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Case Report

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RICKETS- SEVERE VITAMIN D DEFICIENCY: A RARE CASE REPORT

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ABSTRACT

Vitamin D deficiency is the most common cause of rickets. It prevents the efficient absorption of dietary calcium and phosphorus. Rickets is an old disease, a nutritional deficiency and it is the bony manifestation of altered vitamin D, calcium, phosphorus metabolism in a child. We present a rare case of 7 year old female child presented with difficulty in walking, fever cough and cold. Her laboratory and physical examination finding aid in diagnosis of Rickets with Developmental Delay with Lower Respiratory Tract Infection. Treatment was symptomatic therapy and supplementation was initiated with ensuring adequate catch up growth. Vitamin deficiency has become an epidemic in children, and rickets has become a global health issue. Furthermore, inability to properly diagnose rickets can result in incorrect management which may even prove dangerous or futile.

KEYWORDS: Rickets, Vitamin D deficiency, Developmental Delay and Lower respiratory tract infection.

INTRODUCTION

Vitamin D deficiency is known to be the leading cause of nutritional rickets in children remains a major cause of morbidity. This condition was first described in association with skeletal deformities by Glisson during the mid 17th century in London.[1,2] Rickets is often considered an old disease, a nutritional deficiency that has plagued communities for centuries. The earliest reports describing the syndrome of rickets appeared in the English literature around 1650 suggesting the disease is an ancient one.

Rickets attributable to vitamin D deficiency is known to be a condition that is preventable with adequate nutritional intake of vitamin D. The new recommended daily intake of vitamin D is 400 IU/day for all infants and children. Causes of vitamin D deficiency are increased skin pigmentation, decreased nutritional intake of vitamin D, decreased maternal vitamin D stores and exclusive breastfeeding, malabsorption and genetic factors. A severe vitamin D deficiency impairs mineralization of bone tissue (causing osteomalacia) and of growth plates (manifesting as rickets). Clinical manifestations include Lower limb deformities (knock knees) fractures, abnormal dentition and delayed developmental milestones.[1]

In patients with advanced rickets permanent skeletal deformities may occur. Risk factors for developing rickets are children ages 6 months to 24 months are at the highest risk of rickets because of rapid bony growth during this period and other factors include inadequate exposure to sunlight, dark skin, decreased intake of foods containing vitamin D, calcium, phosphorus and breastfeeding without a vitamin D supplement. Treatment depends on the cause and severity with pharmacologic doses of vitamin D in order to buildup the body stores and quickly correct deficiency.

The best method to effectively treat and cure rickets is to give a total of 5-15 mg of vitamin D orally with adequate dietary supply of calcium and phosphorous. These doses can be given safely either as a single day therapy or as daily doses of 2,000-4,000 IU/day for 3-6 months. Exposure to sunlight could prevent and treat rickets and fortification of milk with vitamin D eradicated rickets as a major health problem. The goal of this article is to demonstrate that although there are many osseous diseases that can appear in early ages, Rickets remains a formidable yet treatable disease and here we present a rare case of Rickets with developmental delay and fever and obviously managed conservatively and discharged.

CASE REPORT

A 7 years old female child brought by parents with chief complaints of difficulty in walking since 5 years, fever, cough & cold since 3 days and rapid. Her history of present illness states that fever was high grade, intermittent not associated with chills & rigors. Cold and cough which is dry cough, and more during night times. Past history reveals that the child had difficulty in walking since one year of age, weakness in all four limbs since birth and no history of head injury and convulsions. He was the second living child of non-consanguineous parents, and had two older sisters and one brother. Birth history, developmental history, immunization history and family history was normal.

On examination child was presented with knock knees, short stature, pectus carination and proliberent abdomen. (Anthropometry details include head circumference- 48 cms, weight- 10 kgs, height-90 cms, abdominal girth- 45 cm). Laboratory data included Routine haemogram revealed haemoglobin 10.9 gm%, packed cell volume 32.3%, total leukocyte count 4,600 cells/mm3, platelet count 1,08,000 cells/mm3, serum creatinine 0.6 mg/dl, haematocrit 9.5%, alkaline phosphatase 630 IU/L which was higher than normal, 5.4 mg/dl, phosphorous 4.5 mmol/L, calcium 6.5 mg/dL, vitamin D 48 mg/ml. By above findings the patient was diagnosed as Rickets with developmental delay.

Fig: Presence of Knock knees, Proliberent abdomen.

From the above examinations, the physician was diagnosed as Rickets with Developmental Delay and the child was on treatment with Inj. Cefotaxime 330mg IV TID, Syp. PCT 5ml PO QID, Syp. Salbutamol 3 ml PO TID, Syp.CPM 2ml PO OD, Tab. B Complex PO OD, Inj.Vitamin D 600,00 IU IM OD (given for monthly), Tab. Calcium 1g/day PO OD, Multivitamin drops 5^0 BD, Ultra-D_3 drops 1ml PO OD, The above mentioned therapy was continued until all the symptoms were resolved completely and vitamin supplements were advised to give monthly and counseling regarding vitamin D and calcium rich foods and exposure to sunlight if possible. The treatment was given for 9 days, on 10^{th} day the patient was discharged with vitamin and mineral supplementation.

DISCUSSION

Rickets also called osteomalacaia, a disease of childhood, characterized by softening of the bones as a result of inadequate intake of vitamin D and insufficient exposure to sunlight, also associated with impaired calcium and phosphorus metabolism, disease caused by vitamin D deficiency. It is a chronic can lasts for years or be lifelong. Treatment for rickets may be administered gradually over several months or in a single day dose of 15,000 mcg (600,000 U) of vitamin D. Exposure to sunlight, fortification of milk with vitamin D, and proper education regarding vitamin D and calcium rich foods are necessary to prevent rickets. Here we present a case of rickets with accompanying feature of Developmental Delay which made this case as rare and interesting.

CONCLUSION

Vitamin D and calcium deficiencies causing nutritional rickets have a major impact on health, growth and development of infants, children, and adolescents. Furthermore, inability to properly diagnose rickets can result in incorrect management and may even prove dangerous or futile. Therefore it is inevitable to provide healthcare professionals, diagnosis and management of nutritional rickets and to provide policy makers with a framework to work toward its eradication. Implementation of international rickets prevention programs, including supplementation and food fortification is urgently required.

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