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Case Report

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Vitamin D Deficiency Induced Hypocalcemia as a Leading Cause of Dilated Cardiomyopathy: A Rare Report

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Abstract

Hypocalcemia is an infrequent and essential reversible cause of Dilated cardiomyopathy, in infant's hypocalcemia is usually due to vitamin D deficiency and often results in high mortality. Beside standard treatment of heart failure, restoration of serum calcium levels with calcium and vitamin D supplementation should be considered. This report describes a 5 months old infant case of dilated cardiomyopathy due to hypocalcemia as a result of vitamin D deficiency.

Key words: Dilated cardiomyopathy; Vitamin D deficiency; Calcium deficiency

Introduction

Hypocalcemia, a critical metabolic disorder, is associated with musculoskeletal, neurologic and cardiogenic symptoms. It can lead to seizure disorders, heart failure, muscle tremors and fasciculation, muscle cramping, facial rubbing, stiff gait, restlessness, aggression, hypersensitivity, and disorientation [1]. Among which, heart failure and cardiomyopathy are infrequent complications of hypocalcemia in pediatric population with prevalence rate of affecting 3.4-26 cases in million per year [2]. On the other side, pediatric cardiomyopathies are life-treating rare diseases with an incidence of 1.1 to 1.5 per 100'000. In specific, dilated and hypertrophic cardiomyopathies are frequent, while restrictive non-compaction and mixed cardiomyopathies are uncommon [3] Dilated cardiomyopathy can develop into heart failure with a high mortality and morbidity rate [4]. Hence, hypocalcemia is one of the rare and revocable causes of secondary CMP. By this paper, we aim to cast light on clinical diagnostic and treatment methods of a unique case presentation, a five months old infant with dilated cardiomyopathy and mild left ventricular non compaction due to hypocalcemia.

Case Presentation

A 5 months old infant presented with convulsion, apnea and cyanosis. Before this episode, he was complaining of coughing and had difficulty in breathing associated with poor feeding and muscle spasms that the clinical examination was normal all through. He recovered after taking azithromycin and co-amoxiclav antibiotic regimen.

He was delivered preterm due to Premature Rupture of Membranes (PROM) by cesarean section. Apparently, goat milk was the sole component of his diet and he has no history of Multi vitamin nor Vitamin A+D consumption.

The anterior fontanel was wide open, no murmur was heard on cardiac examination. Cardiac echocardiography was performed showing diffused LVNC, moderate to severe MR with multiple jets, moderate TR, remarkable decrease in LV systolic function (EF=25%) with mild decrease of RV systolic function(RV EF=30-35%), normal PAP and normal PV drainage, without any stenotic lesion or coronary abnormalities. He was admitted to Intensive

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care unit for 30 days. The laboratory data finding were as follows; alkaline phosphatase of 1435IU/L, calcium was 5mg/dL, phosphate was 7mg/dL, BUN was 17mg/dL, Creatinine was 0.5, Sodium was 136mEq/L, Potassium was 5.6meq/L, uric acid 2.6mg/dL, Hemoglobin was 10.4g/dL, white blood cells were 15.59mm3, and platelets were 473,000mm3. Electrocardiography showed evidence of LV enlargement with long numerous R wave from V5 to V6 and 1 and AVL. Chest x ray was compatible with cardiomegaly. A brain ultrasound scan showed no intra- cranial lesion nor abnormal echogenicity.

He was treated with Calcium gluconate 6cc/kg, Calcicare 0.8cc/kg, calcitriol 0.05mg/kg, vitamin D 1000unit/kg for 10 days. At the follow-up echocardiography 11 days post treatment mild to moderate TR, mild to moderate MR, improved systolic function (EF=58%), normal PV drainage, NL PAP, no CoA were reported. The biochemical findings on discharge were; calcium 9mg/dL, phosphate 2.6mg/dL, BUN 4mg/dL, creatinine 0.5 mg/dl, Na 135mEq/L, K 4.3meq/L. The biochemical findings at different treatment stages is well illustrated at the (Table 1).

Table 1: Biochemical findings at different treatment stages.

Biochemical Test	Normal Range	Onset	Discharge
Calcium (mg/dl)	8.6-10.3	5	9
Phosphate (mg/dl)	3.1-6	7	3.8
Magnesium (mg/dl)	1.5-2.3	1.4	2.6
25hydroxy vit D (ng/ml)	>30	<8	40

Discussion

Pediatric Dilated Cardiomyopathies (DCM) defined as a measured LV ejection fraction <45%, may rise from innumerable causes as, coronary artery abnormalities, tachyarrhythmia, exposure to infection or toxins, infectious diseases, metabolic disorders, endocrine diseases, nutritional deficiencies, electrolyte and renal anomalies, deposition diseases, autoimmune disorders, and lastly systemic disorders. However, myocarditis is responsible for more than 50% of known cases of DCM. Infants with DCM often present with symptoms of HF such as tachypnea, dyspnea, tachycardia, and feeding difficulty [3,5].

In conclusion, hypocalcemia is an infrequent and essential reversible cause of DCM. The pathophysiology of hypocalcemia DCM is quite doubtful, even though the physiologic role of calcium on muscle contraction is well recognized, cardiac contractions can be directly affected by calcium through the excitation-contraction coupling [6]. Beside heart failure, elevated cardiac enzyme and ST-segment changes in ECG which can come with acute myocardial infarction may also rise from hypocalcemia [7]. Hypocalcemia cardiomyopathy is unmanageable with cardiac failure treatment individually but responses to calcium replacement [8].

Considering that goat milk was a sole component in our patient diet, and he had no history of vitamins consumption. Each 120 cc of goat milk contains about 29 units of vitamin D, while an infant needs 400 units of vitamin D per-day for their growth [9], hence vitamin D deficiency can be developed if goat milk is not fortified with vitamin D. In conclusion, our patient developed dilated cardiomyopathy due to hypocalcemia. Significant recovery was evident in echocardiographic study after intravenous and oral calcium administration.

Acknowledgement

None.

Conflict of Interest

No conflict of interest.

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