Case Report
Hypocalcemia Nutritional Rickets: A Curable Cause of Dilated Cardiomyopathy

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Summary
Dilated cardiomyopathy is an important cause of heart failure in children. Often it requires transplantation, but on rare occasions it is curable by micronutrient supplementation. Hypocalcemic nutritional rickets was found to be a cause for dilated cardiomyopathy in a 15-month-old child. The patient responded to calcium and vitamin D supplementation promptly and left ventricular systolic function normalized after 3 months of treatment. Nutritional rickets must the considered as an important curable cause for dilated cardiomyopathy among children especially in regions where nutritional rickets is still common.

Key words: hypocalcemia, rickets, dilated cardiomyopathy.

Introduction
Dilated cardiomyopathy is an important cause for heart failure in children. In most of the cases, the cause remains idiopathic, but nutritional deficiencies like carnitine, selenium, calcium and taurine have also been implicated as a cause for it, since their depletion can diminish ventricular contractility [1]. Hypocalcemic Vitamin D deficiency rickets is an important and common problem in developing countries. Although it has been reported that asymptomatic left ventricular dysfunction may develop in patients with hypocalcemic nutritional rickets and it improves with treatment, dilated cardiomyopathy and congestive heart failure are rare [2–4]. We describe here a 15-month-old child with dilated cardiomyopathy in whom severe hypocalcemic nutritional rickets was diagnosed during the etiological assessment and which improved with calcium and vitamin D supplementation.

Case Report
A 15-month-old male child from a village of Sangrur, Patiala was admitted in our hospital with a history of breathlessness and sweating while feeding since last 1 month, which was progressively increasing. He also received treatment for the diagnosis of bronchopneumonia in a private hospital. From there, he was referred to our hospital because of cardiomegaly on chest X-ray.

He was a product of full-term normal delivery with normal birth weight. Child was predominantly top fed since birth on diluted cow’s milk, because of inadequate breast milk production. Developmentally, he had slight delay in motor milestones. His body weight was 7.0 kg (<3rd percentile), length 72 cm (25th percentile) and head circumference 44.5 cm.

Physical examination revealed the presence of signs of rickets (rachitic rosary, Harrison’s sulcus, widening of both wrists) and mild pallor. His vitals were heart rate of 152 beats per minute, respiratory rate of 56 breaths per minute and blood pressure of 90/68 mm of Hg. He had minimal chest retractions with few rhonchi and crepitations on auscultation. On cardiovascular system (CVS) examination, tachycardia, no murmur but apex beat was shifted to left. Liver was palpable 3 cm below right costal margin but there was no splenomegaly. Rest of systemic examination was unremarkable.

His hemogram showed Hb of 7.5 gm%, total leucocyte count (TLC) 8100 mm−3 and platelets...
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252 000 mm⁻³. Peripheral smear showed mild hypochromic, microcytic anemia. Metabolic profile showed serum bilirubin of 0.7 mg dl⁻¹, SGOT 70 U l⁻¹, SGPT 31 U l⁻¹, total protein 6.9 gm%, albumin 3.7 gm dl⁻¹, globulin 3.2 gm dl⁻¹, B urea 25 mg dl⁻¹ and creatinine 0.2 mg dl⁻¹. Chest X-ray showed cardiomegaly [Cardiothoracic (CT) ratio in Chest X-ray: 60%] (Fig. 1).

In electrocardiography, cardiac rhythm was normal sinus rhythm, normal QRS axis, PR interval 0.12 s and QTc 0.35 s. Echocardiography study revealed an enlarged left ventricle and hypokinetic left ventricular wall motion. Left ventricular end diastolic diameter was measured as 45 mm, left ventricular end systolic diameter as 39 mm and ejection fraction (EF) as 25%. Wrist X-ray also revealed signs concordant with severe rickets. Total serum Ca⁺⁺ was detected as 7.0 mg dl⁻¹ (8.6–10.2 mg dl⁻¹), phosphorus as 2.5 mg dl⁻¹ (2.7–4.5 mg dl⁻¹) and alkaline phosphatase as 309 U l⁻¹ (40–129 U l⁻¹). His parathyroid hormone levels were 326 pg ml⁻¹ (12–72 pg ml⁻¹). Child was diagnosed to have dilated cardiomyopathy due to Vitamin D deficiency rickets. Treatment included anti-congestive drugs (digitalis, diuretics and ACE inhibitors) in addition to blood transfusion, Vitamin D (600000 IU intramuscular single dose) and calcium supplementation. Child graduation recovered and signs related to heart failure disappeared in a few days. Child was discharged and calcium supplements were continued. On follow-up at 1 month, wrist X-ray showed calcification zone on distal end of radius and ulna and total serum Ca⁺⁺ was measured as 8.8 mg dl⁻¹. Echocardiography at 3 months was normal with an EF of 60%.

Discussion

Dilated cardiomyopathy is an important cause of heart failure in children. Often it requires transplantation, but on rare occasions it is curable by micronutrient supplements. Various nutritional deficiencies like carnitine, calcium and selenium remain an important treatable cause for dilated cardiomyopathy, which have better prognosis compared to idiopathic variety [5]. Such deficiencies of vitamins and micronutrients are more prevent in developing world, making them more important cause of dilated cardiomyopathy in these areas. Rickets is a metabolic disorder of the bone that develops due to insufficient mineralization of the bone tissue and presents its more striking findings on the skeletal system. Hypocalcemic nutritional rickets is common here due to inadequate nutrition and insufficient exposure to sunlight. Risk factors for developing vitamin D deficiency and rickets include low maternal levels of vitamin D, indoor confinement during the day, living at higher altitudes, living in urban areas with tall buildings, air pollution, darker skin pigmentation, use of sunscreen and covering much or all of the body when outside [6]. The most prominent biochemical feature of that is hypocalcemia. Calcium ions have a key role in the excitation as well as the contraction of the cardiac muscles, and reduction in serum levels because hypocalcemic rickets may affect ventricular contraction [7, 8]. Hypocalcemia is present in most of patients with nutritional rickets and may be severe causing convulsions. However, congestive cardiac failure and cardiomyopathy are rare [2, 5].

In patients with cardiomyopathy and rickets, long standing hyopcalcemia has been considered the leading cause of cardiomyopathy, as happened in our case. Nevertheless, the exact mechanism of cardiomyopathy in these cases has not been completely understood. Disturbed carnitine metabolism in nutritional rickets may be responsible for the development of cardiomyopathy in these children [9].

One of the most common causes of dilated cardiomyopathy in infancy is myocarditis [10, 11]. Its definite diagnosis can be made only by endomyocardial biopsy, which is not routinely done. Presence of hypocalcemic nutritional rickets and prompt response to vitamin D and calcium supplements in cardiac failure suggest that in our case the reason for dilated cardiomyopathy was rickets.

In conclusion, nutritional rickets must be considered in the etiological assessment of dilated cardiomyopathy among infants especially those living in regions in which nutritional rickets is still common. These patients promptly respond to calcium and vitamin D supplementation and appropriate treatment can lead to complete recovery.

References


