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Case Report Open Access

Renal Rickets, A Severe Form in Children

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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.

Patient presented in our institution was bed ridden with renal rickets having very severe deformities. Many cases like this disease and its variants begin in childhood and awareness of the conditions may help to bring patients to treatment earlier& decrease the morbidity.

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.

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).

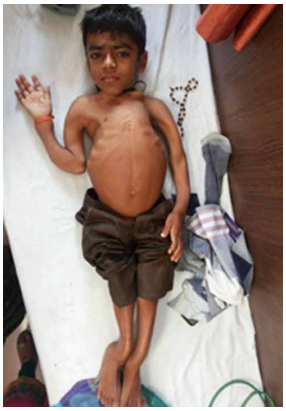

Figure 1: Patient with renal rickets.

On Examination

Patient was febrile, heart rate 120 beats /min, respiratory rate 44 breaths /min, acidotic 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 fontanellae 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.

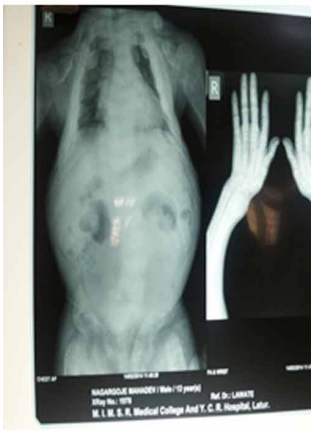

Figure 2: X-ray of patient.

Investigation

X-ray wrist showed findings of florid rickets in the form of cupping at the metaphyseal end lower end of radius and ulna (Figure 2), knee joint & ankle joints , serum alkaline phosphatase was markedly raised (1599 mmol), serum calcium 5.8 mg% , serum phosphorus 3.31 mg%, BUN 31 mg%, serum creatinine 2.6 mg%, sr sodium 138, Sr potassium 5.6, urinary pH was 6.4 (normal 4.5-8), Liver function tests (serum proteins, bilirubin, ALT, AST and prothrombin time) were normal. Hb 10.7 gm%, WBCs 17,100, platelets 3, 24, 000, ABG: pH 7.15, Hco3 15.5. Abdominal ultrasonography showed right kidney absent? Ectopic, left kidney showed hydronephrosis, thinning of cortex.

Treatment

Patient was given antibiotic ceftriaxone, sodium bicarbonate, Vitamin D3 6 lac IU 2 doses, patient responded within 7 days of treatment & was dischared & reffered to orthopedic surgeon for the corrective measures for the deformities. 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.

RTA is a clinical syndrome that causes hyperchloremic metabolic acidosis due to a disorder of urine acidification. The acidification of urine in the distal tubule primarily depends on acid-base exchange transporters in intercalated cells. Most patients with distal RTA have hypokalemia because the inability to excrete H+ is compensated for by increased K+ secretion.

We did not perform a genetic study to identify the mutations accompanying the RTA. The current case also appears to be incomplete distal RTA because the metabolic acidosis was not very severe. An NH4Cl loading test would be required to confirm the diagnosis, but we did not perform it. This could be a limitation of our study. If the distal RTA is accompanied by rickets, calcium, and vitamin D replacement are required, his short stature may be due to another cause or due to insufficiency of the treatment. Some reports have described delayed bone age and decreased growth velocity in distal RTA [3].

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 forthe excessive deformities [8].

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

References

1. Dimitri P, Bishop N (2007) Rickets. Paediatrics and Child Health 17: 279-287.
2. Sahay M, Sahay R (2013) Renal rickets-practical approach. Indian J Endocrinol Metab 17: S35-S44.
3. Lee JH, Park JH, Ha TS, Han HS (2013) Refractory rickets caused by mild distal renal tubular acidosis. Ann Pediatr Endocrinol Metab 18: 152-155.
4. Craviari T, Pettifor JM, Thacher TD, Meisner C, Arnaud J, et al. (2008) Rickets: an overview and future directions, with special reference to Bangladesh. A summary of the Rickets Convergence Group meeting, Dhaka, 26-27 January 2006. J Health Popul Nutr 26: 112-121.
5. Oduwole AO, Giwa OS, Arogundade RA (2010) Relationship between rickets and incomplete distal renal tubular acidosis in children. Ital J Pediatr 36: 54.
6. Nield LS, Mahajan P, Joshi A, Kamat D (2006) Rickets: Not a Disease of the Past. Am Fam Physician 74: 619-626.
7. Hensinger RN (1989) Angular deformities of the lower limbs in children. Iowa Orthop J 9: 16-24.
8. Bahadure RN, Thosar N, Kriplani R, Baliga S, Fulzele P (2012) Dental Aspect of Distal Tubular Renal Acidosis with Genu Valgum Secondary to Rickets: A Case Report. Case Rep Dent 2012: 1-4.