Research Article

A Study of 25 (OH) Vitamin D Serum Levels in Patients with Age Group in Kolkata, West Bengal, India.

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ABSTRACT

A study was conducted from April, 2015 to September, 2015. We measured serum level of 25-hydroxy vitamin D with different age groups. We gathered 72 females and 38 males as different age group. We found the insufficiency 25-hydroxy vitamin D result shows Children (1-18 years) of female, Adults (19-50 years & 51-70 years) of male and Adults (> 70 years) of both male & female as those studies carried out in Kolkata, West Bengal, India.

Keywords: Serum 25(OH) vitamin D level, Different age groups, ELISA method.

INTRODUCTION

Vitamin D refers to a group of fat-soluble secosteroids responsible for enhancing intestinal absorption of calcium, iron, magnesium, phosphate and zinc. In humans, the most important compounds in this group are vitamin D_3 (also known as cholecalciferol) and vitamin D_2 (ergocalciferol).[1] Cholecalciferol and ergocalciferol can be ingested from the diet and from supplements.[1-3] Very few foods contain vitamin D; synthesis of vitamin D (specifically cholecalciferol) in the skin is the major natural source of the vitamin. Dermal synthesis of vitamin D from cholesterol is dependent on sun exposure (specifically UVB radiation).

Vitamin D from the diet or dermal synthesis from sunlight is biologically inactive; activation requires enzymatic conversion (hydroxylation) in the liver and kidney. Evidence indicates the synthesis of vitamin D from sun exposure is regulated by a negative feedback loop that prevents toxicity, but because of uncertainty about the cancer risk from sunlight, no recommendations are issued by the Institute of Medicine (US), for the amount of sun exposure required to meet vitamin D requirements. Accordingly, the Dietary Reference Intake for vitamin D assumes no synthesis occurs and all of a person's vitamin D is from food intake, although that will rarely occur in practice. As vitamin D is synthesized in adequate amounts by most mammals exposed to sunlight, it is not strictly a vitamin, and may be considered a hormone as its synthesis and activity occur in different locations. Vitamin D has a significant role in calcium homeostasis and metabolism. Its discovery was due to effort to find the dietary substance lacking in rickets (the childhood form of osteomalacia).[4]

Beyond its use to prevent osteomalacia or rickets, the evidence for other health effects of vitamin D supplementation in the general population is inconsistent.[5, 6] The best evidence of benefit is for bone health.[7] The effect of vitamin D supplementation on mortality is not clear, with one meta-analysis finding a decrease in mortality in elderly people,[8] and another concluding no clear justification exists for recommending vitamin D.[9] Because it found mounting evidence for a benefit to bone health, though it had not found good evidence of other benefits, the Food and Drug Administration of the United States has proposed requiring manufacturers to declare the amount of Vitamin D on nutrition facts labels, as "nutrients of public health significance". As of August 2015, this is currently still open for public comment.[10]

In the liver, cholecalciferol (vitamin D_3) is converted to calcidiol, which is also known as calcifediol (INN), 25-hydroxycholecalciferol (aka

25-hydroxyvitamin D_3 — abbreviated 25(OH)D_3). Ergocalciferol (vitamin D_2) is converted in the liver to 25-hydroxyergocalciferol (aka 25-hydroxyvitamin D_2 — abbreviated 25(OH) D2). These two specific vitamin D metabolites are measured in serum to determine a person's vitamin D status.[11,12] Part of the calcidiol is converted by the kidneys to calcitriol, the biologically active form of vitamin D.[13] Calcitriol circulates as a hormone in the blood, regulating the concentration of calcium and phosphate in the bloodstream and promoting the healthy growth and remodeling of bone. Calcitriol also affects neuromuscular and immune function.[14]

STUDY PERIOD: Study was conducted from April, 2015 to September, 2015.

STUDY PLACE: Midland Diagnostic Lab, Belgharia, Kolkata, West Bengal, India.

SPECIMEN: We gathered 72 females and 38 males as different age group.

METHOD

Test Name/ Kit Used	25-Hydroxyvitamin D; Human, GmbH
Sensitivity:	1.98 ng/ml (range 0.5-1010 ng/ml)
Assay Time:	~1.5 hours
Applications:	ELISA
Application Notes:	For the quantitative determination of human 25(OH) Vitamin D in plasma and serum.
Species reactivity:	Human Serum
Kit/Set Contains:	Microtiter Plate, Conjugate Concentrate, Antibody, Dissociation Buffer, Conjugate Diluent, Sample Diluent, Wash Buffer Concentrate, Standards, Substrate, Stop Solution, Assay Layout.

Reference Range

1. Deficiency : <12 ng/ml
2. Insufficiency : 12-30 ng/ml
3. Sufficiency : >31-150 ng/ml
4. Toxicity : >151 ng/ml

RESULT

Age group	No. of sample	patients		Deficiency		insufficiency		sufficiency		Toxicity	
		Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
INFANTS [0-12 MONTHS]	01	00	01	00	00	00	00	00	01	00	00
CHILDREN [1-18 YEARS]	20	14	06	00	00	04	05	10	01	00	00
ADULTS [19-50 YEARS	39	10	29	00	00	06	09	04	20	00	00
ADULTS [51-70 YEARS]	39	10	29	00	00	06	09	04	20	00	00
ADULTS > 70 YEARS	11	04	07	00	00	03	04	01	03	00	00
TOTAL	110	38	72	00	00	19	27	19	45	00	00

N.B.: We found the insufficiency 25-hydroxy vitamin D result shows Children (1-18 years) of female, Adults (19-50 years & 51-70 years) of male and Adults (> 70 years) of both male & female.

DISCUSSION

The primary form of vitamin D, colecalciferol (vitamin D3), is available from two sources: skin exposure to ultraviolet B radiation (UVB) in sunlight and diet. UVB in the 290-315 nm range photolysis 7-dehydrocholesterol in the skin to form previtamin D3, which then isomerizes to colecalciferol.[15] Colecalciferol (and ergocalciferol [vitamin D2]) is also available from fortified foods (e.g., milk, cereal, and some orange juice and cheeses), dark fish (e.g., salmon and tuna), and vitamin supplements (colecalciferol). Relative to sun exposure, diet is a poor source of colecalciferol, providing only 40-400 IU per food serving,[16] whereas whole-body UVB exposure for 20 min for a light-skinned person during the summer months will produce at least 10,000 IU.[17,23] However, increased skin pigmentation, age, use of sunscreen, built environment, and environmental factors that reduce the strength of UVB reaching the earth's surface (e.g., winter season, high latitude, pollution, cloud cover, and ozone levels) all contribute to reduce skin colecalciferol production to the point at which diet might become the primary source.[17-24] Because melanin pigment in human skin absorbs UVB,[25] black people have lower 25 hydroxyvitamin D concentrations than white people, and are often vitamin D deficient.[26] The importance of age of exposure and seasonality is uncertain. Studies in migrants implicate postnatal environmental exposures, but do not exclude prenatal effects.[27] In a prospective investigation comprising approximately 200,000 women in the USA, vitamin D intake was measured every 4 years by a comprehensive semi-quantitative food frequency questionnaire.[28] Non-melanoma skin cancer and, less strongly, melanoma are more common in individuals with high levels of sun exposure. Thus, if vitamin D was protective, these cancers would be expected to be rare among individuals with multiple sclerosis. Month of birth has also been suggested as a factor that affects multiple sclerosis risk. This finding suggests that prenatal exposures or exposures in the first months of life could be important in multiple sclerosis etiology, but the link to vitamin D is unclear. Genes also can affect vitamin D metabolism, skin color, and behavior, all of which can influence circulating 25-hydroxyvitamin D concentrations. Furthermore, genetic variations in vitamin-D-related and other genes might influence the effects of vitamin D on the immune system. Therefore, genetic variations in vitamin-D-related genes might also affect multiple sclerosis risk, either directly or by modifying the effects of vitamin D.[29]

CONCLUSIONS

In conclusion, the serum vitamin D deficiency might be a potential environmental predisposing factor for developing multiple sclerosis, and need to follow factors that cause this problem including dietary, genetic, and other ones. Whereas future observational epidemiological studies and genetic and molecular investigations will be useful to strengthen and refine the hypothesis, it might be necessary to do a large randomized trial to establish the safety and efficacy needed to promote large-scale vitamin D supplementation. A test of the hypothesis that vitamin D could reduce multiple sclerosis risk will require the administration of relatively high doses of vitamin D to hundreds of thousands of young adults for several years, and careful monitoring for unforeseen adverse effects is mandatory. We suggest that an international multidisciplinary working group to be set up to oversee the design of future prevention or supplementation studies. Furthermore, screening of serum 25-hydroxyvitamin D concentrations is likely to identify a large proportion of patients who are vitamin D deficient or insufficient, and who might benefit from vitamin D supplementation for prevention of osteoporosis and other complications, especially multiple sclerosis.

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