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Original Research Article

Nutritional rickets among children of Northern Kerala, India

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ABSTRACT

Background: Vitamin D deficiency is a very common micronutrient deficiency in our country and ranks among the 5 most common disease in children worldwide.
Methods: To study the presentation and predisposing factors of rickets among children in Northern Kerala.
Results: This study was conducted in the department of Pediatrics in a tertiary care hospital in Northern Kerala. Nutritional rickets diagnosis was based on clinical, radiological and biochemical parameters and appearance of provisional zone of calcification after vitamin D therapy. Data was analyzed and presented as percentage and mean. Student t-test was used for statistical analysis of the results.
Conclusions: A total of 54 children having a variety of clinical presentations of rickets were seen during the study period. There were 46.3% males and 53.7% females. The mean age of diagnosis was 24 months.70.4% children were exclusively breast fed up to 6 months. None of the breast-fed infants had received vitamin D supplementation according to current guidelines. 66.7% children were not exposed to sunlight. Rickets was commonly found in children with no protein energy malnutrition. Genu varum and wrist widening were the most frequent clinical features observed in 92.6% patients. The radiological and biochemical characteristics of cases were ascertained before and after therapy. The biochemical changes were analyzed by student (t) test. There was a significant decrease in the ALP activity after vitamin D therapy (P= 0.000). Out of these 54 children with rickets 47(87.03%) responded to treatment with vitamin D3 and 7 (12.96%) were resistant to treatment with vitamin D3.

Keywords: Biochemical, Clinical, Nutritional rickets, Radiological profile, Vitamin D

INTRODUCTION

Vitamin D deficiency is a very common micronutrient deficiency in our country and ranks among the 5 most common disease in children worldwide.[1,2] Rickets is a disease of growing bone characterized by deficient mineralization of bone matrix.[3] Though rickets was known for centuries, it is essentially a modern disease which peaked in the industrial age. Francis Glisson gave the first detailed description of the disease early in the seventeenth century.[4]

Early diagnosis of rickets is difficult because the initial symptoms are unremarkable. Children developing rickets are often pale, irritable, and sleepless and perspire profusely.[5] It is manifested by widening (flaring) of metaphysis of long bones, prominence of costochondral junction (rachitic rosary), flaring of lower anterior thoracic wall and frontal bossing. After the child begins to walk and bear weight, genu valgum or genu varum develops. Systemic manifestations include muscular weakness, anorexia and increased susceptibility to infection.[3]

Incidence of rickets has increased recently in normal population due to changes in life habits. Lack of sunlight exposure, inappropriate dietary intake and poor housing would contribute to the development of rickets.[6,7] Early detection, proper treatment and health education can prevent rickets and its long-term effects on growth. This study was aimed to know:

The epidemiological and demographic factors of rickets, various clinical manifestations and biochemical characteristics of the disease. Percentage of cases responding to adequate dose of vitamin D and percentage of cases not responding to adequate recommended dose of vitamin D.

METHODS

This study was conducted in a tertiary care centre in Northern Kerala for a period of 18 months. Infants and children attending OPD or getting admitted in pediatric wards for various diseases who were having rickets were included in this study.

Patients having features of rickets on clinical ground were thoroughly examined and detailed history taken. They were investigated radiologically as well as biochemically. If there was any evidence of rickets radiologically, then blood was tested for calcium, phosphorous and alkaline phosphatase in all the patients to confirm the diagnosis. Reference values for serum levels of calcium, phosphorous and alkaline phosphatase were taken from Nelson text book of pediatrics.[8]

Rickets was confirmed by presence of clinical features, characteristic x-rays changes including splaying, fraying and cupping at the lower end of radius and ulna and sometimes at the lower end of tibia and fibula and biochemically. Biochemical parameters suggestive of rickets are elevated serum alkaline phosphatase (>500 IU/L), low serum phosphorous (normal value 1-3 years: 3.8 to 6.5mg/dl and 4-11 years: 3.7 to 5.6mg/dl) and normal or low serum calcium (<8.8mg/dl).

All confirmed cases of rickets were treated with single intramuscular injection of vitamin D (6,00,000 units) "stoss therapy" followed by oral calcium in a dose of 50 mg/kg/day. All were given advice regarding nutrition and exposure to sunlight. At 6 weeks follow-up, again X-ray was repeated, and blood was tested for calcium, phosphorous and alkaline phosphatase. Healing was considered with appearance of calcification line on X ray and improvement in biochemical profile. If the zone of provisional calcification (sign of healing) was absent it was diagnosed as a case of non-nutritional rickets. Children who were already diagnosed cases of nutritional rickets and receiving treatment and rickets like state like metaphyseal dysplasia, Blount's disease, hypophosphatasia were excluded from the study. Follow up at sixth month and 1 year was done to look for changes in skeletal deformities.

Statistical analysis was done on package of SPSS; version-16 and the distribution of cases among various criteria were presented by their percentage and mean. The biochemical changes were analyzed by student (t) test. P value less than 0.05 was considered statistically significant.

RESULTS

A total of 54 children were enrolled in the study which include 25 (46.3%) males and 29 (53.7%) females. Male to female ratio was1:1.2. Majority of children were less than 2 years age group 24 (44.4%). Mean age of presentation was 24 months.

Socioeconomic status was assessed according to kuppusamy scale. 87.8% belonged to the upper lower and lower middle classes. Family history of rickets was present in 12 (22.2%) children.

36 (73.5%) children presented with skeletal deformity, 9 (18.4%) children with intercurrent infection and 4 (8.1%) children with history of frequent fall. As regards nutritional status, 27 (50%) children were having normal nutritional status whereas only 9.3% were severely malnourished. 20 (37%) patients were anemic in our study. Among these 54 children, 49 (90.7%) were born term and 43 (79.6%) had adequate birth weight. 16 (29.6%) children were exclusively breast fed for less than 4 months and 38 (70.4%) for 6 months. 57.4% children were given ragi as weaning food, 38.9% were given banana and rest (3.7%) were given rice. None of the patients received vitamin D supplements. Only 18 (33.3%) children were exposed to sunlight half an hour at least thrice a week.

Most frequent clinical signs included genu varum 50 (92.6%), wrist widening 50 (92.6%) and double malleolus 49 (90.7%). Other features noticed in decreasing order of frequency were rachitic rosary 36 (66.7%), pectus carinatum 34 (63%), frontal bossing 32 (59.3%) and Harrison's sulcus 28 (51.9%). Wide anterior fontanella was noticed only in 2 (3.7%) patients. No case presented with Hypocalcemic seizure. The frequency of physical findings in children with rickets is displayed in Figure 1.

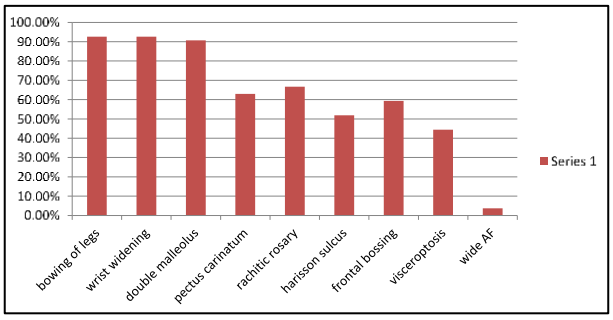

Figure 1: Relative frequency of different clinical presentation of rachitic children.

Table 1: The age at presentation.

Age(years)	Male	Female	Total
<2	13	11	24 (44.4%)
2-3	9	14	23 (42.6%)
>3-4	2	1	3 (5.5%)
>4	2	2	4 (7.4%)

Biochemical profile showed that serum calcium was normal in 35 (64.8%) patients at diagnosis. The serums inorganic phosphate was low in 23 (42.6%) patients while in 29 (53.7%) patients it was normal. Both serum calcium and serum phosphorous level were normal in 19 (35%) children.

In 14 (25.9%) patient's serum alkaline phosphatase was above 1000IU/L, followed by 500-1000 in 29 (53.7%) patients and below 500 for 11 (20.4%) patients. Radiological findings suggestive of rickets were found in all patients.

Table 2: Biochemical values at the time of diagnosis.

Biochemical parameter	Mean	95% CI	Reference value
Calcium (mg/dL)	9.04±0.876	7.32-10.76	8.8-10.8mg/dL
Phosphorous	4.01±1.161	1.74-6.29	1-3 years: 3.8-6.5, 4-11 years: 3.7-5.6 mg/dL
Alkaline phosphatase	853.67±556.883	237.8-1945.16	<500IU/L

In 54 rickety patients it was tried to see the response of the patients to single intramuscular injection of vitamin D3 60,000 units (Stoss therapy) by regular monthly follow up. Table 3 presents the biochemical values after vitamin D treatment. Alkaline phosphatase level decreased significantly after treatment (p value 0.000). It was found that 47 (87.03%) patients showed excellent response to single intramuscular injection of vitamin D3 and labeled them as having nutritional rickets. 7 (12.96%) patients showed no response to treatment as evidence radiologically by no sign of healing in X ray.

Table 3: Biochemical parameters after treatment.

Biochemical parameter	Mean	*ρ* value
Calcium	9.07±0.624	0.773
Phosphorous	4.16±0.881	0.395
Alkaline Phosphatase	391.69±254.718	0.000

Also, there was any change in serum concentrations of calcium, phosphorous and alkaline phosphatase. They were labeled as having non-nutritional rickets. 6 patients were lost to follow-up. Clinical improvement was observed between 6 months to one year of therapy. In majority of patients widening of wrist resolved and in all patient's double malleolus disappeared.

DISCUSSION

Present clinical presentation was in line with the findings of a study conducted in Nigeria9 where the commonest mode of presentation was lower limb deformity (82.2%). In the present study 15 (27.8%) cases had delayed motor mile stone which is also well reported.[10] In addition to defective skeletal maturation vitamin D deficiency causes severe muscle weakness and muscle aches and pains, which can have devastating consequences to the child health. Similarly, 18.4% cases had recurrent intercurrent infections. Yener et al have reported more episodes of bacterial infections in children with vitamin D deficiency as compared to healthy children.[11] In fact T and B lymphocytes have receptors for 1,25(OH)2D. Vitamin D deficiency may predispose to different bacterial infections including pneumonia.[12] 1,25(OH)2D 3 plays an important role in immune modulation.[11]

It was noted that most of the children 38(70.4%) were exclusively breast fed up to six months and none of them received vitamin D supplements, these results are comparable with other studies.[13,14]. Breast milk is indisputably the ideal food for infants; however, breast milk typically contains about 25 IU/L which is insufficient for rickets prevention.[15,16] Although there is evidence that limited sun exposure may prevent rickets in some breast-fed infants concerns over the health risk of sun exposure had led to the recommendation that all breast-fed infants receive supplemental vitamin D.[17,18]

Thacher et al reported that children with rickets had a greater proportion of first degree relatives with a history of rickets.[19] A similar diet and environment may be the reason for rickets in families. Present study supports this view.

Rickets is usually reported in well-nourished children, but it also associated with severe under nutrition and poverty.[20] A study from Pakistan reported that 87% had normal nutritional status and only 5% were severely malnourished which is in agreement with the present study.[21] Regarding sunlight exposure similar results was obtained from many studies which showed that lack of

sunlight exposure increases the risk of rickets (Majeed et al, Mastsuo et al; Molla et al).[22-24]

Our clinical findings were comparable to study carried out by Onyiriuka et al were lower limb deformity was commonest clinical feature followed by wrist widening.[18] Another study from Karachi reported that most common clinical presentation was rachitic rosary (87%) followed by wrist widening (76%).[14]

Biochemical results of the present study are comparable to study carried out by Bouk et al, were 84% patients had raised alkaline phosphatase.[20] A study from Karachi reported that raised alkaline phosphatase levels were found in 100% cases whereas serum calcium was low in 90% cases.[21] Serum phosphate was either low or normal in 44% cases. The biochemical results of this study suggest that serum alkaline phosphatase level is a good screening test for rickets. Previously some experts have recommended measuring alkaline phosphatase levels as a screening test for rickets.[25]

Radiological findings in our study were comparable to study done by Bouk et al where X-ray finding was present in all patients.[20] X ray is very useful for early diagnosis as radiological abnormalities can be found before physical signs. There was no significant change in serum calcium and phosphorous after treatment. At the same time alkaline phosphatase level decreased significantly. This showed that alkaline phosphatase is the most sensitive biochemical criterion for healing.[26]

In the present study 47 (87.03%) patients had nutritional rickets and 7 (12.96%) had non-nutritional rickets. Khattak et al in his study showed that 91% children had nutritional rickets and 9% had non-nutritional rickets which was comparable to our study.[4] One of the major challenges of management was the high rate of default to follow up. Clinical improvement was observed between 6 months to one year of therapy. This finding is in agreement with those obtained by Onyiriuka et al were healing was observed between 3 to 5 months of treatment depending on the severity of the cases.[9]

We presume that the identified cases of nutritional rickets were predominantly vitamin D deficiency rickets, since 70.4% of our patients were exclusively breast fed for 6 months and 57.4% were weaned with ragi which is a calcium rich diet. Major limitation of our study was assay of vitamin D levels were not done in these patients to diagnose and assess the severity of the disease due to lack of financial resources.

CONCLUSION

Clinical, radiological and biochemical markers are reliable indicators for diagnosis of nutritional rickets. Serum alkaline phosphatase is the most reliable biochemical investigation for diagnosis and follow up of rickets. Radiological findings were present in all patients with rickets. It is almost certain that the clinically apparent cases of nutritional rickets represent the tip of the iceberg. Future challenge will be to prevent nutritional rickets by encouraging parents to follow vitamin D supplementation guidelines.

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