Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 16s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Rare case of dilated cardiomyopathy in case of severe vitamin D deficiency

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Cite this paper as: Dr. Karthik D, Dr. Richa, Dr. Nidhish Raval, Dr. Vineeta Pande, Dr. Renuka Jadhav, Dr. Shailaja Mane, (2025) Rare case of dilated cardiomyopathy in case of severe vitamin D deficiency. *Journal of Neonatal Surgery*, 14 (16s), 276-278.

ABSTRACT

Dilated cardiomyopathy (DCM) in infants is a rare but potentially reversible condition when caused by hypocalcemia, most often secondary to vitamin D deficiency. This case report presents a 40-day-old exclusively breastfed female infant who developed non resolving cough and was found to have DCM with significantly decreased left ventricular function. Biochemical investigations revealed low levels of vitamin D and calcium, while parathyroid hormone (PTH) levels remained normal. A similar vitamin D deficiency was identified in the mother. Upon administration of calcium and vitamin D supplementation, the infant's cardiac function improved markedly within a week, with full recovery confirmed by echocardiography at three months. This case underscores the importance of early recognition and treatment of hypocalcemia-induced cardiomyopathy, especially in infants born to vitamin D-deficient mothers. Proactive maternal and infant vitamin D supplementation could prevent such life-threatening manifestations.

1. INTRODUCTION

An estimated 1.13 incidences of dilated cardiomyopathy (DCM) occur for every 100,000 children. Less than half of these kids get the primary etiology diagnosed, although it greatly enhances their prognosis [1]. DCM is primarily idiopathic, although in certain instances, where the cardiac contractility deficit is irreversible, metabolic and infectious factors have been found to be responsible [2]. One known reversible cause of dilated cardiomyopathy is hypocalcemia [3,4,5]. Changes in intracellular calcium concentration are necessary for the function of cardiac myocytes [4]. Following an increase in intracellular calcium concentration via calcium channels, calcium is released from the sarcoplasmic reticulum, binds to the troponin–tropomyosin complex, and stimulates actin and myosin to bind to one another [5]. Thus, cardiac myocyte action is disrupted by intracellular hypocalcemia. Other processes, some of which are yet unclear, are in charge of the cardiomyopathy associated with hypocalcemia [6]. Hypoparathyroidism (86%), vitamin D insufficiency (5%), pseudohypoparathyroidism (2%), and celiac disease (2%) are the main causes of hypocalcemia, which leads to hypocalcemia-related cardiomyopathy [7]. The most often documented cause of hypoparathyroidism is primary hypoparathyroidism, which is followed by hypoparathyroidism resulting after surgery. The ability to reverse cardiomyopathy caused by hypocalcemia with appropriate treatment that includes calcium and vitamin D replenishment is its most significant therapeutically relevant characteristic [8].

2. CASE REPORT

A 40 day old female child was brought to the tertiary care centre with complaint of cough since last 15-20 days. Cough was insidious in onset, progressive in nature and associated with post tussive vomiting. There was no history of fever, difficulty in breathing, bluish discoloration, or difficulty in feeding. There was no history of suck rest suck cycle. Baby was second born child out of a non consanguineous marriage, delivered via normal vaginal delivery with birth weight of 3 kg at term gestation and cried immediately after birth. Exclusive breast feeding was initiated in delivery room. All antenatal scan along with anomaly scan and fetal 2d echo did not show any abnormality. On admission baby had mild subcostal retraction with systolic murmur and bilateral basal crackles. Chest x ray showed increased bronchovascular markings with cardio thoracic ratio of 0.68. On blood investigation, haemoglobin level was 11.3 gm/dl, other parameters were normal in range. The NT-proBNP level was significantly high (2882.40 pg/ml) with increased Troponin I level (18.30) . Echocardiogram showed dilated dysfunctional LV with LV ejection fraction of 40%. Baby's metabolic screening test was done to find out cause of dilated cardiomyopathy, showed no abnormality. on further evaluation vitamin D level was significantly low (3.5ng/ml) with reduced serum calcium level (8.40 mg/dl) and ionised calcium level (0.93mmol/L). Baby had normal serum PTH level of 47 pg/ml. Since the baby was on exclusive breast feeding, we checked vitamin D level of mother which was significantly low (16.20 ng/ml) with normal serum calcium level (9.50 mg/dl) and normal serum PTH level (27.60 pg/ml). Baby was started on vitamin D supplementation and calcium supplementation. With in 7 days LV ejection fraction was improved to 60%. Repeat echocardiogram after 3 months showed resolution of DCM.

3. DISCUSSION

Myocardial contraction coupling depends critically on calcium, and hypocalcemia impairs myocardial function. Although hypocalcemia-induced cardiomyopathy is uncommon and has been documented in a small number of cases, congestive heart failure brought on by hypocalcemia has been documented [3, 4]. DCM-causing hypocalcemia can be reversed, and full recovery occurs after serum calcium levels return to normal. The primary cause of hypocalcemia in newborns and older children is a vitamin D deficiency. Vitamin D insufficiency in breastfed newborns and children is the main cause of nutritional rickets, which is still common[5]. A key factor in controlling cardiac contraction is ionized calcium. Ionized calcium enters cells through depolarization-activated calcium channels when the cardiac action potential is triggered. The sarcoplasmic reticulum (SR) releases calcium in response to ionized calcium that is entered. The heart contracts when Calcium binds to myofilaments proteins like troponin C. Myocardial contractility can be altered in two primary ways. One involves changing the Ca2+ transient's amplitude or duration, and the other involves changing the myofilaments's sensitivity to Ca2+. Thus, a change in the amplitude or length of the Ca2+ transient results in hypocalcemia-induced DCMP [8, 9]. In India, vitamin D deficiency is still a serious public health issue, particularly for exclusively breastfed infants and those born to mothers who have high-risk characteristics like low vitamin D stores, dark skin, and/or sedentary lifestyles, which further reduces exposure to enough UV light [10–12]. Hypocalcemic vitamin D insufficiency can cause early and deadly squeals in infants born to moms who are vitamin D deficient [13–15]. According to recent recommendations from the Canadian Pediatric Society (CPS) and the American Academy of Pediatrics (AAP), infants who are not exposed to enough sunlight or whose mothers are vitamin D deficient should consume 400 IU of vitamin D per day starting shortly after birth, instead of the previous recommendation of 200 IU per day starting in the first two months. Until the baby is weaned to at least one liter of vitamin D-fortified formula per day, the supplement should be continued. Additionally, a healthy, balanced diet and relatively high-dose maternal vitamin D supplementation (2000 IU/day) are required [16, 17].

4. CONCLUSION

With greater suspicion, hypocalcemia-induced DCM can be diagnosed, and early treatment can improve patient outcomes and restore heart function. Vitamin D deficiency is still a serious health issue in children, and particularly for mothers who already have inadequate vitamin D levels as a result of risk factors related to culture, diet, or lifestyle.

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