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RESEARCH ARTICLE

Avoidable Hypocalcaemia in Infants with Dilated Cardiomyopathy Linked to Maternal Vitamin D Deficiency

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Abstract: ***Background***: Hypocalcaemia, though rare, is a reversible cause of dilated cardiomyopathy in infants. We report six infants with dilated cardiomyopathy secondary to hypocalcaemia and vitamin D deficiency whose mothers were also deficient for vitamin D. ***Material and Methods:*** Six infants presented to us between 1 to 5 months of age with respiratory distress, generalized tonic clonic seizures, and features of congestive heart failure. Results: Chest radiograph was suggestive of cardiomegaly. Echocardiography revealed dilated cardiac chambers and low ejection fraction, median LVEF 27.5 (20-40)%. All had hypocalcaemia, hypomagnesemia, low vitamin D levels and high parathyroid hormone. Mothers of all the infants also had vitamin D deficiency. Infants were treated with oxygen support, diuretics, vasoactive and ventilatory support for congestive cardiac failure and cardiogenic shock. Specific treatment included intravenous calcium infusion and vitamin D supplementation. There was improvement in respiratory and hemodynamic status and normalization of serum calcium. 6-12 weeks after discharge, echocardiography revealed normalization of left ventricular systolic function- median LVEF 65 (52-65)% ***Conclusions:*** Maternal vitamin D deficiency remains a preventable yet significant contributor to infantile hypocalcemia and secondary cardiomyopathy. Early detection, adequate supplementation during pregnancy and lactation, and prompt correction of deficiencies in affected infants can prevent morbidity and ensure complete recovery of cardiac function.

Keywords: Dilated cardiomyopathy, hypocalcaemia, vitamin D deficiency, LVEF

INTRODUCTION

Calcium plays an important role in myocardial excitation-contraction coupling and strength of myocardial contractility. Sarcoplasmic reticulum requires sufficient amount of calcium to initiate myocardial contraction and therefore, conditions with low serum calcium leads to decreased myocardial contractility (1). Hypocalcaemia is one of the uncommon causes of dilated cardiomyopathy (DCM). Hypocalcaemia myocardial dysfunction is often refractory to conventional treatment of congestive cardiac failure (CCF) but responds dramatically to correction of serum calcium. There are only few case reports of myocardial dysfunction and DCM due to hypocalcaemia in infants in whom supplementation with vitamin D and calcium lead to rapid resolution of symptoms of CCF and normalization of cardiac functions (1,2,3). We report six infants with DCM who had hypocalcaemia due to vitamin D deficiency and supplementation with calcium and vitamin resulted in symptomatic improvement and normalization of myocardial functions. In infants with DCM, the possibility of correctable causes such as underlying hypocalcaemia and vitamin D deficiency should be kept in mind and appropriate treatment can lead to normalization of myocardial functions.

MATERIAL AND METHODS

Six infants presented to us between 1 to 5 months of age with respiratory distress, generalized tonic clonic seizures and features of congestive heart failure. Cases were managed in PICU.

Case 1

A 2-month-male presented with rapid breathing for 5-7 days. He was born at term with birth weight of 2 kg and mother had history of hypothyroidism and gestational hypertension. He was exclusively breast fed. Examination revealed irritable child with pallor, tachypnoea, tachycardia, subcostal and intercostal retractions, bilateral crepitations, and hepatomegaly. He was started in oxygen support (continuous positive airway pressure) and furosemide. He had 2 episodes of generalized tonic clonic seizures (GTCS) at 13 hours of hospital stay. On day 2 of hospital stay, there was worsening of respiratory distress and he also developed cardiogenic shock. At this point of time he was intubated and mechanically ventilated and given dobutamine (10µg/kg/min) and adrenaline (0.05 µg/kg/min) infusion.

Investigations (Table 1) revealed persistent hypocalcaemia, vitamin D deficiency, hypomagnesemia, cardiomegaly and echocardiography revealed dilated cardiac chambers with left ventricular ejection fraction (LVEF) of 15-20%. Mother was also vitamin D deficient.

Hypocalcaemia was treated with calcium gluconate (10%) 2 ml/kg bolus followed by intravenous infusion up to 100 mg/kg/day for 48 hours, Magnesium and vitamin D 2000 units/day till discharge (and

continued at home till 3months along with calcium supplements). Over next 48 hours, there was improvement in serum calcium, respiratory and hemodynamic status and LVEF to 40%. Vasoactive drugs were tapered and stopped and he was extubated on day 6. Oral calcium and vitamin D were continued. Mother was also given vitamin D supplements. He was discharged after 10 days of hospital stay. On follow-up after 7 weeks, he was asymptomatic and echocardiography revealed left ventricular ejection fraction of 60%.

Case 2

A 2-month-male presented with history excessive irritability and rapid breathing for 5 days and 1 episode of GTCS. The antenatal and perinatal period was normal and he was exclusively breast fed. Examination revealed tachypnoea, tachycardia, subcoastal retractions, bilateral basal crepitations and hepatomegaly. He was started on oxygen support and furosemide. Respiratory and hemodynamic worsening lead to institution of mechanical ventilation and vasoactive drugs (dobutamine 10µg/kg/min, adrenaline 0.1 µg/kg/min, and milrinone 0.5 µg/kg/min).

Investigations (Table 1) revealed persistent hypocalcaemia, vitamin D deficiency, cardiomegaly and poor myocardial contractility with LVEF of 30%. Mother also had vitamin D deficiency. The persistent hypocalcaemia was treated with calcium correction followed by calcium infusion and vitamin D supplementation. Mother also treated with vitamin D. Over next 36 to 48 hours, there was gradual improvement in clinical status, normalization of serum calcium and LVEF. He was extubated on day 4 and discharged after a hospital stay of 7 days. Follow up after 8 weeks revealed asymptomatic child with normal LVEF.

Case 3

A 1-month-female presented with rapid breathing for 2 days. She was born at term with birth weight of 2.5 kg and mother had no antenatal significant history. She was exclusively breast fed. Examination revealed irritability, pallor, tachypnoea. She was started in oxygen support (nasal prongs) and managed in lines of bronchiolitis. However, she had crepitations and oxygen requirement was there even after distress settled on second day and x ray was suggestive of cardiomegaly. On investigations, she had hypocalcaemia, vitamin D deficiency. Echocardiography revealed dilated cardiac chambers with left ventricular ejection fraction (LVEF) of 25%. Hypocalcaemia was treated with calcium gluconate (10%) 2 ml/kg bolus followed by oral calcium and vitamin D supplementation. Over next 48 hours, there was improvement in serum calcium, respiratory and hemodynamic status. Mother was also given vitamin D supplements as she was also found to be deficient in vitamin D. She was discharged after 7 days of hospital stay. On follow-up after 12 weeks, she was asymptomatic and LVEF improved to 65%.

Case 4

A 1.5-month-male presented with rapid breathing for 4 days and 1 episode of tonic posturing followed by unresponsiveness. The antenatal and perinatal period was normal and he was exclusively breast fed. Examination revealed bilateral basal crepitations and hepatomegaly. He was started on oxygen support and furosemide. Baby developed shock and was started on inotropes and furosemide was stopped. Baby was kept on NIMV mode of ventilation.

Investigations revealed persistent hypocalcaemia, vitamin D deficiency, cardiomegaly and poor myocardial contractility with LVEF of 30%. The persistent hypocalcaemia was treated with calcium correction and vitamin D supplementation. Mother also treated with vitamin D in view of maternal deficiency. There was gradual improvement in clinical status, normalization of serum calcium and LVEF. He was discharged after a hospital stay of 7 days. Follow up after 4 weeks revealed asymptomatic child with normal LVEF.

Case 5

A 3-month-female presented with rapid breathing for 10 days. She was born at term with birth weight of 3 kg and mother had history of hypertension. She was exclusively breast fed. Baby was referred from nearby PHC for respiratory distress and abnormal movements of body. On arrival baby was jittery. Investigations revealed hypocalcaemia and x ray was suggestive of increased hilar markings. Cause of seizures was considered to be low calcium levels. 2D Echo was done that was suggestive of dilated cardiac chambers and low LVEF. Furosemide was started and calcium correction was given. As clinical improvement was there, calcium and vitamin D oral supplements were added. Mother was also found to be vitamin D deficient and supplemented accordingly. On follow up at 6 week LVEF was within normal limits.

Case 6

A 5-month-male born at term, 3kg birth weight, with history of 2 previous admissions due to seizures secondary to low calcium levels, was referred to our centre for further evaluation. At presentation baby had features of congestive heart failure and was maintaining on nasal CPAP. Furosemide was started and investigations revealed low total and ionized calcium levels. Iv calcium was started followed by maintenance calcium and vitamin D. baby required dobutamine that was tapered and stopped after 84 hrs. No further seizures were noted at our centre and antiepileptics were tapered. 2D echo done showed mild pericardial effusion with low LVEF of 25% that improved to 65% at 6 week follow up. Mother was also found to be having low vitamin D levels and

supplemented accordingly. Baby was discharged in satisfactory condition after 12 days of hospital stay.

. Table 1 summarizes the important investigations in these infants.

RESULTS AND OBSERVATIONS:

Table 1: Laboratory investigations in infants with dilated cardiomyopathy due to hypocalcaemia.

Laboratory parameters	Normal values	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age		2m	2m	1m	1.5m	3m	5m
Haemoglobin (gm%)	9.5-13	8	9.4	9	7.4	12	12
Blood glucose (mg%)	60-100	84	114	112	120	92	132
Ionized calcium (mmol/L)	1.12-1.23	0.5	0.6	0.4	0.4	0.3	0.4
Total calcium (mg%)	8.8-10.8	5	5.5	6	7	5.4	6.6
Phosphate (mg%)	3.8-6.5	3.7	2.1	2.1	1.6	1.4	1.8
Alkaline phosphatase (U/L)	145-420	650	769	1040	1430	2100	645
Magnesium (mg%)	1.6-2.6	1.2	1.8	1.9	2	1.3	2.6
25 hydroxy vitamin D (ng/ml)	>30	6	2.7	<1.5	12	10	14
Parathyroid hormone (pg/ml)	10-65	780	620	550	555	90	120
Renal and liver function tests		Normal	Normal	Normal	Normal	Normal	Normal
Mother's 25 hydroxy vitamin D (ng/ml)	>30	6.4	6.2	8.4	8	11	6.6
Chest radiograph		Cardiomegaly (CT ratio >0.6)	Cardiomegaly (CT ratio >0.6)	Cardiomegaly (CT ratio >0.6)	Cardiomegaly (CT ratio >0.6)	Cardiomegaly (CT ratio >0.6)	Cardiomegaly (CT ratio >0.6)
Echocardiography at admission		Dilated cardiac chambers, biventricular dysfunction, global left ventricular hypokinesia, and left ventricular ejection fraction (LVEF) of 15-	Thin walled and dilated cardiac chambers, poor myocardial contractility, and LVEF of 30%	LV hypokinesia, EF 25%	LVEF 30%	Dialted cardiac chambers, LVEF 40%	Mild pericardial effusion, LVEF 25%

		20%					
Echocardiography at follow-up		LVEF 60% at 7 weeks	LVEF 65% at 12 weeks	LVEF 65% at 12 weeks	LVEF 52% at 6 weeks	LVEF 65% at 6 weeks	LVEF 65% at 6 weeks

DISCUSSION

Six infants with DCM are reported who presented with features of CCF and in addition also had hypocalcaemia seizures. The cause for hypocalcaemia was vitamin D deficiency as all were exclusively breast fed and mothers also had vitamin D deficiency. All infants responded dramatically to therapeutic doses of vitamin D and calcium. The underlying cause for myocardial dysfunction was hypocalcaemia due to vitamin D deficiency as suggested by clinical features, laboratory investigations, and rapid response to supplementation with vitamin D and calcium. Coexistent hypomagnesemia may have further contributed to myocardial dysfunction.

Normally calcium binds to troponin–tropomyosin complex inside the sarcoplasmic reticulum and links actin with myosin. Amount of calcium influx dictates the strength of cardiac contraction and is responsible for the myocardial action potential. When extracellular calcium is low, the activation of the action potential gets shifted to a lower membrane electro-potential. This results in increased excitability. Presence of hypocalcaemia prolongs the QT interval, impairs myocardial contractility and may induce ventricular arrhythmias. Role of vitamin D in cardiac contractility is established in various studies. Calcitriol has receptors on heart myoblasts and myocytes. It increases heart tissue calcium uptake (4).

Previously, there are small case series and case reports highlighting hypocalcaemia as treatable cause of DCM. Sanyal et al (3) described 12 infants (median age 4 months, range 38 days to 11 months) who presented with CCF and DCM. All had cardiomegaly, low vitamin D, high alkaline phosphatase, high PTH, and low maternal vitamin D (except in 1). Echocardiography in all cases revealed moderate to severe left ventricular dysfunction with dilated left ventricle and no structural heart defect. The median LVEF was 24% (range 17-34%). All improved with vitamin D and calcium supplementation with normalization of LVEF over next 3 months. Elidrissy et al (5) conducted a review of 61 infants with hypocalcaemia cardiomyopathy in age range from 1 month to 15 months (mean age 5 months). All cases were presented with heart failure and hypocalcaemia; had high alkaline phosphatase, low vitamin D, and high PTH; and echocardiology was suggestive of cardiomyopathy. Most of these cases responded well to supplementation with vitamin D and calcium, cardiotonics and diuretics. Recently, few other authors also reported infants with DCM due to hypocalcaemia and vitamin D deficiency. Supplementation with vitamin D and calcium resulted in rapid recovery of myocardial functions (6,7,8).

Vitamin D deficiency is a major cause of hypocalcaemia in younger age group. The prevalence of vitamin D deficiency is high in our country and possible reasons are inadequate dietary sources of vitamin D, high levels of skin pigmentation, inadequate sunlight exposure, indoor lifestyle, recent increase screen time, use of sunscreens and lack of vitamin D supplementation in infants and in antenatal period (9,10). In infants, maternal vitamin D deficiency could be contributing factor for developing hypocalcaemia, especially in exclusively breast fed babies and those with no vitamin D supplementation. Infants born to vitamin D deficient mothers are at increased risk of early and fatal sequelae (11). Therefore, routine vitamin D supplementation to pregnant mothers and infants should be stressed to prevent serious complications due to vitamin D deficiency and hypocalcaemia (12).

The presented cases underscores the importance of recognizing maternal vitamin D deficiency as an underappreciated yet preventable cause of infantile dilated cardiomyopathy secondary to hypocalcemia. Recognition of these risk factors during routine antenatal care can facilitate early prevention through maternal vitamin D screening and supplementation.

Echocardiographic abnormalities typically resolve within weeks of calcium and vitamin D replacement, highlighting the reversible nature of the condition when diagnosed promptly. Median LVEF at presentation in our series was 27.5 (20-40)% that improved to near normal median LVEF 65 (52-65)% between 6-12 week follow up. However, delayed recognition may result in prolonged cardiac dysfunction, hospitalization, and potentially fatal outcomes. Infants presenting with cardiomyopathy should be evaluated for metabolic causes, including calcium and vitamin D status, before attributing the condition to idiopathic or viral etiologies. Equally, public health strategies should prioritize maternal vitamin D supplementation, particularly in high-risk populations, to prevent neonatal complications.

A recent observational cohort of 148 neonates with maternal vitamin D deficiency found no DCM, which suggests that early detection before prolonged hypocalcemia may protect against overt cardiac involvement (13, 14, 15).

CONCLUSION

In summary,

1. In infants presenting with dilated cardiomyopathy and no obvious viral or structural cause, serum calcium, 25-hydroxyvitamin D, PTH and magnesium should be screened.
2. Maternal vitamin D deficiency remains a preventable yet significant contributor to infantile hypocalcemia and secondary cardiomyopathy.
3. Routine antenatal visits should include screening for vitamin D deficiency.
4. Maternal vitamin D supplementation during pregnancy and lactation should be strengthened especially in high-risk populations; and newborns to be supplemented throughout the infancy.
5. Early intervention in hypocalcemia (calcium + vitamin D) can lead to full cardiac recovery

Limitations & Future Directions

Given the rarity of DCM due to hypocalcemia, available data are largely case-reports and small series. There is a need for larger prospective studies to quantify incidence, identify thresholds of vitamin D/calcium disturbance at which cardiac involvement occurs.

REFERENCES

1. Tomar M, Radhakrishnan S, Shrivastava S. Myocardial dysfunction due to hypocalcemia. Indian Pediatr. 2010;47(9):781-3.
2. Maiya S, Sullivan I, Allgrove J, Yates R, Malone M, Brain C, et al. Hypocalcaemia and vitamin D deficiency: an important, but preventable, cause of life-threatening infant heart failure. Heart. 2008;94(5):581-4.
3. Sanyal D, Raychaudhuri M. Infants with dilated cardiomyopathy and hypocalcemia. Indian J Endocrinol Metab. 2013;17(1):221-3.
4. Cheng Z, Yadav SK, Liu X, Yi Q A reversible hypocalcemic dilated cardiomyopathy caused by primary hypoparathyroidism. Asian J. Med. Sci. 2019;10(2):65-8.
5. Elidrissy AT, Munawarah M, Alharbi KM. Hypocalcemic rachitic cardiomyopathy in infants. J Saudi Heart Assoc. 2013;25(1):25-33.
6. Velayuthan S, Gungor N, McVie R. Hypocalcemic cardiomyopathy as initial presentation of primary hypoparathyroidism. Pediatr Int. 2014;56(4):23-5.
7. Elidrissy AT, Alharbi KM, Mufid M, AlMezeni I. Rachitic hypocalcemic cardiomyopathy in an infant. J Saudi Heart Assoc. 2017;29(2):143-7.
8. Uday S, Fratzl-Zelman N, Roschger P, Klaushofer K, Chikermane A, Saraff V, et al. Cardiac, bone and growth plate manifestations in hypocalcemic infants: revealing the hidden body of the vitamin D deficiency iceberg. BMC Pediatr. 2018;18(1):183.
9. Angurana SK, Angurana RS, Mahajan G, Kumar N, Mahajan V. Prevalence of vitamin D deficiency in apparently healthy children in north India. J Pediatr Endocrinol Metab. 2014;27(11-12):1151-6.
10. Taliercio CP, Seward JB, Driscoll DJ, Fisher LD, Gersh BJ, Tajik AJ. Idiopathic dilated cardiomyopathy in the young: clinical profile and natural history. J Am Coll Cardiol 1985; 6: 1126-1131.
11. Abdullah M, Bigras JL, McCrindle BW. Dilated cardiomyopathy as a first sign of nutritional vitamin D deficiency rickets in infancy. Can J Cardiol. 1999;15(6):699-701.
12. Khadilkar A, Khadilkar V, Chinnappa J, Rathi N, Khadgawat R, Balasubramanian S, et al. Prevention and Treatment of Vitamin D and Calcium Deficiency in Children and Adolescents: Indian Academy of Pediatrics (IAP) Guidelines. Indian Pediatrics. 2017;54(7):567-73.
13. Cayir A, Akyigit A, Gullu UU, Kahveci H, Yildiz D, Kurnaz E, et al. Clinical, biochemical, and echocardiographic evaluation of neonates with vitamin D deficiency due to maternal vitamin D deficiency. Cardiol Young. 2022 Jan;32(1):88-93. doi: 10.1017/S1047951121001633. Epub 2021 May 4. PMID: 33941295.
14. Arola A, Tuominen J, Ruuskanen O, Jokinen E. Idiopathic dilated cardiomyopathy in children: prognostic indicators and outcome. Pediatrics. 1998; 101(3 Pt 1): 369-76.
15. Burch M, Siddiqi SA, Celermajer DS, Scott C, Bull C, Deanfield JE. Dilated cardiomyopathy in children: determinants of outcome. Br Heart J. 1994; 72: 246-50.

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Abbreviations: LVEF- left ventricular ejection fraction, GTCS- generalized tonic clonic seizures, DCM- dilated cardiomyopathy, CCF- congestive cardiac failure, CPAP- continuous positive pressure ventilation.