Risk Factors for Nutritional Rickets in Children of Northern Kerala

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Abstract:
Objective: To assess the risk factors causing nutritional rickets among children in our part of world.

Subjects And Methods: 47 children with rickets and 47 control children matched for age and sex were recruited over a 18 month period in a tertiary care hospital in northern kerala. Diagnosis was based on clinical, radiological, biochemical parameters and response to treatment. Children who presented in our OPD with non nutritional illness were used as control. A specially designed questionnaire was administered by one of the investigators to both mothers of patients and control subjects to assess the role of social, nutritional and other related factors in the pathogenesis of nutritional rickets.

Results: There was no significant difference between cases and controls for prematurity, birth weight, birth order, breast feeding and weaning practices. 68% rickets patients did not have significant sun exposure but this was not statistically significant (p value 0.69). None of the children in study and control group received vitamin D supplementation according to current guidelines. The study group had significantly higher percentage of first degree relatives with rickets than controls (21% v/s 4%; p value 0.01).

Conclusion: Nutritional rickets is multifactorial condition. We presume that most important predisposing factor for nutritional rickets in our area is gestational vitamin D deficiency.

Keywords: Nutritional rickets, risk factors, vitamin D

I. Introduction
Rickets is the term signifying failure in mineralization of growing bone or osteoid tissue. There are many causes of rickets; among them nutritional vitamin D deficiency remains the most common cause globally. A severe vitamin D deficiency impairs mineralization of bone tissue (causing osteomalacia) and of growth plates (manifesting as rickets)².

Recent data indicates that vitamin D deficiency is pandemic, even the healthy and the young are not spared. High prevalence rates are reported in otherwise healthy infants, children and adolescents², and also from diverse countries around the world including India³.

Despite having adequate sunlight throughout the year, a substantial number of children suffer from this preventable disease. Several factors, such as inadequate exposure of infants to sunlight, exclusive breast feeding, darker skin, poor housing, fully covered dressing style of mothers and multiparty have been implicated. The purpose of this study was to determine the role of different possible risk factors causing nutritional rickets among the children in our part of world.

II. Subjects And Methods
47 children with nutritional rickets and 47 control children matched for age and gender were included. Diagnosis was made on clinical, radiological and biochemical parameters. Children responding to single intramuscular injection of vitamin D (6lakhs IU) were diagnosed to have nutritional rickets. A specially designed questionnaire was administered to both mother of patients and control subjects to assess the role of social, nutritional and other related factors in the pathogenesis of nutritional rickets. Control group were children who presented in our OPD with non nutritional illness.

The clinical criteria considered for the diagnosis of rickets were deformity of the lower limbs, wrist widening and rickety rosary. Serum levels of following biochemical parameters were determined: hemoglobin, serum calcium, serum phosphorous, alkaline phosphatase. Rickets was diagnosed by radiographic signs at the wrist or knee. After getting informed consent details of the medical history, clinical and lab data were recorded on specially designed forms. The medical history included gestational period, birth weight, birth order, developmental aspects, illnesses and treatment received etc. Special emphasis was given to recording the frequency and duration of exposure of the child to sunlight. The detailed nutritional history of the child included the duration of breast feeding, weaning age and type of food given. Skin color was determined by the pediatrician’s subjective assessment. We did thorough physical examination, including measurement of weight, height and head circumference.
The statistical analysis of the study was conducted using SPSS 16.0. Chi-square was used to identify the significance of the relations, associations and interactions among various variables. Odd ratios (OR) were applied to explore the magnitude of the difference between cases and controls variables of our concern. The result was accepted as statistically significant when the p value was less than <0.05.

III. Results

Data of 47 rickets patients and 47 control subjects were included for analysis. Table 1 shows comparison of social, nutritional and general features between control and rickets group.

Table 1. Relation of various study variables in children with rickets and control

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Case(47)</th>
<th>Control(47)</th>
<th>OR(95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm</td>
<td>5(10.6%)</td>
<td>7(14.8%)</td>
<td>0.68(0.2-2.3)</td>
<td>0.37</td>
</tr>
<tr>
<td>LBW</td>
<td>10(21.1%)</td>
<td>17(36%)</td>
<td>0.47(0.1-1.1)</td>
<td>0.08</td>
</tr>
<tr>
<td>Birth order (≥3)</td>
<td>8(17%)</td>
<td>11(23.4%)</td>
<td>0.67(0.24-1.8)</td>
<td>0.3</td>
</tr>
<tr>
<td>Associated Disease</td>
<td>7(14.9%)</td>
<td>44(93.6%)</td>
<td>0.01(0-0.04)</td>
<td>0.00</td>
</tr>
<tr>
<td>Weaning age (4-6/12)</td>
<td>35(74.4%)</td>
<td>31(65.9%)</td>
<td>0.6(0.2-1.6)</td>
<td>0.25</td>
</tr>
<tr>
<td>Weaning food (cereal)</td>
<td>28(59.5%)</td>
<td>24(51%)</td>
<td>0.7(0.3-1.6)</td>
<td>0.26</td>
</tr>
<tr>
<td>Long term medicine</td>
<td>5(10.6%)</td>
<td>5(10.6%)</td>
<td>1(0.26-3.7)</td>
<td>0.63</td>
</tr>
<tr>
<td>Gross motor delay</td>
<td>13(27.7%)</td>
<td>4(8.5%)</td>
<td>4(1.2-13.7)</td>
<td>0.015</td>
</tr>
<tr>
<td>Consanguinity</td>
<td>3(6.3%)</td>
<td>2(4.2%)</td>
<td>1.50(0.2-9.6)</td>
<td>0.5</td>
</tr>
<tr>
<td>Family history</td>
<td>10(21.3%)</td>
<td>2(4.2%)</td>
<td>6(1.2-29.5)</td>
<td>0.014</td>
</tr>
<tr>
<td>Sun exposure</td>
<td>15(31.9%)</td>
<td>19(40.4%)</td>
<td>0.69(0.2-1.6)</td>
<td>0.69</td>
</tr>
<tr>
<td>Complexion (dark)</td>
<td>2(4.2%)</td>
<td>0</td>
<td>2(1.6-2.5)</td>
<td>0.24</td>
</tr>
<tr>
<td>Normal HC</td>
<td>38(80.9%)</td>
<td>42(89.4%)</td>
<td>0.5(0.15-1.6)</td>
<td>0.19</td>
</tr>
<tr>
<td>Severe (gd 3) stunting</td>
<td>8(17%)</td>
<td>0</td>
<td>0.45(0.36-0.57)</td>
<td>0.003</td>
</tr>
<tr>
<td>PEM (gd 3,4)</td>
<td>3(6.3%)</td>
<td>0</td>
<td>0.48(0.3-0.5)</td>
<td>0.12</td>
</tr>
<tr>
<td>Hb&lt;11</td>
<td>17(36.2%)</td>
<td>29(61.7%)</td>
<td>0.39(0.15-0.81)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

21% patients had family history of rickets which was statistically significant (OR 6(1.2-29.5) and p value 0.014). There was no significant difference between cases and controls for social and nutritional factors. None of the children in both groups had received vitamin D supplementation according to current guidelines. Significant gross motor delay was noted in rickets patients when compared to controls (OR 4.1 and p value 0.015) Comparing nutritional status of children in both groups, 17% rickets patients had severe stunting which was statistically significant (p value 0.003).

IV. Discussion

The etiopathogenesis of rickets is thought to be multicausal. This study is an attempt to assess various risk factors contributed to the disease.

The incidence of rickets was 32% in premature infant with birth weight below 1.5kg. Rickets in preterm infant has decreased with improvement in care and nutrition and our study supports this view.

The primary source of vitamin D for a newborn baby is the vitamin D that passes transplacentally to the baby from the mother in the intrauterine period. Currently, vitamin D intake during pregnancy and breastfeeding has been reported to be insufficient throughout the world. In a study done in Turkey maternal serum 25(OH)D3 concentrations were not significantly related to the number of pregnancies or births. Similarly in our study no statistically significant difference was found in the incidence of rickets and birth order. Concerning infant feeding practices no significant differences between rickets and non-rickets children were found. While in many studies prolonged breastfeeding and lack of good quality weaning food were reported as risk factors.

The main source of vitamin D is cutaneous synthesis after exposure to ultraviolet B rays and several studies support this view. 68% of our children in the study group were not getting sunlight exposure even half an hour at least thrice a week. But this was not statistically significant. Thecher et al reported that children with rickets had a greater proportion of first degree relatives with a history of rickets and our study support this view.

Regarding association between nutritional rickets and different predisposing factors, it was observed from our results that postnatal factors play not much significant role in the etiopathogenesis of rickets. The most important predisposing factor is presumed to be gestational vitamin D deficiency. Unfortunately we didn’t take a detailed maternal history. While it was beyond the scope of this paper to measure vitamin D levels of both mothers and children involved in both groups. It is still important to acknowledge that this may have played a role in the high prevalence of nutritional rickets cases.

There is an urgent need for heightened awareness among health care providers and general public about the importance of vitamin D. Attention to vitamin D status during pregnancy and lactation and implementation of current recommendation in our area is warranted.
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Reference