ORIGINAL RESEARCH PAPER

Medical Science

EARLY IMPLANT FAILURE ASSOCIATED WITH VITAMIN D DEFICIENCY

KEY WORDS:

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INTRODUCTION

Vitamin D, a fat-soluble secosteroid, is pivotal for skeletal health owing to its regulation of calcium and phosphate metabolism, vital for bone mineralization. Synthesized endogenously in the skin upon exposure to ultraviolet B (UVB) radiation, it also finds dietary sources including fatty fish, fortified dairy, and supplements.

Beyond its classical role in bone health, vitamin D exhibits pleiotropic effects. It modulates immune function by influencing both innate and adaptive immunity, thereby impacting inflammatory responses and defense against pathogens. Additionally, it regulates cellular proliferation and differentiation, potentially exerting anti-cancer properties.

Deficiency in vitamin D is associated with rickets in children and osteomalacia in adults, characterized by impaired mineralization of bones. Furthermore, suboptimal levels have been linked to increased susceptibility to infections, autoimmune diseases, and certain malignancies.

Optimizing vitamin D status is imperative for maintaining overall health and preventing associated adverse outcomes. The absorption process of vitamin D involves photolysis of 7-dehydrocholesterol in the epidermis upon exposure to ultraviolet B (UVB) radiation, leading to the formation of previtamin D3. This thermally isomerizes to form vitamin D3 (cholecalciferol). Subsequently, cholecalciferol is bound to vitamin D-binding protein (DBP) in the bloodstream for transportation to the liver.

In the liver, cholecalciferol undergoes hydroxylation by the enzyme 25-hydroxylase, resulting in the formation of 25-hydroxyvitamin D3 [25(OH)D3], also known as calcidiol. Calcidiol then enters the circulation and is transported to the kidneys.

In the kidneys, calcidiol undergoes further hydroxylation, mediated by the enzyme 1 -hydroxylase, to form the biologically active form of vitamin D, 1,25-dihydroxyvitamin D3 [1,25(OH)2D3], also known as calcitriol.

Calcitriol acts on the enterocytes of the small intestine, promoting the synthesis of calcium-binding proteins and enhancing the active transport of dietary calcium and phosphate across the intestinal epithelium.

This orchestrated process ensures adequate levels of vitamin D are maintained in the body, facilitating optimal calcium and phosphate absorption for skeletal health and various other physiological functions.

The accuracy of vitamin D assay results hinges on several critical factors inherent to the testing process. Methodological variability among laboratories, stemming from differences in assay techniques and standardization protocols, can introduce inconsistencies in reported values. Seasonal variations in ultraviolet radiation exposure, a primary determinant of endogenous vitamin D synthesis, may confound results, necessitating careful consideration of sample collection timing. Moreover, factors such as dietary supplementation, adipose tissue sequestration, renal or hepatic dysfunction, pharmacological interventions, and genetic polymorphisms can intricately modulate vitamin D metabolism, potentially skewing test outcomes. Hence, meticulous attention to these multifaceted variables is imperative for ensuring the accuracy and clinical relevance of vitamin D assessments in guiding patient care decisions.

Accurately measuring vitamin D levels in the body poses several scientific challenges stemming from the intricacies of its metabolism and external influences. One key obstacle is the lack of standardization in assay methods and reference materials, resulting in variability in results among laboratories and methodologies. Seasonal variations in ultraviolet radiation exposure, the primary source of endogenous vitamin D synthesis, introduce additional complexity, necessitating meticulous timing of sample collection for accurate assessment. Moreover, factors such as adipose tissue sequestration, pharmacological interventions, and metabolic disorders can significantly influence vitamin D metabolism and distribution, impacting the reliability of measurements. Despite advancements in assay technology, achieving precise and reproducible measurements of vitamin D levels remains a formidable task, demanding rigorous attention to methodological nuances and confounding variables for meaningful clinical interpretation.

Vitamin D deficiency engenders a spectrum of physiological perturbations stemming from its multifaceted roles in human physiology. Skeletally, inadequate vitamin D levels precipitate compromised bone mineralization, fostering osteomalacia in adults and rickets in children, characterized by diminished bone density and structural integrity. Concurrently, diminished vitamin D status incites muscular weakness and heightened propensity for falls, reflective of its regulatory influence on muscle function. Immunologically, vitamin D insufficiency compromises the intricate orchestration of innate and adaptive immune responses, fostering an environment conducive to infections and autoimmune dysregulation. Emerging research implicates vitamin D deficiency in mood disorders and cardiovascular pathologies, albeit mechanisms remain incompletely

elucidated. A confluence of factors, encompassing limited sunlight exposure, dietary inadequacies, malabsorption syndromes, adiposity-mediated sequestration, and demographic predispositions, collectively contribute to the prevalence of vitamin D insufficiency. Comprehensive management strategies entail targeted supplementation, dietary fortification, and addressal of underlying malabsorptive states, emphasizing the multifaceted nature of this clinical entity.

Whereas, Vitamin D toxicity, scientifically termed hypervitaminosis D, manifests when there is an excessive accumulation of vitamin D in the body, precipitating adverse effects. Primarily, it arises from prolonged consumption of supra-physiological doses of vitamin D supplements, surpassing recommended intake levels. Additionally, overconsumption of fortified foods, particularly when combined with supplementation, can contribute to toxicity. Rarely, certain medical conditions, such as granulomatous diseases and lymphomas, can induce excessive production of activated vitamin D within the body, further exacerbating toxicity risk. The principal consequence of vitamin D toxicity is hypercalcemia, wherein elevated serum calcium levels induce gastrointestinal distress, including nausea, vomiting, and diarrhea. Severe cases may culminate in renal impairment, characterized by kidney stones and compromised renal function. Paradoxically, excessive vitamin D can augment bone resorption, potentially engendering bone loss and fractures. Emerging evidence also suggests a potential association between elevated vitamin D levels and cardiovascular morbidity, albeit necessitating further elucidation. Given the severity of potential complications, vigilance in adhering to recommended vitamin D intake levels, especially through supplementation, is imperative, particularly among individuals with underlying medical conditions.

Dental Corelation

50 healthy individuals ranging from age group 21 -55 years of age suffering from no other underlying co morbities underwent single implant placement at healed sites. 50 implants failed to integrate (also known as early implant failure). All implant failure were noted between 2-4 weeks of initial implant surgery.

Four different brands of implant systems were used with various implant surface coating in all patients. One Design was parallel other three were tapered in shape.

There is critical importance of vitamin D in the efficacy of dental implant procedures. Studies consistently demonstrate a correlation between vitamin D levels and key factors influencing implant success. Firstly, vitamin D is essential for bone health, particularly bone density and mineralization, which are vital for the osseointegration process. Insufficient levels of vitamin D have been associated with compromised bone quality and reduced bone-implant contact, heightening the risk of implant instability and failure. Additionally, vitamin D plays a significant role in immune modulation, which is crucial for post-surgical healing and defence against peri-implant infections. Inadequate levels of vitamin D may hinder immune responses, prolonging inflammation and increasing susceptibility to complications. Moreover, research suggests a link between vitamin D deficiency and increased susceptibility to periodontal diseases, which can impact the surrounding tissues of dental implants. Therefore, maintaining optimal levels of vitamin D is imperative for promoting successful outcomes in dental implant procedures, from facilitating osseointegration to reducing the risk of peri-implant complications.

All subjects were asked to undergo Vitamin D test to check their levels. The results show that all individuals had Vitamin D deficiency.

Implant failure can be due to a number of reasons not just Vitmin D deficiency and may include inadequate osseointegration due to poor bone quality or quantity, biomechanical issues such as occlusal overload, peri-implantitis resulting from microbial infection, or surgical factors like improper implant placement or inadequate primary stability. In addition to the aforementioned factors, several other causes contribute to early implant failure. These include systemic conditions such as uncontrolled diabetes or autoimmune diseases, which can impair the body's ability to heal and integrate the implant. Smoking has also been identified as a significant risk factor due to its adverse effects on vascularization and wound healing. Furthermore, complications during the healing process, such as excessive inflammation or fibrous encapsulation, can compromise the stability and longevity of the implant. Additionally, poor oral hygiene practices and inadequate postoperative care may predispose the implant to bacterial colonization and peri-implant inflammation, further exacerbating the risk of failure. Understanding and addressing these multifaceted contributors to early implant failure are crucial for optimizing treatment outcomes and ensuring the long-term success of dental implant therapy.

S.No.	Patient Name	Age	Sex	Vitamin D level(nmol/L)
1	PN	46	M	21.8
2	ZD	28	F	20.6
3	LM	50	M	18
4	KB	34	F	24
5	RD	58	M	23
6	AD	37	F	25.7
7	SSD	56	F	20.9
8	SRD	64	M	22.9
9	SLD	71	M	21
1	SD	56	F	24
11	AB	45	M	18.7
12	SB	39	F	13
13	SL	66	F	15.6
14	AS	41	M	14.3
15	GS	45	M	17.8
16	KJB	40	M	10.3
17	RB	68	M	18
18	RA	56	F	17.3
19	AA	59	F	12.6
20	KK	63	M	19.8
21	SK	35	M	16.7
22	SAK	67	M	17.2
23	AB	34	F	12.4
24	YB	38	F	9.7
25	RB	69	M	18.9
26	SN	46	M	12.5
27	RK	38	F	14.3
28	SK	66	M	15.8
29	TK	61	F	17.9
30	MB	70	M	16
31	NB	65	F	12.9
32	VD	40	F	10.6
33	ND	39	F	7.8
34	TV	68	F	8.5
35	SNB	56	M	21.6
36	SB	54	F	8.9
37	RB	44	M	9.6
38	AP	48	F	6.9
39	KSO	46	M	19
40	MM	58	M	18
41	AC	37	M	12.5
42	JG	40	F	14.8
43	RN	65	M	15.4
44	AH	70	F	12.9
45	RA	39	F	16
46	PR	45	M	18
47	PD	41	F	15.6

48	MP	36	F	19.4
49	GT	35	M	15
50	SK	27	M	15.9

Interpretation:

Levels	Reference range in nmol/L	Comments
Deficient	<50	High risk of developing bone disease
Insufficient	50-74	Vitamin D concentration which normalises parathyroid hormone concentration
Sufficient	75-250	Optimal concentration for maximal health benefit
Potential intoxication	>250	High risk for toxic effects

CONCLUSION

Role of vitamin D in body's normal functioning is paramount. Its deficiency leads to many complications, and failure of dental implants to integrate is one of them. In This retrospective study done on 50 patients whose implants failed to integrate over a period of 1 year, showed early implant failure (within 2-4 weeks) due to vitamin D deficiency. Hence, authors stress upon the importance of blood investigations, and strongly recommend Vitamin D test for patients going for implant surgery. In case of low-levels, Vitamin D supplementation is absolutely necessary followed by another test after 3 months.

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