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BMH Medical Journal 2015;2(1):20-23 **Case Report**

Infant with Hypocalcemia and Dilated Cardiomyopathy Secondary to Vitamin D Deficiency

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Abstract

Hypocalcaemia is a rare but reversible cause of dilated cardiomyopathy with limited cases being reported in the literature. Vitamin D deficiency is the main cause of hypocalcaemia in almost all reported cases. We report a newborn who presented with hypocalcaemia induced dilated cardiomyopathy secondary to Vitamin D deficiency. After calcium therapy, the baby showed a rapid recovery of the cardiac function.

Introduction

Dilated cardiomyopathy (DCM) has an estimated incidence of 1.13 cases per 100,000 children. Diagnosing the primary etiology occurs in fewer than half of these children but significantly improves their outcome [1]. DCM is mostly idiopathic, however infection and metabolic causes haves been identified in some cases in which the defect in myocardial contractility is irreversible [2]. Here we report a case of dilated cardiomyopathy secondary to hypocalcaemia that occurred due to Vitamin D deficiency in an infant.

Case Report

An exclusively breastfed male child with a birth weight of 3.20 kilograms, who had a term delivery by LSCS and discharged on the third day of life after an uneventful post natal period, presented to us at the age of 53 days with poor feeding, shortness of breath and sweating of 3 days duration. On examination he had tachycardia (PR 166/minute) and tachypnoea (RR 68/minute) with cold extremities (CRT > 3 seconds). All the peripheral pulses were felt with no radio-femoral delay. The O2 saturation was less than 90% in room air but improved to 100% with inhalation of 5 liter oxygen/min. The infant's weight was 5.5 Kg, length 58 cm, and head circumference 38 cm, all within the normal range.

Examination of the cardio-vascular system revealed that the apex was in the sixth left intercostal space in the mid-clavicular line. Auscultation revealed a loud third heart sound and a gallop rhythm with normal first and second heart sounds. On auscultation of the respiratory system there were

bilateral crackles. Examination of the abdomen revealed a liver enlargement with a span of 9 cms.

Three hours after admission the infant had a generalized tonic seizure and went into apnoea with bradycardia, which was promptly managed and resuscitated with bag and mask ventilation.

The infant's initial investigation reports were as follows: Random Blood sugar: 198 mg%, Serum Calcium: 5 mg%, Serum Phosphates: 8.8mg%, Serum Alkaline phosphatase: 475 IU/litre, Blood Urea: 27 mg%, Serum Creatinine: 0.4 mg%, Serum Magnesium: 2.1 mg%, Serum electrolytes and Complete Blood Counts were within the normal ranges.

Chest X-Ray showed cardiomegaly (CT ratio of 65%) with clear lung fields and Electrocardiography showed prolonged QT interval with ST segment prolongation. ECHO showed depressed left ventricular function and ejection fraction was only 41%. The impression given by the cardiologist was Dilated Cardiomyopathy (DCM) with possibility of myocarditis.

The infant was managed with oxygen, intravenous fluids, intravenous anti-convulsants (Lorazepam followed by Fosphenytoin) and anti-biotics (Co-amoxyclav) initially. Intravenous Calcium gluconate was given as bolus followed by a continuous infusion after getting the serum calcium results. Even after 3 days of Calcium infusion the serum Calcium was still low (7.5mg%). In the meantime his Serum Vitamin D levels were obtained. Serum 1,25-di-hydroxy-cholecalciferol was 53 pmol/litre (normal) but Serum 25-hydroxy-cholecalceferol was low at 13.4 (normal: 30.0 to 74.0 ng/mL), and he had a raised Serum Parathyroid hormone level of 140.80 pg/ml (normal being 15.0 - 68.0 pg/ml). The infant's mother's Vitamin D level was within normal limits.

A diagnosis of Vitamin D deficiency was made and Injection Cholecalciferol followed by oral 1,25 di-hydroxy-cholecalciferol was given daily. He gradually improved, his serum Calcium level normalized and his ECHO showed an improved left ventricular ejection fraction of 71% with improved left ventricular contractility. He was discharged on the 12th day of admission, on oral calcium supplement. The infant is now 6 months old, completely asymptomatic, on maintenance dose of calcium orally and has normal growth and development.

Discussion

Calcium has a central role in myocardial contraction coupling and hypocalcaemia reduces the myocardial function. Congestive cardiac failure due to hypocalcaemia have been reported, though rare and only few cases of hypocalcaemia induced cardiomyopathy have been reported [3,4].

Hypocalcaemia causing DCM is reversible with complete recovery after normalization of serum calcium is seen. Vitamin D deficiency is the main cause of hypocalcaemia in infants and older children. Nutritional rickets is still prevalent with the primary etiology being Vitamin-D deficiency in breastfed infants and children [1,5].

Ionized Calcium has a central role in regulating myocardial contraction. During the activation of cardiac action potential, ionized calcium enters the cell through depolarization activated calcium channels. Plasma membrane (PM) depolarization triggers the opening of voltage-gated Ca2+ channels (VGCC), allowing influx of calcium ions. This triggers the release of a greater amount of Ca2+ from the sarcoplasmic reticulum (SR) via ryanodine receptors (RyR2). After Ca2+ - induced contraction of the sarcomere, myocyte relaxation occurs when calcium ions are pumped back into the SR by sarco/endoplasmic reticulum Ca2+ ATPase (SERCA) or removed from the myocyte by the Na+/Ca2+ exchanger (NCX) [6]. In immature heart, this SR calcium transport system is less well developed and such hearts consequently have an increased dependence on transport of calcium from outside the cell for contraction [7].

Vitamin D deficiency remains a major public health problem in India especially among infants who

are exclusively breastfed and born to mothers with high-risk factors such as low Vitamin D stores, dark skinned and or living a sedentary life style that further limit adequate ultraviolet light exposure [8-10]. Infants born to mothers with deficiency of Vitamin D are at risk of developing early and fatal sequelae of hypocalcaemic Vitamin D deficiency [11-13].

In a study where a hospital database search was conducted from the year 1997 to 2007 to identify patients with confirmed Vitamin D deficiency in addition to DCM, four exclusively breastfed African American infants were identified. These infants presented with congestive heart failure secondary to DCM and on admission they were found to have laboratory evidence consistent with hypocalcaemic rickets. These patients responded dramatically to treatment with Vitamin D and Calcium and cardiac functions returned to normal within months [11].

In another study, 15 Indian infants (age between 45 days and 5 months) presented with severe left ventricular dysfunction, and were found to have hypocalcaemia with or without hypomagnesaemia. Vitamin D was identified as the main cause of hypocalcaemia. These children improved on supplementation of Vitamin D and Calcium [14].

In our case, the baby presented with severe hypocalcaemic DCM at a very young age of one and half months. He was exclusively breastfed. Our baby was investigated for other possible causes for his DCM; like metabolic disorders, viral infections and other cardiac causes leading to DCM. There was no family history of any similar problem and the investigations were negative for other causes, hence it was evident that it was only due to Vitamin D deficiency. The child also improved very well with Calcium and Vitamin D administration. Most cases of nutritional rickets can be prevented by universal administration of daily multivitamin containing 400 IU of Vitamin D to infants who are breast fed; older children should receive 600 IU/ day [15].

Conclusion

The association of hypocalcemia-induced DCM is of noted importance when encountering a patient with a new diagnosis of DCM. With increased suspicion, the diagnosis of hypocalcemia-induced DCM can be made and prompt treatment can lead to recovery of cardiac function and better patient outcome. This report adds to the already existing evidence that Vitamin D deficiency remains a major health problem in India. It highlights the importance of administering Vitamin D prophylaxis for pregnant mothers at a daily dose of 800 - 1000 units and for babies at a daily dose of 400 units soon after birth. This can prevent hypocalcemia-induced cardiac problems which is a serious complication of nutritional vitamin D deficiency.

References

1. G.F. Cox, L.A. Sleeper, A.M.Lowe et al. Factors associated with establishing a causal diagnosis for children with cardiomyopathy, Pediatrics. 2006; 118(4):1519-1531.

2. B.G. Kim, S.K. Chang, S.M. Kim, J.S. Hwang, and J.W. Jung, Dilated cardiomyopathy in a 2 month - old infant: a severe form of hypocalcemia with vitamin D deficient rickets. Korean Circulation J. 2010; 40(4):201-203.

3. S. Gulati, A. Bajpai, R. Juneja, M. Kabra, A. Bagga, and V. Kalra. Hypocalcemic heart failure masquerading as dilated cardiomyopathy. Indian J Pediatr. 2001; 68(3):287-290.

4. K.S. Joong. A case of hypocalcemia induced dilated cardiomyopathy. J Cardiovasc Ultrasound. 2010; 18(1):25-27.

5. M.S. Al-Atawi, I. A. Al-Alwan, A. N. Ai-Mutair, H. M. Tamim, and N. A. Al-Jurayyan. Epidemiology of nutritional rickets in children. Saudi J Kidney Disease and Transplantation. 2009; 20(2):260-265.

6. A Garbino, X Wehrens. Emerging role of junctophilin-2 as a regulator of calcium handling in heart. Acta Pharmacologica Sinica. 2010; 31; 1019-21.

7. D. Bernstein. Developmental changes in cardiac function. In: Kliegman R.M., Stanton B.F., Scholar N.F., eds. Nelson Textbook of Pediatrics. 19th edition. Elsevier Inc. 2012; p1854.

8. J.M. Nozza and C.P. Rodda. Vitamin D deficiency in mothers of infants with rickets. Med. J Australia. 2001; 175(55):253 -255.

9. S.R. Grover and R. Morley. Vitamin D deficiency in veiled or dark-skinned pregnant women. Med. J Australia. 2001; 175(5) 251 – 252.

10. R. Namgung and R.C. Tsang. Factors affecting newborn bone mineral content: in utero effects on newborn bone mineralization. Proceedings of the Nutrition Society. 2000; 59(1): 55 – 63.

11. J. Brown, S. Nunez, M. Russell, and C. Spurney. Hypocalcaemia rickets and dilated cardiomyopathy: case reports and review of literature. Pediatr Cardiol. 2009; 30(6): 818-823.

12. A. Mustafa, J.L. Bigras, and B.W. McCrindle. Dilated cardiomyopathy as a first sign of nutritional vitamin D deficiency rickets in infancy. Canadian J Cardiol. 1999; 15(6): 699-701.

13. R.E. Kleinman. Pediatric Nutrition Handbook. American Academy of Pediatrics, 4th edition. Elk Grove Village, III, USA. 1998.

14. M. Tomar, S. Radhakrishnan, and S. Shrivastava. Myocardial dysfunction due to hypocalcemia. Indian Pediatr. 2010; 47(9): 781-783.

15. Larry A. Greenbaum. Rickets and hypervitaminosis D. In: Kliegman R M, Stanton BF, Scholar NF, eds. Nelson Textbook of Pediatrics. 19th edition. Elsevier Inc; 2012. p.205.