Case Report On Vitamin D Resistant Rickets, With Hypothyroidism And Growth Hormone Deficiency

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Abstract

INTRODUCTION: vitamin D deficiency can result in osteoporosis and fractures (broken bones). Other disorders can develop as a result of severe vitamin D deficiency. It may result in rickets in youngsters. The rare condition of rickets makes the bones brittle and prone to breaking. Vitamin D is obtained from food and solar exposure. Vitamin D insufficiency is often not a problem for adults. Some people are more susceptible to the illness than others, particularly people with dark complexion and seniors over 65. Most folks don’t exhibit any symptoms. A significant deficiency may result in bones that are thin, brittle, or malformed.

Patient History:-Patient 14-year-old male admitted to the hospital, Informant is the mother. The patient was alright 12 and half years back when he had a history of fever followed by weakness in both lower limbs. The patient went to the private hospital where he was managed conservatively and advised surgery due to money issues patient came to the hospital for further management. In 2016 he was diagnosed with a metabolic disorder and deformity at the knee joint was managed conservatively then the patient was again admitted in 2017 and managed with ORIF with osteotomy with plating followed by cast application on 03/04/17. Now patient present with deformity in both knees and unable to walk for the last 3 months. Post operatively patient was walking on his own but for the last 3 months, he was unable to bear weight to walk. The patient can stand with support Presenting Complaints and Investigation: The patient 14-year-old male admitted to the hospital with a complaint of deformity in both knees since 12 and a half year

Unable to walk since 3 months, Hb-12.4, MCHV-33.9, MCV-86.7, MCH-29.4, Total RBC Count-4.22, Total Platelet Count-2.27, HCT-36.6, Monocytes-03, T3-1.02, T4-9.89, TSH-7.73 Past History: Underwent investigations and was given medications at the private hospital, and in 2017 for deformity at both knee, he was managed with open reduction and internal fixation with osteotomy followed by cast application, Milestone is delayed. The main diagnosis, therapeutic intervention, and outcome: All examination and investigation doctors diagnosed vitamin D-resistant rickets, with hypothyroidism and growth hormone deficiency with treatment inj. growth hormone 2IU od Subcutaneous, tab.levothyroxine 50 mg 0d in morning,tab.limeee od,syp.calcimax-p 5mlbd, KCIT solution bd,syp mvbc 5mlbd, vitamin d3 sachet one's week. Conclusion: Preventive measures targeted at lowering the vitamin D shortfall in pediatric age could be taken in response to these findings.

Keywords: Vitamin D deficiency, Growth Hormone, Thyroid.

INTRODUCTION

Background: Numerous conditions that affect bone strength can be found on the metabolic bone disease spectrum. These conditions are typically brought on by anomalies in vitamin D, calcium, phosphorus, bone mass, or bone structure. The most prevalent metabolic bone disease in the industrialized world is osteoporosis. The effects of poor mineral density, which include the prevalence of pain and fractures among the elderly in an aging population, it has a significant negative influence on society. However, in children, osteopenia may coexist with a wide range of genetic and acquired disorders with various etiologies that affect the bone tissue's metabolism.1 Growth is a crucial objective indicator of a child’s general health. The issue of short stature is one that physicians in practice frequently face. It is the ultimate result of a complex process that integrates genetic potential, a healthy endocrine system, nutritional state, the effects of chronic diseases, and the degree of physical activity. A disruption at any one of these stages could have a negative impact on growth, resulting in short stature.2-3 Growth hormone deficiency (GHD) can present at any time of life from the neonatal period to adulthood, as a result of congenital or acquired insults. It may
appear as a standalone issue (IGHD) or in conjunction with additional pituitary hormone deficiencies. After a diagnosis of GHD, pituitary abnormalities might develop at any time. The development of additional endocrinopathies, including their frequency, severity, and timing, is highly variable. The information that is now available supports the need for a cautious and ongoing follow-up for any additional pituitary hormone deficits in all individuals who have been diagnosed with IGHD.5 Between the placebo and GH groups, there were no appreciable differences in total T4, free T4, T3, and TSH levels at baseline. The two groups did not significantly differ in their TSH response to TRH or nocturnal TSH rise. We conclude that GH treatment for 12 months does not result in sustained changes in thyroid function in children who are not GH deficient, despite the possibility of an early transient effect of GH treatment.6

Patient Information:- Patient 14-year-old male admitted to the hospital, Informant is the mother. He was a full-term normal delivery at home and cried immediately at birth, with no history of neonatal complications. The patient was alright 12 and half years back when he had a history of fever followed by weakness in both lower limbs. The patient went to the private hospital where he was managed conservatively and advised surgery due to money issues patient came to the hospital for further management. In 2016 he was diagnosed with a metabolic disorder and deformity at the knee joint was managed conservatively then the patient was again admitted in 2017 and managed with ORIF with osteotomy with plating followed by cast application on 03/04/17. Now patient present with deformity in both knees and unable to walk for the last 3 months. Post operatively patient was walking on his own but for the last 3 months, he was unable to bear weight to walk. The patient can stand with support.

Primary concerns and symptoms of the patient: The patient 14-year-old male admitted to the hospital, Informant is a mother patient who presents with deformity in both knees and unable to walk for the last 3 months. Post operatively patient was walking on his own but for the last 3 month, he was unable to bear weight to walk. The patient can stand with support.

Past medical and surgical history:- In 2016 he was diagnosed with some metabolic disorder and deformity at the knee joint was managed conservatively then the patient was again admitted in 2017 and managed with ORIF with osteotomy with plating followed by cast application on 03/04/17.

Family history:-

Habits: - No bad habits

Clinical Findings:- Patient 14-year-old male admitted to the hospital with the complaint of deformity in both knees since 12 and half year Unable to walk since 3 months. Hb-12.4,MCHV-33.9,MCV-86.7,MCH-29.4,TotalRBCCount-4.22,TotalPlateletCount-2.27,HCT- 36.6,Monocytes-03,T3-1.02,T4-9.89,TSH-7.73.

General Examination: Pulse-110, Resrration-18,Bp-140/90mmhg,Temperature-Afebrile,Height-88cm,Weight-15kg,BMI-19.37,BSA(Mosteller)0-0.61

Systemic Examination-respiratory system-Air Entry Equal and Normal

Cardiovascular System-Normal

Central Nervous System-Conscious and Oriented

Abdominal Examination-Soft Non-Tender

Local Examination:- Patient can stand with support, gait could not be assessed as the patient is unable to walk on his Patient is examined in a supine position On inspection of both knees No swelling Fixed flexion, and genu valgus Deformity present at knee Excessive Anterior bowing of femur and twisting deformity in both tibia Muscle wasting at thigh and calf present Limb length discrepancy seen Overlying skin normal over knee 7cm longitudinal healed scar present over the lateral aspect of distal thigh Palpation No Swelling Genu valgus at right side knee is 30 degrees and left side knee is 20 degrees Fixed flexion deformity at knee right side 10-degree left side 5 degrees No local rise of temperature Healed scar is free from underlying soft tissue, non-tender, healed by primary wound healing No bony and soft tissue tenderness present Excessive Anterior bowing of femur present Active toe and ankle movement present Active knee and hip ROM present Distal circulation intact.
Timeline: The patient was admitted, and because her general health was terrible then, he took treatment at Hospital, where she received the appropriate care.

Diagnostic assessment: All investigation and treatment patient diagnosis Vitamin D Resistant Rickets, With Hypothyroidism And Growth Hormone Deficiency, treatment inj. growth hormone 2IU od Subcutaneous, tab.levothyroxine 50 mg 0d in morning, tab. limcee od, syp. calcimax-p 5mlbd, KCIT solution bd, syp mvbc 5mlbd, vitamin d3 sachet one's week.

Diagnosis: All examination and investigation doctors diagnosed vitamin D-resistant rickets, with hypothyroidism and growth hormone deficiency.

Therapeutic Intervention: Treatment inj. growth hormone 2IU od Subcutaneous, tab. levothyroxine 50 mg 0d in morning, tab. limcee od, syp. calcimax-p 5mlbd, KCIT solution bd, syp mvbc 5mlbd, vitamin d3 sachet one's week.

Medical intervention: Treatment inj. growth hormone 2IU od Subcutaneous, tab. levothyroxine 50 mg 0d in morning, tab. limcee od, syp. calcimax-p 5mlbd, KCIT solution bd, syp mvbc 5mlbd, vitamin d3 sachet one's week.

Results and follow-up:

A follow-up: review after 1 month at pediatric opd

Intervention adherence and tolerability: Patients took all given medications regularly, followed a healthy diet, and did not require any intervention. The treatment was favorably received by the patient.

Discussion:

Childhood is fundamentally about growth, which is a result of the intricate interactions between genetic, hormonal, and environmental influences. Monitoring the growth of children is crucial for tracking their health and development. A child's height can exceed percentile lines on a growth chart during the first few years of life, reflecting, at least in part, his or her genetic makeup. After this point, children continue to grow in a fairly regular fashion, and any divergence from the norm may indicate an underlying chronic disease. Therefore, accurate growth measurements ought to be included in every pediatric exam.7 Patients receiving GH had marginally greater PTH levels than untreated patients. This is consistent with a prior study that showed a tendency for GH to exacerbate hyperparathyroidism in kids with XLH. This is most likely caused by the phosphate-raising abilities of GH, and it emphasizes the necessity of vigilant PTH monitoring and calcitriol dosage adaptation in these individuals.8 Lack of vitamin D in the body leads to the condition known as vitamin-D deficiency rickets, which manifests in early childhood. Vitamin D deficiency may be brought on by inadequate dietary intake, a lack of sun exposure, or malabsorption disorders, in which the intestines are unable to properly absorb nutrients from food. The body’s metabolism of calcium and phosphorus depends on vitamin D. Vitamin D is necessary for healthy bone growth and development because it regulates how calcium is deposited in the bones. The main signs of vitamin D deficiency rickets are fractures, seizures, sluggish growth, bone abnormalities, and discomfort. In rare cases, it can be effectively treated with vitamin D supplementation and extra calcium intake.9-34

Conclusion:

Rickets is common. The primary environmental and cultural factors that contributed to its prevalence were aversion to the sun's year-round illumination. Vitamin D supplementation should be administered because rickets is a disease that can be avoided. Although many systemic disorders have an impact on thyroid function, it can be challenging to pinpoint thyroid malfunction as the sole cause of growth retardation in these cases because the retardation is frequently complex. Most of the aforementioned disorders have negative effects on the target organs as well as the hypothalamic-pituitary-IGF-1 axis. As a result, complex mechanisms that control growth throughout disease processes exist. To comprehend and treat the growth retardation linked to
particular disorders, more research must be done on endocrine system interactions as well as the communication between the immunological, neurological, and endocrine systems.

REFERENCES