Clinical Profile, Intensive Care Needs and Outcome of Children with Dilated Cardiomyopathy Associated with Vitamin D Deficiency: A 5-year PICU Experience

Surjeet Kumar[1], Manjinder Singh Randhawa[2], Suresh Kumar Angurana[3], Karthi Nallasamy[4], Arun Bansal[5], Manoj Rohit Kumar[6], Naresh Sachdeva[7], Muralidharan Jayashree[8]

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Abstract

Aim: To describe the clinical profile, treatment details, intensive care needs, and long-term outcome of children with dilated cardiomyopathy (DCM) associated with Vitamin D deficiency (VDD).

Materials and methods: Case records of 14 children with DCM associated with VDD [25(OH)D3 levels <20 ng/mL] admitted to the pediatric intensive care unit (PICU) of a tertiary care teaching hospital between January 2017 and December 2021 were retrospectively analyzed for clinical features, echocardiographic findings, treatment details, intensive care needs, and outcomes.

Results: The median (IQR) age was 6 (2–9) months and 71% (*n*=10) were males. The common modes of presentation included respiratory distress or failure (78.6%), congestive cardiac failure (71.4%), cardiogenic shock (37.5%), and seizures and encephalopathy (14.3% each). The median (IQR) serum calcium was 8.7 (7–9.5) mg%, ionized calcium 0.7 (0.7–1.1) mmol/L, alkaline phosphatase 343 (316–415) IU/L, phosphate 3.5 (2.6–4.5) mg%, PTH 115 (66–228) pg/mL, and 25(OH)D3 5 (3–7) ng/mL. The median (IQR) left ventricular ejection fraction (LVEF) at admission was 22 (17–25)%. The treatment included intravenous calcium infusion (35.7%), vitamin D supplementation in all (57.1% parenteral and 42.9% oral), mechanical ventilation (35.7%), and vasoactive drugs (57.1%). There was no mortality. The median (IQR) duration of PICU and hospital stay was 76 (31–98) hours and 6 (4.7–10) days, respectively. Out of 14 children, 10 (71.4%) were followed-up till median (IQR) of 10 (7–58) months. All were asymptomatic and had normal LEVF (except one had residual moderate mitral regurgitation).

Conclusion: Vitamin D deficiency is a potentially treatable and reversible cause of DCM in children.

Keywords: Inotropes, Intensive care units, Pediatric intensive care units, Vitamin D deficiency.

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Highlights

Vitamin D deficiency (VDD) is common among infants and children. Vitamin D deficiency and resultant hypocalcemia are uncommon but treatable and reversible causes of dilated cardiomyopathy (DCM) in children. With prompt diagnosis and treatment of VDD, the short- and long-term cardiac outcome is usually good among children with DCM due to or associated with VDD.

Introduction

Calcium plays an important role in myocardial excitation–contraction coupling and strength of myocardial contractility. In the presence of low serum calcium, the sarcoplasmic reticulum is unable to maintain a sufficient amount of calcium content to initiate myocardial contraction, leading to decreases in myocardial contractility.[1–3] Hypocalcemia is one of the uncommon causes of dilated cardiomyopathy (DCM), which is usually refractory to conventional treatment of congestive cardiac failure (CCF) but responds dramatically to the correction of serum calcium. Vitamin D is an important regulator of calcium metabolism and bone health and helps in maintaining normal serum calcium. Vitamin D deficiency is widespread throughout the world in healthy population as well as in several disease states. The prevalence of VDD in healthy children ranged from 10% to 90% depending upon geographical location, population and age group studied, the method used to estimate vitamin D levels, and the cutoff value of 25-hydroxy vitamin D [25(OH)D3] used.[4–9] The reasons for high prevalence of VDD are due to inadequate sunlight exposure, indoor lifestyle, wearing clothes, use of sun screens, high levels of skin pigmentation, inadequate dietary sources of vitamin D, and lack

[1–5,8]Division of Pediatric Emergency and Critical Care, Department of Pediatrics, Advanced Pediatrics Center, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[6]Department of Cardiology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

[7]Department of Endocrinology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

Corresponding Author: Suresh Kumar Angurana, Division of Pediatric Emergency and Critical Care, Department of Pediatrics, Advanced Pediatrics Center, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India, Phone: +91 2147483647, e-mail: sureshangurana@gmail.com

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of vitamin D supplementation.[6,10,11] Vitamin D deficiency is also common among hospitalized and critically ill children.[12–16] It has been demonstrated that VDD is not associated with higher mortality among critically ill children, however, VDD increases susceptibility to sepsis and requirement for ventilator support.[17]

As calcium and vitamin D are important for the functioning of the myocardium, the long-standing deficiency of either or both may lead to myocardial dysfunction or DCM. In infants and children with DCM, the possibility of correctable causes such as hypocalcemia and VDD should be kept in mind, and appropriate treatment can lead to normalization of myocardial function. However, there are only a few case reports/series of myocardial dysfunction and DCM due to or associated with hypocalcemia and/or VDD in infants and children in whom supplementation with vitamin D and calcium leads to rapid resolution of symptoms and normalization of myocardial function.[1–3,18–25] Therefore, we planned this study to describe the clinical profile, treatment details, intensive care needs, and long-term outcome of DCM in infants associated with VDD.

Materials and Methods

This retrospective study was conducted in the PICU of a tertiary care teaching hospital in North India over a period of 5 years (January 2017–December 2021) involving 14 infants with DCM associated with VDD. The study protocol was approved by the Institutional Ethical Committee, and the paper was approved by the Departmental Review Board.

The data were collected from the electronic database records and case files on predesigned study proforma regarding biometric details, symptoms and signs at admission, laboratory findings (complete blood count, renal and liver function tests, serum calcium, phosphate, alkaline phosphatase, parathyroid hormone (PTH), 25(OH)3), chest radiograph, electrocardiograph (ECG), and echocardiographic findings [left ventricular ejection fraction (LVEF)]. The details of treatment in the form of calcium and 25(OH) D3 supplementation, mechanical ventilation, and the need for vasoactive support were noted. The mortality, duration of PICU and hospital stay, and outcome at follow-up were also noted.

Definitions

Vitamin D deficiency was defined as serum 25(OH)D3 levels <20 ng/mL.[6,10,11] Left ventricular ejection fraction was classified as normal (> 55%), mildly reduced (40–55%), moderately reduced (30–40%), and severely reduced (< 30%).[26]

Statistical Analysis

The data entry and statistical analysis were performed using Microsoft Excel 2007 (Microsoft, Redmond, WA, USA) and SPSS software version 15 (SPSS, Inc., Chicago, IL, USA). The data variables were described as number, percentages, and median (IQR).

Results

During the study period, a total of 3956 children (aged 1 month–12 years) were admitted to the PICU, and out of them, 28 (0.71%) had DCM. Out of these 28 children, 14 (50%) had DCM associated with VDD.

The median (IQR) age at presentation was 6 (2–9) months, all were infants, and 71% (n = 10) were males. All were exclusively breastfed and none of them were receiving vitamin D or calcium supplementation. The common modes of presentation included respiratory symptoms (distress or failure) (78.6%, n = 11) for a duration of 13.5 (11.7–22.7) days, congestive cardiac failure (71.4%, n = 10), cardiogenic shock (37.5%, n = 5), and neurological manifestations (seizures and encephalopathy in 14.3% each) (Table 1). There were no clinical signs of any dysmorphism, structural heart disease, severe anemia, malnutrition, or hypothyroidism in any of the cases.

Table 1: Baseline characteristics and clinical presentation of children with dilated cardiomyopathy associated with vitamin D deficiency

Characteristics	*Total cases, n = 14*
Age (in months), median (IQR)	6 (2–9)
Males, *n* (%)	10 (71)
Weight (kg), median (IQR)	6 (4–7.6)
Duration of illness (days), median (IQR)	13.5 (11.7–22.7)
Cough, *n* (%)	6 (42%)
Fever, *n* (%)	2 (14.2)
Respiratory distress or failure, *n* (%)	11 (78.6)
Congestive heart failure, *n* (%)	10 (71.4)
Cardiogenic shock, *n* (%)	5 (37.5)
Seizures, *n* (%)	2 (14.3)
Encephalopathy, *n* (%)	2 (14.3)
AKI, *n* (%)	2 (14.3)

Twelve out of 14 had hypocalcemia with median (IQR) ionized calcium levels of 0.7 (0.7–1.1) mmol/L and total calcium of 8.7 (7–9.5) mg%. All cases had elevated serum alkaline phosphatase 343 (316–415) IU/L, high PTH levels 115 (66–228) pg/mL, and low 25(OH)D3 levels 5 (3–7) ng/mL. All cases had reduced LVEF (<30%) at admission with median (IQR) of 22 (17–25)%. Left ventricular ejection fraction was severely reduced (<30%) in the majority of children [92.9% (n = 13)], and only one (7.1%) had moderately reduced (31–40%) LVEF (Table 2). None had any echocardiographic evidence of structural heart disease or coronary artery abnormalities.

All cases received vitamin D supplementation [25(OH)D3], parenteral in 57.1% and oral in 42.9%) and calcium supplementation (50–100 mg/kg/day). Activated vitamin D [1, 25(OH)D3] was administered in 35.7% (n = 5) cases, and 35.7% (n = 5) required intravenous infusion of calcium (Table 3).

The PICU needs included high-flow nasal cannula oxygen therapy (14%, n = 2), mechanical ventilation (35.7%, n = 5), and vasoactive drugs (57.1%, n = 8). The most commonly used vasoactive drugs were milrinone (57.1%), adrenaline (28.6%), and dobutamine (21.4%). One child developed healthcare-associated infections (HCAI) in the form of catheter-associated urinary tract infections (CAUTI). There was no mortality. The duration and PICU and hospital stay was 76 (31–98) hours and 6 (4.7–10) days, respectively (Table 3). All children were discharged on calcium and vitamin D supplements.

Ten (71.4%) children were followed till 10 (7–58) months after discharge, and all were asymptomatic, had normal LEVF [55 (55–60)%], and normal pediatric overall performance category score. Only one child had residual moderate mitral regurgitation (Table 3).

Discussion

In this retrospective study, infants with DCM associated with VDD presented with respiratory distress, CCF, cardiogenic shock, and seizures. All had low LVEF, hypocalcemia, and VDD. All showed

Table 2: Baseline laboratory investigations in children with dilated cardiomyopathy associated with vitamin D deficiency

Parameters	*Total cases (n = 14)*
Blood glucose (mg%), median (IQR)	92 (85–104)
Lactate (mmol/L), median (IQR)	3.2 (2.6–4.2)
Ionized calcium (mmol/L), median (IQR)	0.7 (0.7–1.1)
Total calcium (mg%), median (IQR)	8.7 (7–9.5)
Phosphate (mg/dL), median (IQR)	3.5 (2.6–4.5)
Alkaline phosphatase levels (IU/L), median (IQR)	343 (316–415)
25(OH)D3 levels (ng/mL), median (IQR)	5 (3–7)
PTH levels (pg/mL), median (IQR)	115 (66–228)
Serum aspartate transaminase (U/L) median (IQR)	59 (37–272)
Serum alanine transaminase, (U/L), median (IQR)	44 (23–180)
CK-MB (IU/L), median (IQR)	42 (34–54)
Hb (gm/dL), median (IQR)	8.8 (8.0–9.8)
Total leukocyte count (per cumm), median (IQR)	13,900 (9552–15,100)
Platelet count (units), median (IQR)	3,52,500 (2,30,250–4,14,000)
Urea, (mg%), median (IQR)	34 (21.7–55)
Creatinine (units), median (IQR)	0.3 (0.2–0.5)
Left ventricular ejection fraction (%) at admission, median (IQR)	22 (17–25)
LVEF <30%, *n* (%)	13 (92.9)
LVEF 31–40%, *n* (%)	1 (7.1)

Table 3: Treatment details, intensive care needs, and outcome of children with dilated cardiomyopathy associated with vitamin D deficiency

Characteristics	*Total cases (n = 14)*
Intravenous calcium infusion, *n* (%)	5 (35.7)
Vitamin D supplementation, *n* (%)	14 (100)
Parenteral, *n* (%)	8 (57.1)
Oral, *n* (%)	6 (42.9)
Activated vitamin D, *n* (%)	5 (35.7)
Oxygen therapy, *n* (%)	14 (100)
HFNC, *n* (%)	2 (14)
Invasive mechanical ventilation, *n* (%)	5 (35.7)
Duration of mechanical ventilation (hours), median (IQR)	41 (27–107)
Vasoactive drug requirement, *n* (%)	8 (57.1)
Milrinone	8 (57.1)
Epinephrine	4 (28.6)
Dobutamine	3 (21.4)
Required > 1 vasoactive drugs, *n* (%)	5 (35.7)
Renal replacement therapy, *n* (%)	0 (0)
PICU mortality, *n* (%)	0 (0)
Length of PICU stay (hours), median (IQR)	76 (31–98)
Length of hospital stay (days), median, (IQR)	6 (4.7–10)
Follow-up (*n* = 10)	
Long term follow-up (months), median (IQR)	10 (7–58)
Left ventricular ejection fraction at last follow-up, median (IQR)	55 (55–60)

dramatic improvement in clinical status and LVEF after therapeutic doses of vitamin D and calcium. The long-term outcome was good. The causes for hypocalcemia and VDD in infants could be due to lack of calcium and vitamin D supplementation to mother and child, maternal VDD, and exclusively breastfeeding. The underlying cause for myocardial dysfunction and DCM was possibly hypocalcemia due to VDD as suggested by clinical features, laboratory investigations, and rapid response to supplementation with vitamin D and calcium.

There are few reports highlighting hypocalcemia and VDD as treatable cause of DCM. Tomar et al.[1] noted that among 94 children with severe left ventricular dysfunction, 16% (*n* = 15) had hypocalcemia. Among these 15 infants (median age of 2 months, range 45–5 months), 12 presented with CCF, 3 had CCF with shock, and 7 also had seizures. All had cardiomegaly on chest radiograph, tachycardia on ECG, low serum calcium, high alkaline phosphatase, high PTH, and low vitamin D levels in 6 children; and echocardiography revealed dilated left ventricle, severely decreased LVEF and fractional shortening (FS), normal coronaries, and no structural heart defect. Vitamin D deficiency was noted in six cases, and the mother of only one case had VDD. Treatment with decongestive measures and supplementation with vitamin D and calcium lead to improvement and normalization of LVEF over 8–12 weeks. Sanyal and Raychaudhuri[3] described 12 infants (median age 4 months and range 38 days–11 months) who presented with CCF and DCM. All had cardiomegaly, hypocalcemia, low vitamin D, high alkaline phosphatase, high PTH, and low maternal vitamin D (except in one). 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 the next 3 months. Elidrissy et al.[24] conducted a review of 61 infants with hypocalcemic cardiomyopathy (mean age 5 months, range 1 month–15 months). All cases presented with heart failure and had hypocalcemia, high alkaline phosphatase, low vitamin D, and high PTH; and echocardiology was suggestive of cardiomyopathy. Most of these cases responded well to supplementation with calcium and vitamin D, cardiotonic, and diuretics. In another observation, a case–control study by Aryafar et al.,[27] it was demonstrated that among children with DCM, 27.3% had VDD as compared with the control group (8.6%). Recently, few other authors also reported infants with DCM due to hypocalcemia and VDD. These studies highlighted that hypocalcemia and VDD are important treatable causes of myocardial dysfunction, and DCM and supplementation with vitamin D and calcium resulted in rapid recovery of myocardial functions.

Vitamin D deficiency is the main cause for hypocalcemia in infants and children. The prevalence of VDD is high in our country, and possible reasons are inadequate dietary sources of vitamin D, inadequate sunlight exposure, indoor lifestyle, high levels of skin pigmentation, and lack of vitamin D supplementation.[6,10,11] In infants, maternal VDD could be a contributing factor for developing hypocalcemia, especially in exclusively breastfed babies and those not on vitamin D supplementation.[2] Infants born to mothers with VDD are at risk of early and fatal squeals of VDD and hypocalcemia.[3] Therefore, routine vitamin D supplementation for pregnant mothers and infants should be stressed to prevent serious complications due to VDD and hypocalcemia.[28,29] Any child presenting to pediatric emergency with features suggestive of DCM should be evaluated for VDD as this condition is not only uncommon, but it is completely

reversible without significant residual myocardial dysfunction. In a descriptive cross-sectional single-center study by Raafat et al.[30] involving children with idiopathic DCM (n = 44), it was noted that 90.9% had VDD with mean levels being 13.5 ng/mL. They documented that lower vitamin D levels significantly correlated with the degree of left ventricular dysfunction.

Children with DCM secondary to VDD may require PICU admission for hemodynamic monitoring, vasoactive drugs, ventilation, and supportive care. With vitamin D and calcium supplementation and optimum organ supportive care, the short- and long-term outcomes of DCM secondary to VDD are usually excellent.

The strengths of this study include demonstration of the association between VDD and DCM/myocardial dysfunction. The limitations of this study include retrospective study design and lack of vitamin D levels in the follow-up. Also, not all the infants were worked up for other etiologies like acute viral myocarditis, nutritional deficiencies (selenium, beriberi), metabolic/inborn errors of metabolism, etc. due to limited availability. At this stage, we can say that VDD was associated with DCM in these infants. However, we must remember that VDD could be an important treatable and reversible cause of DCM/myocardial dysfunction.

Conclusion

Vitamin D deficiency and the resultant hypocalcemia are common and important treatable and reversible causes of DCM and should be considered in infants with DCM, especially in regions where VDD is common. Supplementation with vitamin D and calcium can lead to rapid recovery of myocardial function. Since VDD remains a major public health problem with serious consequences in infants born to mothers with low vitamin D stores, routine vitamin D supplementation in pregnant females and infants should be stressed.

Author's Contributions

SK: Data collection, data analysis, and preparation of initial draft of the paper; SKA: Conceptualized the study, data analysis, and finalized the paper; KN: literature review and data analysis; AB: Supervised the data collection and analysis and finalized the paper; MJ: Supervised the study and critically reviewed the paper. All the authors approved the final manuscript.

Orcid

Surjeet Kumar https://orcid.org/0000-0001-8727-0270
Manjinder Singh Randhawa https://orcid.org/0000-0001-7998-9170
Suresh Kumar Angurana https://orcid.org/0000-0001-6370-8258
Karthi Nallasamy https://orcid.org/0000-0002-6713-9846
Arun Bansal https://orcid.org/0000-0001-6212-6889
Manoj Rohit Kumar https://orcid.org/0000-0001-5866-2742
Naresh Sachdeva https://orcid.org/0000-0001-6289-4749
Muralidharan Jayashree https://orcid.org/0000-0002-6149-1355

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