

A case report of hypervitaminosis D in 2 months old infant

Ahmadshah farhat^{*}, Ashraf Mohamadzadeh¹ Said Javad Sayedi², zeinab Nourbakhsh³, Mehrieh Rezaei⁴

1. Neonatal Research Center, Faculty of Medicine, Mashhad University of Medical Sciences (MUMS), Mashhad, Iran

2., Department of pediatric, Faculty of Medicine, Mashhad University of Medical Sciences (MUMS), Mashhad, Iran

3. Pediatric resident of Faculty of Medicine, Mashhad University of Medical Sciences (MUMS), Iran.

4. Intern of Faculty of Medicine, Mashhad University of Medical Sciences (MUMS), Mashhad, Iran.

Abstract

Introduction

Vit D level is low in human milk (20-60 IU/Lit) thus 400 Vit D is advised since birth for all infants. On the other hand upper limit dose for long term Vit D intake is 1000 IU for children less than one year of age. Excessive Vit D more than upper limit by physicians or parents can cause hypervitaminose D.

We report a rare case of hypervitaminose D in two months old infant.

Key words

vitamin D, I infant, intoxication

Background:

The concentration of vit D level is low in human milk (20-60 IU/Li), if breast feed infant not receiving vit D supplements, vit D stores could be depleted within 8 weeks (1), thus vitamin D has been added to milk in the United States since the 1930s to prevent rickets. On the other hand formulas designed for premature infants ("premature formulas"), which contain more calcium and vitamin D than standard formulas, are given to premature infants. The recommendation upper limit dose for long term vit D intake is 1000 IU for children less than one year age. Most cases of hypervitaminosis D during childhood are due to an excessive supplementation of vitamin D by physicians or parents, to prevent metabolic bone disease (2).

Excessive amounts of vitamin D results in signs and symptoms similar to those of

hypercalcemia. Symptoms which developed after 1-3 months of excessive intake include hypotonia, anorexia, irritability, constipation, polydipsia, polyuria and pallor. Hypercalcemia and hypercalciuria are notable. Proteinuria may be present and if excessive intake continues, renal damage occurs. Serum levels of 25(OH) D are a better indicator of hypervitaminosis D than 1, 25(OH) D because 25(OH) D has a longer half-life. Treatment includes discontinuing vitamin D intake and decreasing calcium intake. For severely affected infants, aluminum hydroxide can be given by mouth. Chelation therapy is rarely necessary.

We reported a rare case of hypervitaminosis D in infancy after premature formula feeding and Vit D supplementation.

*Corresponding Author: Ahmadshah Farhat

Assistant professor of Neonatology Neonatal Research Center, Faculty of Medicine, Mashhad University of Medical Sciences

Email: farhata@mums.ac.ir

Tell=+98-511-8521121

Fax=+98-511-8525316

Case introduction: A 50 days old female infant was referred to NICU of Emam Reza hospital Mashhad university medical sciences north east Iran, because of respiratory distress and hypotonia. The past history included low birth weight (birth weight 1750 gr length 45 cm and head circumference 31 cm) with cesarean section delivery because of fetal distress; she had been admitted because of anemia and high level of urea with a diagnose of birth asphyxia. Laboratory tests showed red blood cells count 2.7m/ml; hemoglobin level 6.9 mg/dl ; hematocrit 22.9 percent; MCHC 30 ;MCH 25.4 white blood cell count 4.2m/ml with lymphosytosis. Plasma creatinine was 1.4 mg/dl; BUN 11 mg/dl; serum calcium level 18.3 mg/dl; in 24 hours urine creatinine was 14 mg/dl and calcium was 11.45mg/dl. Infant's blood level of 25OH (D) was 75ng/dl which was higher than normal range. Because of hypercalcemia, calcium, phosphor, alkaline phosphates and 25OH (D) levels of parents were checked which were normal After more search in past history, it was find out the constant usage of premature infant formula and high dose vitamin D drop supplement (800 units per day).

Discussion:

Vitamin D plays an essential role in calcium homeostasis and the development and maintenance of the skeleton, is recommended for the prevention of rickets, optimization of peak bone mass, and prevention of bone loss. In addition, vitamin D has potential extra skeletal effects on the neuromuscular and immune systems. The two main sources of vitamin D are dietary intake and skin synthesis in response to exposure to ultraviolet B light (290–320 nm). Food sources of vitamin D in infants are breast milk or other milks and supplement of vitD. The amount of vitamin D synthesized in the skin varies by factors such as latitude, season, time of day, degree of skin exposure, use of sunscreen, and skin pigmentation or race. Previous estimates suggest that a single minimal erythmal skin dose of simulated sunlight will raise circulating levels of 25(OH)D comparable to ingestion of 10,000 to 25,000 IU of vitamin D3.(3) There are rare cases about hypervitaminosis D in

infancy. Jacobus and Holick have showed that Hypervitaminosis D associated with drinking milk. In this study all eight patients drank milk produced by a local dairy in amounts ranging from 1/2 to 3 cups (118 to 710 ml) daily. All had elevated serum 25-hydroxyvitamin D concentrations (mean $[\pm SD]$, 731 ± 434 nmol per liter [293 ± 174 ng per milliliter]). Six of the eight patients had elevated serum vitamin D3 concentrations. Of the eight patients, seven had hypercalcemia and one had hypercalciuria but normocalcemia (mean serum calcium, 3.14 ± 0.51 mmol per liter [12.6 ± 2.1 mg per deciliter]). Analysis of the dairy's vitamin D—fortified milk revealed concentrations of vitamin D3 (cholecalciferol) that ranged from undetectable to as high as 232,565 IU per quart (245,840 IU per liter). An analysis of the concentrate that was used to fortify the milk, labeled as containing vitamin D2 (ergocalciferol), revealed that it contained vitamin D3.(4) Nako and Fukushima also had a study about Hypervitaminosis D after prolonged feeding with a premature formula. Results of this study showed that concentrations of 25(OH) D in sera associated with sole feeding of premature infant formula ($n = 40$) were significantly higher than sera corresponding to regular formula or breast milk ($n = 25$; 175 versus 115 nmol/L, $P < 0.0001$). No sample showed a serum 25(OH)D concentration below 25 nmol/L. Of 65 samples, 49 (75.4%) showed 25(OH) D concentrations exceeding 100 nmol/L, but serum calcium and phosphorus concentrations were normal. Unexpectedly, urinary calcium correlated negatively with serum 25(OH) D.(5) In another study in Japan serum 25(OH) D was higher in premature infants who feed with optimal formula, although their serum calcium and phosphor levels were normal.(6) Ross in Canada in 2006 had done a study about Vitamin D intoxication in infancy. Four cases of hypervitaminosis D in infants has been presented, all of which showed the characteristic clinical picture. The radiological examination of the bones in two patients showed the typical appearance. The pathological study of the organs in one case demonstrated the phenomenon of metastatic calcification in

certain organs of the body as well as degenerative changes in the heart muscle. Clinical evidence of renal damage was present in two cases. That the condition is reversible was shown by the recovery of the first two patients. In the first patient, after a prolonged period of reparation the kidney functions and bone architecture returned to normal. (7) Dawodu A has showed that increased vit d level is common in summer in exclusively breast-feeding infants and their mothers. Thus season is an important factor on vitd level. (8) In our case respiratory distress , hypotonia and unexplained weight loss were the primary symptoms which followed by renal failure and anemia. The clinical presentation and increase calcium level with higher level of 25OH (D) which were different from reported cases.

Conclusion: Premature infants are in risk of deficiency and less likly over dosage of vit D. Hypervitaminosis D can be began with signs and symptoms like weight loss and renal failure. Supervision on Vit D supplement in preterm infants can help to solve the complicated cases which have signs of toxicity or overdose.

References:

1. Rigo J, Mhamed WM, Curtis M, Disorder of calcium and magnisium metabolism., Martin RJ, Fanaroff AA, Walsh MC, editors. Fanarrof and Martin' s Neonatal - Perinatl medicine disease of the fetus and infant. 9 th ed. St. Louis: Elsevier, 2011.p1523-1556
2. Greenbaum LA. Nutrition .In Kliegman RM, Stanton BF, Geme JW, Schor Nf, Behrman RE. Nelson Textbook of pediatrics. 19 th ed. Philadelphia: Elsevier, 2011,p200
3. Cranney A, Horsley T, O'Donnell S, Weiler H, Puil L, et all ,Effectiveness and safety of vitamin D in relation to bone health. Evid Rep Technol Assess (Full Rep). 2007 Aug;(158):1-235
4. Jacobus CH, Holick MF, Shao Q, Chen TC, Holm IA, Kolodny JM, Fuleihan GE, Seely EW. Hypervitaminosis D associated with drinking milk. N Engl J Med. 1992 Apr 30;326(18):1173-7.
5. Nako Y, Fukushima N, Tomomasa T, Nagashima K, Kurome T. Hypervitaminosis D after prolonged

feeding with a premature formula. Pediatrics. 1993 Dec;92(6):862-4.

6. Nako Y, Tomomasa T, Morikawa A. Risk of hypervitaminosis D from prolonged feeding of high vitamin D premature infant formula. Pediatr Int. 2004 Aug;46(4):439-43.

7. ROSS SG. Vitamin D intoxication in infancy; a report of four cases. J Pediatr. 1952 Dec;41(6):815-22.

8. Dawodu A, Agarwal M, Hossain M, Kochiyil J, Zayed R. Hypovitaminosis D and vitamin D deficiency in exclusively breast-feeding infants and their mothers in summer: a justification for vitamin D supplementation of breast-feeding infants. J Pediatr. 2003 Feb;142(2):169-7