

## Case Report

# Maternal hypovitaminosis D: a cause of neonatal hypocalcemic seizures

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**Received:** 27 April 2017

**Revised:** 17 July 2017

**Accepted:** 24 July 2017

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### ABSTRACT

Neonatal late onset hypocalcemia is the one which occurs after 72 hours of life. 1,25 (OH)<sub>2</sub>- vitamin D and Parathormone (PTH) play crucial role in regulation of calcium and phosphorus homeostasis in the body. Vitamin D helps in the absorption of calcium and phosphorus from the gut. PTH promotes absorption of calcium from thick ascending loop of henle and distal tubule whereas it increases excretion of phosphorus. During hypocalcemia, parathormone level shoot up in the body to maintain normal calcium levels. In a patient with poor resources of vitamin D, there is disturbance of Calcium, phosphorus homeostasis leading on to clinical manifestations. Exclusively breast-fed infants without vitamin D supplementation and infants born to mothers with Vitamin D deficiency are at risk to develop Hypovitaminosis D and manifest symptoms of hypocalcemia.

**Keywords:** Hypocalcemia, Maternal hypovitaminosis D, Neonatal seizures, Vitamin D deficiency

### INTRODUCTION

Vitamin D deficiency has been an increasing global interest in health and disease.

Prevention of vitamin D deficiency and achieving adequate intake of vitamin D and calcium throughout childhood may reduce the risk long-latency disease processes that have been associated with vitamin D-deficiency states in adults.

In countries with high prevalence of vitamin D deficiency, maternal vitamin D supplementation during pregnancy and early supplementation of vitamin D to newborns should be considered to avoid hypocalcemia and skeletal abnormalities in the newborns and growing infants.

These cases are the babies admitted in a Level III NNF-accredited Neonatology department in India.

### CASE REPORT

#### Case 1

32days old baby, born to non-consanguineous parents, thriving well, on exclusive breastfeeding, not on micronutrient supplementation, was admitted with convulsions and lethargy. Baby was afebrile, euglycemic, had multiple convulsions. Investigations revealed normal blood count and CSF analysis. Chest xray, Echocardiography and CT brain were normal. Metabolic workup showed hypocalcemia with Serum calcium of 5.6mg/dl and ionised calcium of 0.5mmol/l, serum phosphorus of 6.3mg% and Serum Magnesium of 1.1mg/dl. Baby was treated with 10% calcium gluconate and 50% magnesium sulphate. Further investigations revealed Vitamin D of 10ng/ml and iPTH levels of 9pg/ml (Table 1). Maternal investigations showed Hypovitaminosis D with level 12ng/ml with normal iPTH. The diagnosis of Congenital Hypoparathyroidism

was made. Baby was supplemented with therapeutic dose of Vitamin D, oral calcium and magnesium. Baby remained seizure free, discharged and doing well on follow up.

**Case 2**

35days old baby with uneventful neonatal period, on exclusive breast feeding and not on vitamin D

supplements, was brought with convulsions and noisy breathing.

Baby was afebrile, euglycemic. Investigations revealed Serum calcium of 6mg/dl and ionised calcium of 0.7mmol/L, Serum magnesium, phosphorus, Chest Xray, CSF analysis and CT Brain were normal. Next investigations showed Vitamin D levels of 13ng/ml and normal iPTH (Table 1).

**Table 1: Investigations related to hypocalcemia in all 3 cases.**

Investigation	Case 1	Case 2	Case 3	Normal values
Serum total Calcium / Ionised	5.6mg/dl/0.5mmol/L	6mg/dl /0.7 mmol/L	5mg/dl/0.6mmol/L	8.5-10.5mg/dl 1.1-1.35mmol/L
Sr. Magnesium	1.1 mg/dl	1.7mg/dl	1.9 mg/dl	1.7-2.4 mg/dl
Serum Phosphorus	6.3mg/dl	4.5mg/dl	4.2mg/dl	2.5-4.5 mg/dl
25-OH- vitamin D	10ng/ml	13ng/ml	11ng/ml	20-50 ng/ml
i-parathormone	9 pg/ml	33pg/ml	43pg/ml	10-65 pg/ml
Urine calcium creatinine ratio	0.18	0.16	0.2	< 0.2
Maternal Vitamin D	12ng/ml	18ng/ml	15ng/ml	20-50 ng/ml

Maternal Vitamin D level was assessed, was low (18ng/ml). Baby was treated with oral vitamin D and calcium. Mother was supplemented with the same. Repeat calcium was normal, the baby was discharged and on follow up.

**Case 3**

9 days old baby, on exclusive breastfeeding was admitted with poor feeding, lethargy and convulsions. Baby was febrile, dehydrated, but euglycemic. Investigations revealed Hypocalcemia with serum levels of 5mg/dl, ionised calcium of 0.6mmol/L, with hypernatremia and prerenal azotemia (Na+ 165mEq/L, Urea-120mg% and creatinine 0.8mg%). CSF analysis, sepsis screening, CT brain were normal. Baby was given fluid correction and calcium supplementation. Repeat workup showed persistent hypocalcemia with Serum ionised Calcium of 0.5mEq/l with normal Sodium, Magnesium and Phosphorus. Second line investigations revealed Vitamin D levels of 11ng/ml and normal iPTH (Table 1). Maternal blood levels of Vitamin D were also deficient (15ng/ml). Baby was supplemented with oral Vitamin D and calcium. Mother was given oral supplementatation. Baby remained seizure free and was discharged.

**DISCUSSION**

There is a global discussion regarding vitamin D in health and disease. Prevention of vitamin D deficiency reduces the risk of diseases that have been associated with it. The main source of vitamin D is from UV-B light. Maternal vitamin D concentrations determine the vitamin D status of the fetus and the newborn.<sup>1</sup> Adequate vitamin D status

during pregnancy is important for fetal growth and development. Infants who are exclusively breastfed and who do not receive supplemental vitamin D are at risk of developing vitamin D deficiency. Intake of vitamin-D supplement by the infant, sunlight exposure and maternal 25OH-D levels were found to have positive correlation with the infants' 25OH-D.<sup>2</sup> Breast milk has very low levels of vitamin D which leads to vitamin D deficiency in babies who are exclusively breastfed for a prolonged period of time.<sup>3</sup>

Vitamin D deficiency presents as symptomatic hypocalcemia or like that of rickets and/or decreased bone mineralization.<sup>4</sup> Vitamin D deficiency results in hypocalcaemia, secondary hyperparathyroidism, hypophosphataemia and elevated alkaline phosphatase. Serum 25-OHD concentration provides the best indication of vitamin D status.<sup>5</sup>

Vitamin D deficiency is the most common cause of hypocalcaemia after the first 3 days of life.<sup>6</sup> High-dose vitamin D therapy with 1000-5000 IU/day for 3 months is preferred for less than 12 months old. Stoss therapy is effective for treating vitamin D deficiency.<sup>7</sup> in patients with poor compliance. It involves oral or intramuscular administration of vitamin D as 300 000 IU to 500 000 IU, as a single dose, or two to four divided doses for 3months.

All pregnant women should have their serum 25-OHD concentration evaluated during the first trimester.<sup>8</sup> If they are severely vitamin D deficient, they should be treated with 3000–5000 IU daily until the serum 25-OHD concentration is over 20 ng/ml. After this serum

concentration is achieved, they should receive 400 IU daily, as should women with a mild deficiency. The American Academy of Pediatrics recommends supplementing all breastfed infants with vitamin D at a dose of 400 IU per day until weaning. Though many treating paediatricians follow this, there are good number of babies discharged without supplementation. Emphasis should be given to supplement Vitamin D deficient pregnant women and infants who are exclusively breast-fed even in tropical countries like India, to prevent hypovitaminosis D related complications to the growing brain.<sup>9</sup>

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: Not required*

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**Cite this article as:** Deepika G, Raghoji CR, Ashwini RC, Guruprasad G. Maternal hypovitaminosis D: a cause of neonatal hypocalcemic seizures. *Int J Contemp Pediatr* 2017;4:1900-2.