Brief Communication

Infants with dilated cardiomyopathy and hypocalcemia

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ABSTRACT

Hypocalcemia is a rare reversible cause of dilated cardiomyopathy (DCM) and congestive heart failure; however, there are few reported cases, especially in infants. We describe 12 infants presenting with DCM and congestive cardiac failure who were found to have hypocalcemia. Vitamin D deficiency was the cause of hypocalcemia in all cases. The infants improved on supplementation with vitamin D and calcium.

Key words: Dilated cardiomyopathy, hypocalcaemia, vitamin D deficiency

Introduction

Calcium has a central role in myocardial contraction coupling and hypocalcemia decreases myocardial contractility.[1] However, dilated cardiomyopathy (DCM) due to hypocalcemia in infants has been rarely reported. In some reported cases, correction of hypocalcemia was associated with resolution of congestive heart failure and the left ventricular (LV) geometry and systolic function.[2] We herein report our experience with 12 infants who had presented with DCM and were found to have hypocalcemia.

Methods

We retrospectively analyzed the records of 124 infants, who presented with DCM without any structural heart defect over a 6 year period between 2007 and 2013. We assessed the number of infants who had hypocalcemia, the cause of hypocalcemia and the response to correction of hypocalcemia.

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Results

A total of 12 infants (9.68%), including 8 males, had hypocalcemia as the main cause of congestive cardiac failure (CCF)/DCM among 124 infants included in this retrospective analysis. Age of the 12 infants ranged from 38 days to 11 months (median 4 months), with normal weight (50th-69th percentile). A total of 10 cases presented with DCM and CCF (one with shock) and 2 with DCM without overt CCF of 1-3 weeks duration. Two children had convulsions after hospitalization. 10 infants were breastfeed, 2 formula feed. None had signs of rickets.

All cases had cardiomegaly (cardiothoracic ratio >55%) in chest radiograph and prolonged corrected QT (QTc) interval, median 0.54 s (range 0.49-0.59) (normal <0.45) in 12 lead electrocardiogram. Echocardiography (ECHO) revealed moderate to severe depressed LV function with dilated LV, but no structural heart defect was identified in any of the cases. Left ventricular ejection fraction (LVEF), median 24% (range 17-34) (normal >45).

Hemoglobin, serum magnesium, sepsis screen, renal and liver function test and carnitine level was normal in all. Serum total calcium (corrected for albumin) was low in all infants, median 6.1 mg/dL (range 5.4-7.2) (normal 8.5-10.5), with raised alkaline phosphatase, median 1800 U/L (range 1300-3160) (normal 160-450),

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serum phosphorus was median 3.4 mg/dL (range 2.4-4.1) (normal 2.7-4.5). Serum 25 hydroxy vitamin D (25[OH] D), median level 7 ng/mL (range 2-12) (normal 30-55) was low in all cases. In 10 (83.3%), 25(OH) D values were below 10 ng/mL and <15 ng/mL in two patients (16.7%).

Parathyroid hormone (chemiluminescent immunoassay), median 346 pg/mL (range 278-769) (normal: 8-65) was elevated (secondary hyperparathyroidism) in all cases. Maternal serum vitamin D level was found to be low (8.99 ± 6.63 ng/mL) in all except one case, but serum calcium and phosphorus were within normal limit in all cases.

All cases received routine therapy for CCF (digoxin, frusemide and angiotensin converting enzyme [ACE] inhibitor). Intravenous calcium gluconate (10%) was given as continuous intravenous infusion (2 mg/kg/h). Simultaneously oral calcium 80 mg/kg/day of elemental calcium in three divided doses and vitamin D3 (cholecalciferol) 2000 unit daily orally was started. Serum calcium levels normalized in 1-4 days in all. All improved within 3 days of treatment of restoration of normocalcemia and were discharged in about 10 days with normal QTc and significantly improved of LVEF, median of 38% (range 30-57%). After discharge oral decongestants, ACE inhibitors were gradually stopped over 4 months in 10 patients. On follow-up, serum calcium level remained normal on oral calcium and vitamin D. LV size and ejection fraction (EF) normalized in 10 infants at a median of 3 months (range 2-5 months) from presentation. Two infants had improved, but some persistent LV dilatation and systolic dysfunction with EF of 40% and 44% (from baseline of 21% to 24% respectively).

Discussion

Calcium has a direct effect on the strength of myocardial contraction through excitation-contraction coupling. Hypocalcemia reduces myocardial contractility, but the incidence of CCF and cardiomyopathy due to hypocalcemia is very rare.[2] Hypocalcemic cardiomyopathy is usually refractory to conventional treatment for cardiac failure, but responds favorably to restoration of normocalcemia. 12 (9.68%) among 124 of our patients with DCM and CCF had severe hypocalcemia due to vitamin D deficiency and most responded dramatically to calcium correction and vitamin D.

In adults, hypocalcemic ventricular dysfunction has been mainly attributed to hypoparathyroidism.[3] Vitamin D deficiency is the main cause of hypocalcemia in infants and old children. Nutritional rickets is still prevalent with the primary etiology being vitamin D deficiency in the breastfed infants and children.[4] Maiya, *et al.* reported 16 cases of cardiomyopathy due to hypocalcemia in children, associated with vitamin D deficiency.[5] Another study found four exclusively breastfed African American infants with CCF and DCM due to hypocalcemia, whose cardiac function returned to normal within months of treatment with vitamin D and calcium.[6] An Indian series found hypocalcemia in 16% infants with severe LV dysfunction, vitamin D deficiency was identified as the main cause of hypocalcemia. These children improved on supplementation of vitamin D and calcium.[7]

A prospective study found asymptomatic ECHO abnormalities in patients with rickets.[8]

Vitamin D deficiency in developing countries is mainly nutritional, especially among exclusively breastfed infants born to mothers with high-risk factors such as low vitamin D stores, dark skin and unexposed to sunlight.[5] Infants born to vitamin D deficient mothers are at risk of early and fatal squeals of hypocalcemic vitamin D deficiency.[9]

Conclusion

Hypocalcemia though rare is a very important reversible cause of DCM. Routine measurements of serum calcium should be considered in all infants and children, where the etiology of cardiomyopathy is unknown. Prompt correction of hypocalcemia can lead to recovery of cardiac function. Vitamin D deficiency remains a major health problem in infants of mothers with low vitamin D stores especially those who are exclusively breastfed. Routine vitamin D prophylaxis for pregnant mothers and infants should be stressed to prevent serious complication like hypocalcemic DCM.

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