony prominence on the chest. Patient was bed-ridden since age of 5 years (Figure 1).

Keywords: Renal rickets; Vitamin D; Renal tubular acidosis (RTA); Hypophosphatemic rickets

Introduction

Worldwide, rickets is the most common form of metabolic bone disease in children. Vitamin D deficiency is the main cause of rickets, though nutritional deficiency of calcium and phosphorous generates the same clinical picture [1]. Refractory cases include rickets of Renal Tubular Acidosis (RTA), Hypophosphatemic rickets, and Vitamin D Dependent Rickets (VDDR) [2].

Rickets is a disease of growing bone that is due to unmineralized matrix at the growth plates. Vitamin D deficiency is the most common cause of rickets, but any condition that disturbs bone mineralization can cause rickets [3]. Rickets was first reported in the mid-1600s in Europe. Gilson and others described typical findings of bone-deformity with curving of the legs [4]. Rickets a disease of deficiency is still a common finding in the developing countries amongst the under 5 children, peak age of presentation is between the age 1-3 years, It is being contemplated that the basis of rickets in older children may be through another mechanism or the calcium deficiency which was also observed, not purely a nutritional deficiency [5].

In the United States, rickets should be included in the differential diagnosis of children presenting with failure to thrive, developmental delay, and orthopedic abnormalities. Early diagnosis is essential because morbidity can be minimized if children are treated before eight months of age. Nutritional rickets is the main type reported outside the United States, followed by vitamin D-dependent, vitamin D-resistant, and renal rickets [6]. In this case report patient presented with us with severe deformities over the limbs due to rickets secondary to the renal cause.
On Examination

Patient was febrile, heart rate 120 beats /min, respiratory rate 44 breaths /min, acidic breathing was present, bp 110/70 mm of Hg, pallor present, no edema, no icterus, no frontal bossing, no teeth anomalies, no hearing defects, no cataract, no facial dysmorphism, anterior fontanelle closed, bowing of the upper limbs & lower limbs, widened wrist, double malleoli, ricketic rosary & convexity curvature on the lower & upper limbs was present. Anthropometry-height 92 cm (<3rd percentile), weight- 13 kg (<3rd percentile), us: ls 1:1, abdominal wall was lax. Rest systemic examination was normal.

Treatment

Patient was given antibiotic ceftriaxone, sodium bicarbonate, Vitamin D3 6 lac IU 2 doses, patient responded within 7 days of treatment & was discharged & referred to orthopedic surgeon for the corrective measures. Repeat X-ray was done after 2 weeks showed radiological improvement.

Discussion

The clinical presentation of our patients fits with the renal rickets, however response to vitamin D was found improvement radiological but the deformities formed will not be reversible.

Rickets is a childhood disease characterized by impeded growth and deformity of the long bones. Renal tubular acidosis may also interfere with the process of mineralization and cause rickets. Rickets can only occur in the presence of unfused epiphyses as it manifests itself in the growth plate. During the early years of childhood, genu valgum and genu varum are common concerns for parents. These problems represent normal physiologic variations in most children. However, a few children will experience pathologic lower extremity malalignment leading to cosmetic and functional deficits. Although many exist, the most frequent causes of pathologic genu varum and genu valgum are blount’s disease and renal rickets, respectively. As suggested by hensinger [7], genu valgum is typically associated with renal osteodystrophy because the onset of chronic renal disease generally occurs while children are in the valgus phase. Metabolic conditions such as rickets affect the entire epiphyseal plate. Treatment of genu valgum and genu varum includes observation for the lesser deformities, bracing for moderate deformities and surgical correction for the excessive deformities [8].

Present case was diagnosed as bilateral genu recurvatum secondary to renal rickets and referred to orthopedic surgeon for corrective measures. Early administration of vitamin D may prevent such dreadful morbidies in children with renal rickets.

References