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Mini Review: Assessment Hypervitaminosis and Hypovitaminosis D during Pregnancy and its Impact on Offspring Outcomes

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Abstract

Vitamin D is one of the most important compounds for human health, especially the skeletal and the immunological system. Vitamin D and pregnancy are linked together. Despite the numerous reports of the association on hypovitaminosis D with disease and health disturbance during pregnancy originating in part from the diet and from the transformation by exposure to sunshine. A great deal of the pregnant population suffers from vitamin D deficiency, especially during winter months and suffering from the present of several disorders, including cancer, hypertension, multiple sclerosis, rheumatoid arthritis, osteoporosis, muscle weakness, and diabetes. Pregnant mothers should get the recommended suitable amounts of vitamin D during pregnancy for both their own health and their baby development with not increase or decrease. The safety of using higher doses of vitamin D in various clinical settings in pregnancy and their offspring had to be discussed.

Keywords: *hypervitaminosis D; hypovitaminosis D; pregnancy; offspring*

Introduction

Vitamin D within your body is actually a hormone, not a vitamin(1). In fact, Vitamin D is a steroid vitamin from the fat-soluble pro hormones group. The body receives Vitamin D (from sunlight, food, or supplements), turns it into a hormonecalled activated Vitamin D or calcitriol (2). The body makes around 90% of the Vitamin D needs when the skin gets enough direct UV light from sunshine. The sunlight on the skin body produces a substance called cholecalciferol. This is then turned into calcidiol and then calcitriol by the liver and kidneys (3). Calcitriolis the active form of Vitamin D measured to assess the Vitamin D levels(4). The most important forms of Vitamin D in the human body are D3 and a lesser extent D2 (5) figure (1). Vitamin D is important to a huge number of functions of the body from supporting strong and healthy bones to maintaining your immune system(6).

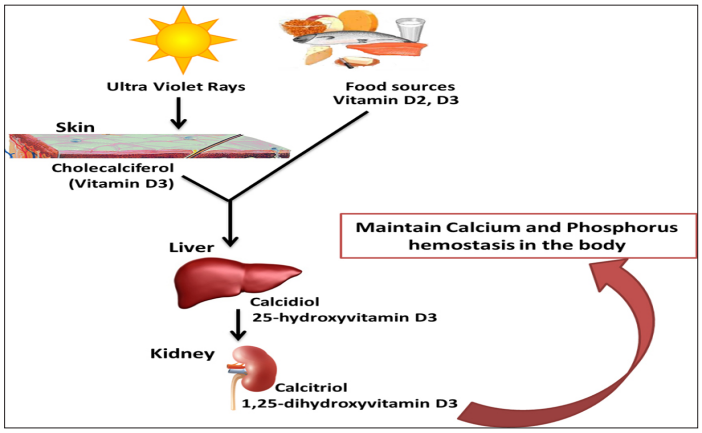

Fig 1. *Diagrammatic representation of vitamin D metabolism*

After taking Vitamin D supplement, the vitamin passes from bloodstream into the liver (7). Then, to the kidneys where it turns into calcitriol (8). Then released back into your bloodstream and can now enter your body's cells where it attaches to Vitamin D receptors (9) which working in regulating calcium and phosphorous minerals (10). Vitamin D receptors are present in a large variety of cell types, including myocytes, cardiomyocytes, pancreatic beta-cells, vascular endothelial cells, neurons, immune cells, and osteoblasts (2, 11). Vitamin D helps recycle calcium in the kidneys so bones can reabsorb it (12). Rest of Vitamin D excreted through urination(13). In intestines Vitamin D helps the body to absorb the calcium from the healthy diet and from any calcium supplements (14).The purposes of this article are to examine the risk assessment of vitamin D for pregnant health.

Hypovitaminosis and Hypervitaminosis D during Pregnancy

Vitamin D deficiency or insufficiency are widespread globally. Many studies reveal the high prevalence of vitamin D in women, antenatal and lactating mothers (15-17). There are many causes of hypovitaminosis D generally, they can be divided into two reason UVB-related deficiency and medical/physical condition-related deficiencies (3). Hypovitaminosis D is associated with increased parathyroid secretion, increased bone turnover, osteoporosis, osteomalacia and increased risk of bones fractures (18). Hypovitaminosis Discommon towomen, including menopausal, post menopausal, maternal and lactating mothers (19). Vitamin D requirements are probably greater in pregnancy for enhancing intestinal calcium absorption and enhancingt fetal requirement of calcium (20).The relationship between Hypovitaminosis D and adverse maternal outcomes such as pregnancy induced gestational hypertension and/or gestational diabetes mellitus, pregnancy loss, preterm delivery, primary Caesarian section, and postpartum depressionhas been documented in recent years (21, 22). For detection of vitamin D deficiency in pregnancyserum alkaline phosphatebecause of the placental secretion of this enzyme (23). Hydroxy vitamin D levels may be measured in each trimester with screening for hypercalcemia (24). With so many factors putting us at the risk of hypovitaminosis D, it makes sense to look at ways to supplement.

According to the **Food and Nutrition Board (25)** the standard dosing regimen of Vitamin D for pregnant women is 400 IU/day and upto 2000 IU/day. Hypervitaminosis D results when pharmacologic doses of vitamin D are consumed for a prolonged period of time and is defined by a large increase in circulating 25(OH)D concentrations that can result inhypercalciuria, hypercalcemia, and extraskeletal calcification (26). On the other hand, **Hollis and Wagner (27)** found that supplementation of 4,000 IU Vitamin D /day for pregnant women(12–16 weeks' gestation) was safe for theirs and their neonates and neither of hypercalciuria, hypercalcemia, and extraskeletal calcification were observed in their study.

Impact of Hypervitaminosis D and Hypovitaminosisd on Offspring Outcomes

The offspring is wholly dependent on the mother for vitamin D(28). The 25(OH)D passes from the placenta into fetus bloodstream (29). Because the half-life for 25(OH)D is approximately 2–3 weeks, the infant can remain vitamin D sufficient for several weeks after birth, as long as the mother was vitamin D sufficient (7). Evidence has also accumulated regarding the impact of maternal vitamin D levels on the health of the offspring (30). Data related to the effects of maternal vitamin D on skeletal bone integrity and skin color in childhood (31, 32). Maternal vitamin D deficiency was found to correlate with asthma and impaired lung function in offspring (33, 34). Vitamin D acts as a neurosteroid with direct effects on brain development. The brains of offspring from hypovitaminosis D dams are characterized by mild distortion in brain shape, increased lateral ventricle volumes, reduced differentiation and diminished expression of neurotrophic factors (29, 35).

Conclusion

Vitamin D is critical for both pregnant mother and their offsprings. Vitamin D sufficiency is very important for fetal development, especially for their brain development and immunological functions. While, Vitamin D insufficiency during pregnancy is associated with risks of preeclampsia, gestational diabetes and insulin resistance. Increasing pregnant women's awareness of the importance of maintaining enough vitamin D stores during pregnancy, especially

for those at greatest risk of hypovitaminosis D. More researches are needed to establish the exact dose required to supplement pregnant women, especially those with pre-existing deficiency, and determine at which gestation period should vitamin D supplementation be started.

References

[1] Krall EA, Sahyoun N, Tannenbaum S, Dallal GE, Dawson-Hughes B. Effect of vitamin D intake on seasonal variations in parathyroid hormone secretion in postmenopausal women. New England Journal of Medicine. 1989; 321(26): 1777-83.

[2] Lee JH, O'Keefe JH, Bell D, Hensrud DD, Holick MF. Vitamin D deficiency: an important, common, and easily treatable cardiovascular risk factor? Journal of the American College of Cardiology. 2008;52(24):1949-56.

[3] Zhang R, Naughton DP. Vitamin D in health and disease: Current perspectives. Nutrition Journal. 2010 2010/12/08;9(1):65.

[4] Wang TJ, Pencina MJ, Booth SL, Jacques PF, Ingelsson E, Lanier K, et al. Vitamin D deficiency and risk of cardiovascular disease. Circulation. 2008; 117(4): 503-11.

[5] Armas LA, Hollis BW, Heaney RP. Vitamin D2 is much less effective than vitamin D3 in humans. The Journal of Clinical Endocrinology & Metabolism. 2004;89(11):5387-91.

[6] Schwalfenberg GK. A review of the critical role of vitamin D in the functioning of the immune system and the clinical implications of vitamin D deficiency. Molecular nutrition & food research. 2011;55(1):96-108.

[7] Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. The Journal of Clinical Endocrinology & Metabolism. 2011; 96(7): 1911-30.

[8] Holick MF, Smith E, Pincus S. Skin as the site of vitamin D synthesis and target tissue for 1, 25-dihydroxyvitamin D3: use of calcitriol (1, 25-dihydroxyvitamin D3) for treatment of psoriasis. Archives of dermatology. 1987; 123(12): 1677-83a.

[9] Jones G. Pharmacokinetics of vitamin D toxicity–. The American journal of clinical nutrition. 2008; 88(2): 582S-6S.

[10] Haussler MR, Whitfield GK, Haussler CA, Hsieh JC, Thompson PD, Selznick SH, et al. The nuclear vitamin D receptor: biological and molecular regulatory properties revealed. Journal of Bone and Mineral Research. 1998; 13(3): 325-49.

[11] Ferder M, Inserra F, Manucha W, Ferder L. The world pandemic of vitamin D deficiency could possibly be explained by cellular inflammatory response activity induced by the renin-angiotensin system. American Journal of Physiology-Cell Physiology. 2013; 304(11): C1027-C39.

[12] Kovacs CS. Calcium, phosphorus, and bone metabolism in the fetus and newborn. Early human development. 2015; 91(11): 623-8.

[13] Nykjaer A, Dragun D, Walther D, Vorum H, Jacobsen C, Herz J, et al. An endocytic pathway essential for renal uptake and activation of the steroid 25-(OH) vitamin D3. Cell. 1999;96(4):507-15.

[14] Holick MF, editor. High prevalence of vitamin D inadequacy and implications for health. Mayo Clinic Proceedings; 2006: Elsevier.

[15] Mithal A, Wahl DA, Bonjour J-P, Burckhardt P, Dawson-Hughes B, Eisman JA, et al. Global vitamin D status and determinants of hypovitaminosis D. Osteoporosis international. 2009; 20(11): 1807-20.

[16] Sahu M, Bhatia V, Aggarwal A, Rawat V, Saxena P, Pandey A, et al. Vitamin D deficiency in rural girls and pregnant women despite abundant sunshine in northern India. Clinical endocrinology. 2009; 70(5): 680-4.

[17] Dasgupta A, Sarma D, Saikia UK. Hypomagnesemia in type 2 diabetes mellitus. Indian journal of endocrinology and metabolism. 2012; 16 (6): 1000.

[18] Lips P. Vitamin D deficiency and secondary hyper parathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocrine reviews. 2001; 22(4): 477-501.

[19] Kovacs CS. Vitamin D in pregnancy and lactation: maternal, fetal, and neonatal outcomes from human and animal studies. The American journal of clinical nutrition. 2008; 88(2): 520S-8S.

[20] Pitkin RM. Calcium metabolism in pregnancy: A review. American Journal of Obstetrics & Gynecology. 1975; 121(5): 724-37.

[21] Uwitonze AM, Uwambaye P, Isyagi M, Mumena CH, Hudder A, Haq A, et al. Periodontal diseases and adverse pregnancy outcomes: Is there a role for vitamin D? The Journal of steroid biochemistry and molecular biology. 2018.

[22] Posch KR. Current Supplementation Recommen dations for Vitamin D Intake in Pregnancy; What Research Says after 2011. 2018.

[23] Hollis BW, Johnson D, Hulsey TC, Ebeling M, Wagner CL. Vitamin D supplementation during pregnancy: Double-blind, randomized clinical trial of safety and effectiveness. Journal of bone and mineral research. 2011; 26(10): 2341-57.

[24] Aghajafari F, Nagulesapillai T, Ronksley PE, Tough SC, O'Beirne M, Rabi DM. Association between maternal serum 25-hydroxyvitamin D level and pregnancy and neonatal outcomes: systematic review and meta-analysis of observational studies. Bmj. 2013; 346: f1169.

[25] Food, Nutrition Board IoM. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. National Academy Press Washington, DC; 1997.

[26] Hollis BW, Wagner CL. Assessment of dietary vitamin D requirements during pregnancy and lactation. The American Journal of Clinical Nutrition. 2004; 79(5): 717-26.

[27] Hollis BW, Wagner CL. Vitamin D requirements and supplementation during pregnancy. Current Opinion in Endocrinology, Diabetes and Obesity. 2011;18(6):371-5.

[28] Harvey NC, Holroyd C, Ntani G, Javaid K, Cooper P, Moon R, et al. Vitamin D supplementation in pregnancy: a systematic review. Health technology assessment (Winchester, England). 2014; 18(45): 1-190.

[29] Eyles DW, Feron F, Cui X, Kesby JP, Harms LH, Ko P, et al. Developmental vitamin D deficiency causes abnormal brain development. Psychoneuro endocrinology. 2009 2009/12/01/; 34: S247-S57.

[30] Eyles DW, Burne TH, McGrath JJ. Vitamin D, effects on brain development, adult brain function and the links between low levels of vitamin D and neuropsychiatric disease. Frontiers in neuroendocrinology. 2013; 34(1): 47-64.

[31] Ward LM, Gaboury I, Ladhani M, Zlotkin S. Vitamin D–deficiency rickets among children in Canada. Canadian Medical Association Journal. 2007; 177(2): 161-6.

[32] Yuen A, Jablonski N. Vitamin D: in the evolution of human skin colour. Medical hypotheses. 2010; 74(1): 39-44.

[33] Searing DA, Zhang Y, Murphy JR, Hauk PJ, Goleva E, Leung DY. Decreased serum vitamin D levels in children with asthma are associated with increased corticosteroid use. Journal of Allergy and Clinical Immunology. 2010; 125(5): 995-1000.

[34] Zosky GR, Hart PH, Whitehouse AJ, Kusel MM, Ang W, Foong RE, et al. Vitamin D deficiency at 16 to 20 weeks' gestation is associated with impaired lung function and asthma at 6 years of age. Annals of the American Thoracic Society. 2014; 11(4): 571-7.

[35] Eyles D, Burne T, McGrath J, editors. Vitamin D in fetal brain development. Seminars in cell & developmental biology; 2011: Elsevier.

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