PRESENTATION AND PREDISPOSING FACTORS OF NUTRITIONAL RICKETS IN CHILDREN OF HAZARA DIVISION

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Background: There is no local data about rickets available in Hazara Division, while clinical experience suggests that problem exists in this area with abundant sunlight. We carried out this study with an objective to determine presence, presentation and predisposing factors of rickets in pediatrics population of Hazara Division. **Methods:** This study was conducted in Department of Pediatrics, Ayub Teaching Hospital Abbottabad over a twelve months period from July 2003 to July 2004. Children from newborns to fifteen years of age presenting with signs and symptoms of rickets were included and information regarding signs, symptoms, predisposing factors (crowded housing, isolated housing with deficient sun exposure, abundant sun but lack of awareness, malnutrition and antenatal factors) and investigations was recorded on a proforma. Diagnosis was based on clinical signs, radiological changes on x-ray wrist joint and biochemical disturbances in serum levels of alkaline phosphatase, calcium, and inorganic phosphorus. **Results:** Sixty Children with rickets reported during the study period. The main clinical presentation was in the form of delayed motor milestones in 20 (33.33%) children, recurrent lower respiratory infections in 11 (18.33%) children, recurrent diarrhea in 12 (20%) children, and fits in 3 (5%) children. Skeletal changes on clinical examination were present in 40 (66.6%) children. Radiological signs of rickets were present in 51 (85%) children. Symptoms and signs reverted to normal in all cases after vitamin D supplementation. The apparent risk factors were lack of awareness, malnourishment and antenatal factors. **Conclusion:** Rickets is common in Hazara Division presenting with variable signs and symptoms, predisposing the childhood population to different illnesses and skeletal deformities. In the presence of abundant sunshine lack of awareness of exposure to sun, malnutrition and antenatal factors may be the important predisposing factors for development of nutritional rickets.

Key words: Rickets, vitamin D deficiency, and malnutrition.

INTRODUCTION

Nutritional rickets is a disorder of growing children due to defective mineralization of newly formed bone matrix because of vitamin D deficiency. An English Physician Dr.Daniel Whistler first described rickets in 1645.[1] In 1861, Trousseau of France linked rickets with lack of sun exposure and faulty diet.[2] At the start of 20th century McCollum discovered vitamin D by the technique of biological analysis of the food.[3] The main source of vitamin D is cutaneous synthesis when 7-dehydrocholestrol in the skin is converted to cholechalciferol (vitamin - D_3) by the sun ultraviolet – B radiations. A number of factors can impair cutaneous synthesis of vitamin D for example decreased sun exposure,[3] increased skin pigmentation[3,4] and atmospheric pollution.[5] Small amount of vitamin D is also derived from the diet.[6] High intake of the fibers and phytate also decrease intestinal absorption of vitamin D and calcium.[7] Rickets may be subclinical[8] or present with the clinical signs and symptoms, for example, bowed legs, rickety rosary, frontal bossing of the skull, widened wrist and ankle joints, poor growth, delayed motor development[9], recurrent lower respiratory infections, chronic diarrhea and fits.[10] Radiological findings in rickets include metaphyseal flaring, irregularity and widening of physis.[11]

Hazara Division is an area of Pakistan with ample sunlight through out the year. However clinical experience shows that rickets is present in this area. Here we are reporting an analysis of rickets cases presenting in a calendar year

MATERIAL AND METHODS

The study was carried out in the Department of Pediatrics, Ayub Teaching Hospital Abbottbad over a 12 months period from July 2003 to July 2004. All infants and children (indoor and outdoor) presenting with signs and symptoms suggestive of rickets were enrolled in study and information was recorded on a proforma prepared for this purpose. Detailed history from the mother was taken regarding sun exposure, weaning, housing, developmental history, and repeated illnesses in the form of respiratory infections and diarrhea. Nutritional assessment was done according to Gomez classification[12] (table-1). Children with hepatic and renal diseases or on anticonvulsant medicines were excluded from the study. Radiographs of the wrist, serum alkaline phosphatase, serum calcium, and serum inorganic phosphorus were the initial investigations at the time

of enrollment. Other laboratory investigations included full blood count, liver function test, and renal function test.

Table-1: Gomez classification of malnutrition[12]

Grades of malnutrition	Weight for age
Normal	>90%
Mild	75-89%
Moderate	60-74%
Severe	<60%

RESULTS

During the study period **s**ixty children with rickets were detected which included 47 (78.33%) male and 13 (21.66%) females. Numbers of the cases under twelve months of age were 38 (63.33%) while the distribution of the rest is shown in table-2. Clinical presentation in these children is shown in tables 3 and 4 while table-5 shows the radiological findings.

Malnutrition was present in 24 (40%) children. All children in this study were from sunnier climes. Housing in disadvantageous position for sunrays was predisposing factor in 15 (25%) cases. In 40 (66.66%) children weaning was either not started, delayed, or inadequate. In 51 (85%) children radiological evidence of rickets (metaphyseal splaying/fraying, cupping at the lower ends of radius and ulna, and decreased bone density) was present. X-ray wrist showed delayed bone age in 3 (5%) cases absent ossification centers in 9 (15%) infants more than six months old.

Table-2: Age distribution

Age in months	No.	%
6-12	38	63.33
13- 24	10	16.66
25- 36	4	6.66
37- 48	3	5
49- 60	2	3.33
> 60	3	5

Table-3: Clinical presentations

Symptoms	No	%
Repeated lower respiratory tract infections	11	18.33
Delayed motor milestones	20	33.33
Recurrent diarrheas	12	20
Seizures	3	5

Table: 4 Clinical signs of rickets

Clinical signs	No	%
Wide wrists	37	61.66
Rickety rosary	22	36.66
Bow legs	5	8.33
Craniotabes	5	8.33

Table: 5 Radiological findings on x-ray wrist

Radiological signs	No	%
Cupping	27	45
Metaphyseal splaying/fraying	30	50
Delayed bone age	3	5
Absent ossification centers	9	15

DISCUSSION

Vitamin D deficiency is a common problem in Pakistan as highlighted in different studies.[5,10] In developed countries resurgence of rickets is noted among the immigrants.[13,14] Mughal et al[14] describe six cases of rickets from 1995-97, while in Copenhagen 5 cases/year of rickets were described over a 10 year period.[15] High incidence of sub clinical rickets (without clinical signs) in immigrant children has been reported in Denmark (46% in 0-8 years and 81% in 9-16 years)[13]. Presentation of rickets in present study seems to be the tip of the iceberg as 60 cases with apparent signs of rickets were documented in a 12 months period in a single tertiary care teaching hospital.

In present study 20 (20%) cases presented with delayed motor milestones. 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.[16] Similarly 18.33% cases, had recurrent lower respiratory tract infections. High frequency of pneumonia has been reported in multiple national and international articles.[10,17,18] In fact T and B lymphocytes have receptors for $1,25(OH)_2$ D. Vitamin D deficiency may predispose to different bacterial infections including pneumonia.[19] 1-25 $(OH)_2$ D_3 plays important role in immune modulation.[20]. Yener et al have reported more episodes of bacterial infections in children with vitamin D deficiency as compared to healthy children[20]. Recurrent diarrhea was presenting complaint in 12 (20%) cases. Diarrhea is common in developing countries like Pakistan due to poor hygiene, non-availability of clean water and malnutrition. In present study, symptoms improved with vitamin D supplementation. Hameed et al[10], Jamal et al[5] and Khattak et al[17] have also reported association of rickets with recurrent or chronic diarrhea. Non-febrile fits were present in 3 (5%) cases. In present study all children presenting with fits were less than 24 months. Hypocalcaemia is more common in initial stages of nutritional rickets, especially during rapid growth period.[9] Hameed et al have reported 10 % of children with non-febrile fits suffering from nutritional rickets. [10]

In different studies multistory buildings and heavy atmospheric pollution of metropolis

obstructing sunrays have been mentioned as a risk factor for vitamin D deficiency rickets.[5, 10] In present study all the children were from sunnier clime of hilly areas. In 25% of the cases housing was at a disadvantageous position for sunshine. Other studies have also reported prevalence of rickets in sunnier climes.[21-23] Dark skin complexions may be the predisposing factor in present study (75% children had dark complexions). Most cases of nutritional rickets in United Kingdom have been reported in children of Black or Asian origin.[24] Children with dark skin are as much capable of synthesizing vitamin D, but to do so require greater exposure to the ultraviolet rays.[25] In 15 (25%) children skin complexions were fair. These children belonged to hilly areas of district Kohistan and Mansehra facing extremes of cold. In these areas wood is used to generate the heat, with formation of huge smoke that hides the fair skin complexion and because of danger of pneumonia parents are reluctant to give bath to their children. Heavy clothing obstructing the ultraviolet rays is also a risk factor.[10]

In a study conducted in slum areas of Karachi 99% of the children with nutritional rickets were malnourished.[5] In present study 24 (40%) children were underweight according to Gomez classification of malnutrition. Malnutrition has been reported in literature as a contributing factor for nutritional rickets.[26]

In this study 14 (23.33%) children were below 6 months of age while 38(68.33%) children were below 13 months. Poor maternal vitamin D status during pregnancy, in addition to other factors, may be important risk factor in these babies.[27] Thick dark veils and habit of indoor staying contributes to vitamin D deficiency in pregnant and lactating mothers.[10]

The clinical signs of the rickets (widened wrists, rickety rosary of costochondral junctions, and bowing of the legs) were present in 40 (66.66%) children while Hameed et al have reported these clinical signs in 70% of the children with nutritional rickets.[10]

In present study 51(85%) children had radiological findings of rickets, while these findings were detected in 73.84% [9], 38.09% [23], and 100% [11] in different studies.

In 3(5%) children bone age was delayed ((Fig-1), while no ossification center was present in wrist joint x-ray in 9(15%) children (age more than six months) (Fig-2). Glerup et al have reported delayed bone age in 11.76% of the children with nutritional rickets.[13]

Figure-1: Delayed Bone age in a 5 years old child with rickets. Only 3 ossification centers are present at carpal bones on X-ray left wrist joint

Figure-2: No ossification center is visible at carpal bones in X-ray of left wrist joint is this 7 months old infant

CONCLUSION

Non-infectious rickets remains an important preventable illness, which predisposes to infectious diseases like bacterial pneumonias, recurrent diarrheas and bone deformities in cases of late presentation. Nutritional rickets in sunny clime of Hazara Division presenting with multiple clinical presentation is predisposing factor for different childhood illnesses. Lack of awareness of importance of sun exposure, malnutrition and antenatal factors were found to be the predisposing factors for nutritional rickets.

REFERENCES

1. Cone TE Jr. A rachitic infant painted by Burgkmair 136 year before Dr. Whistler described rickets. Clin Pediatr (Phil) 1980;19:194.
2. Dunn PM. Professor armed trousseau (1801-67) and the treatment of rickets. Arch Dis Child Fetal Neonatal ED1999;80:F155-F157
3. Rajakumar M. Vitamin D, Cod –Liver Oil, Sunlight, and Rickets: A historical Perspective. Pediatrics 2003;112(2):el32-el35
4. Clemens TL, Adams JS, Henderson SL, Holick MF.Increased skin pigment reduces the capacity of skin to synthesize vitamin D3. Lancet 1982;I:74-76
5. Jamal A, Khanani AR, Biloo G, Asghar A, Jafri Z.Rickets in a slum of Karachi. Pakistan's J Med Sci 1996;12(3): 247-50
6. Crocombe S, Mughal MZ, Berry JL.Symptomatic vitamin D deficiency among non-Caucasian adolescents living in

the United Kingdom. Archives of diseases in childhood 2004; 89:197-9
7. Pettifor JM, Daniels ED. Vitamin D deficiency and nutritional rickets in children. In: Feldman D, Glorious FH, Pike JW, Eds. VitaminD. San Diego, Calif:Academic Press;1997.p.663-78
8. Spene JT, Serwint JR. Secondary prevention of Vitamin D-Deficiency Rickets. Pediatrics 2004; 113(1) 70-2
9. Ladhani S, Srinivasan L, Buchanan C, Allgrove J. Presentation of vitamin D deficiency. Archives of disease in childhood 2004; 89:781-84
10. Hameed A, Ahmad S, Rehman S, Urakzai AA, Gandapoor AJ.A study of rickets-Morbidity and aetiology of a Low Profile Disorder. J Post Med Inst 1998;12(2):14-21
11. Kriter SR, Schwartz RP, Kirkman HN, Charlton PA, Calikoglu AS, Davenport ML. Nutritional rickets in Africans-American breastfed infants Pediatr 2000; 137:153-7
12. Gomez F, Galvan RR, Frank S. Mortality in second and third degree malnutrition. Trop Pediatr 1956;2:77.
13. Gerup H, Rytter L, Mortenson L, Nathan E. Vitamin D deficiency among immigrant children in Denmark. Eur J Pediatr 2004;163:272-73
14. Mughal MZ, Salama H, Greenaway T, Laing I, Mawer EB.Florid rickets associated with prolonged breast-feeding without vitamin D supplementation. BMJ 1999;318:39-40
15. Pedersen P, Michaelsen KF, Molgard C. Children with nutritional rickets referred to hospitals in Copenhagen during a 10-year period Acta Pediatr Scand 2003; 92:87-90
16. Holick MF. Vitamin D: under appreciated D-lightful hormone that is important for skeletal and cellular health. Current Opinion in Endocrinology and Diabetes 2002;9:87-98
17. Khattak AA, Rehman G, Shah FU, Khan MK. Study of rickets in admitted patients at Lady Reading Hospital Peshawar. J Post Med Inst 2004; 18:52-8
18. Najada AS, Habashneh MS, Khader M .The frequency of nutritional rickets among hospitalized infants and its relation to respiratory diseases. J Trop Pediatr 2004; 50:364-8
19. McGrath J. Does “imprinting” with low prenatal vitamin D contribute to the risk of various adult disorders. Med Hypothesis 2001; 56:367-71
20. Yener E, Coker C, Cura A, Keskinoglu, Mir S.Lymphocytes subpopulation in children with vitamin D deficient rickets. Acta Pediatr Jpn 1995; 37(4): 500-2
21. Garabedian M, Ben-Mekhbi H.Deficiency rickets: The current situation in France and Algeria. Pediatric 1989; 44:259-64
22. Lulseged S. Severe rickets in a children’s hospital in Addis Ababa.Ethiop Med J 1990; 28:175-81
23. Narchi H, EI Jamil M, Kulaylat N. symptomatic rickets in adolescence. Arch Dis Child 2001;84:501-3
24. Allgrove J. Is nutritional rickets returning? Archives of diseases in childhood 2004; 89:699-701
25. Lo CW, Paris PW, Holick MF. Indian and Pakistani immigrants have the same capacity as Caucasian to produce vitamin D in response to ultraviolet irradiation. Am J Clin Nutr 1986;44:683-5.
26. Salimpur R. Rickets in Tehran. Study of 200 cases. Arch Dis Child 1975; 500:63-6
27. Serenius F, Elidrissy AT, Dandona P. VitaminD nutrition in pregnant women at term and in new born babies in Saudi Arabia. J Clin Pathol 1984;37(4): 444-7.

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