

Vitamin D deficiency presenting with cardiogenic shock in an infant

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ABSTRACT

A 2-month-old child was referred as a case of dilated cardiomyopathy with cardiogenic shock. On evaluation, hypocalcemia secondary to severe vitamin D deficiency was found. There were no clinical or radiological features of rickets. The child had developed multiorgan failure due to cardiogenic shock at the time of admission and could not be saved despite adequate ventilatory and pharmacologic support. Hypocalcemia should be considered as an important differential diagnosis in cases of dilated cardiomyopathy in infants.

Keywords: Cardiogenic shock, dilated cardiomyopathy, hypocalcemia, vitamin D deficiency

INTRODUCTION

Vitamin D deficiency resulting in hypocalcemia and rickets is a major problem in developing and developed countries.^[1] Majority of children present with symptomatic hypocalcemia (including seizures, tetany, laryngospasm, etc.) and/or with clinical features of rickets. Although hypocalcemia secondary to vitamin D deficiency is an important cause of dilated cardiomyopathy in infants, cardiogenic shock as a presenting feature without clinical or radiological features of rickets is very rarely reported in the literature.^[2] The present communication describes a 2-month-old child presenting with cardiogenic shock secondary to hypocalcemia due to severe vitamin D deficiency.

CASE REPORT

A 2-month-old child was referred to the hospital with a diagnosis of dilated cardiomyopathy. According to the mother, the child was well till 2 days back, when he developed rapid breathing without fever or cough. X-ray chest revealed cardiomegaly. Echocardiography was advised by the attending pediatrician, which showed marked left ventricular hypokinesia, with ejection fraction of 20%. Left ventricular end diastolic diameter was 38 mm, end systolic diameter was 35.1 mm and fractional shortening was 7.6%.

The patient was first in birth order, delivered normally at term to non-consanguineous hindu parents. Birth weight was not known. The mother had not received adequate antenatal care, including iron and calcium supplementation. The mother's serum calcium, phosphorous and alkaline phosphatase levels were within the normal limit (9.5 mg/dl, 3.4 mg/dl and 264 IU/L, respectively), but serum vitamin D₃ level could not be determined. He was mainly breastfed and, past history was unremarkable. Anthropometry at admission was weight – 4.0 kg (5th percentile), length – 55 cm (5th percentile) and head circumference – 38 cm (5th percentile). There was no clinical or radiological evidence of rickets.

On admission, the child was very sick, with gasping respiration, feeble pulses, cold extremities, HR – 210/min, BP was not recordable and SPO₂ – 76%. The chest was full of coarse crepitations. Cardiovascular system examination revealed faint first and second heart sounds, gallop rhythm and no murmur. Liver was palpable 6 cm below the subcostal margin. Clinical examination was suggestive of congestive cardiac failure with shock. He was immediately transferred to the pediatric intensive care unit and put on a mechanical ventilator.

As a part of the initial management, inj. Lasix was given, followed by Dobutamine infusion and Digoxin (half the recommended dose). Possibility of hypocalcemia as a cause of cardiomyopathy was entertained and samples for calcium, serum Parathormone (PTH) and 25-OH vitamin D were withdrawn. Injection calcium gluconate 10% was given as IV bolus (200 mg/kg over 10 min) followed by continuous infusion (800 mg/kg/day). Injection vitamin D (cholecalciferol) was given intramuscularly (6,00,000 IU). As a part of cardiogenic shock management, inj. Milrinone was started to reduce peripheral vascular resistance and increase cardiac contractility.

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The initial blood investigation report revealed total serum calcium of 3.0 mg/dl, ionized calcium – 0.4 mmol/L, phosphorous – 2.1 mg/dl and alkaline phosphatase – 1040 IU/L. Complete blood count was within normal limits and C-reactive protein was not raised. Initial blood urea and serum creatinine level were deranged (67 mg/dl and 1.3 mg/dl, respectively). The serum magnesium level was normal (1.9 mg/dl). ECG did not reveal any evidence of arrhythmia, although the QTc interval was prolonged (0.48 s). Further doses of Digoxin were not given in view of deranged renal function.

After 2 h, his condition stabilized, BP recorded as 64/32 mm/Hg and SpO₂ was 96%, but the urine output remained <0.5 ml/kg/h. After 16 h of admission, the repeat blood urea and serum creatinine levels were further deranged to 130 mg/dl and 1.7 mg/dl, respectively. Repeat ionized serum calcium improved (0.6 mmol/L), but did not normalize. Serum PTH was high, 550 pg/ml, (normal – 14–72 pg/ml) and 25-OH vitamin D level was very low, <1.5 ng/ml (normal – 25–40 ng/ml). Based on the above laboratory findings, the diagnosis was made as dilated cardiomyopathy and cardiogenic shock with multiorgan failure due to hypocalcemia secondary to severe vitamin D deficiency.

Treatment was continued with inotropes, inodilator and calcium gluconate infusion. His condition remained same for another 24 h, after which he started deteriorating, with fall in blood pressure and oxygen saturation. The urine output further decreased and he finally succumbed to shock with multiorgan failure after 40 h of admission.

DISCUSSION

Ionized calcium plays an important role in cardiac excitation contraction coupling, and reduction in the ionized calcium level may affect ventricular contraction.^[3] There are isolated case reports of hypocalcemic-dilated cardiomyopathy resulting in heart failure due to nutritional vitamin D deficiency.^[2,4-7] Case series of dilated cardiomyopathy secondary to vitamin D deficiency and hypocalcemia have been reported in exclusively breastfed African American infants, ethnic minority population of southeast England and Indian children.^[8-10] However, cardiogenic shock as a presenting feature secondary to severe hypocalcemia due to hypovitaminosis D and without clinical features of rickets in a 2-month-old child has not been reported earlier.

Vitamin D deficiency is seen commonly due to inadequate nutrition and insufficient exposure to sunlight. Exclusive breastfeeding without adequate sun exposure or vitamin D supplementation is an important risk factor for hypocalcemia due to vitamin D deficiency. In a study from Chennai, 13 exclusively breastfed infants with hypocalcemic seizure were found to have a low serum vitamin D₃ level. Nutritional status of all the infants

was normal, and none of them had received vitamin D supplementation. All mothers had biochemical evidence of vitamin D deficiency.^[11]

Our case was unique as the duration of illness was very short and he did not develop other features of severe hypocalcemia, like seizure or apnea, before going into shock. Also, there was no radiological evidence of rickets. Vitamin D deficiency can occur in infants and children without radiological evidence of rickets because of the increased metabolic demands due to rapid growth, resulting in hypocalcemia before any radiological changes could occur.^[12] Although the attending pediatrician was very quick in making the diagnosis of dilated cardiomyopathy and early referral, hypocalcemia was not considered as an etiology. Earlier case reports have shown good prognosis, with full recovery of cardiac function 6–12 months after presentation. Our case had developed multiorgan failure due to cardiogenic shock by the time he reached the hospital, and he could not be saved despite appropriate ventilatory and pharmacologic support. Failure to respond to therapy may be explained by late referral and delay in treatment with calcium.

The possibility of myocarditis cannot be ruled out in this case, but the alterations in calcium and parathormone levels were severe suggesting to us that hypocalcemia played a major role in his illness.

To conclude, hypocalcemia secondary to hypovitaminosis D should always be considered as a possibility in cases of dilated cardiomyopathy, especially in regions where vitamin D deficiency rickets are common. All breastfed and partly breastfed babies (consuming <1000 ml of vitamin D-fortified formula milk/whole milk) should be supplemented with vitamin D 400 IU/day.^[13]

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