REVIEW ARTICLE

VITAMIN D SUPPLEMENTS FOR OVERALL DENTAL AND IMPLANT HEALTH: SENSE OR NONSENSE?

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Periodontitis is a multifactorial disease involving both genetic and environmental risk factors. Initiated by a consortia of oral bacteria that elicit local inflammatory responses leading to bleeding on probing (BOP), loss of attachment, as well as bone[1]. Vitamin D deficiency is contemplated as one of the risk factors for periodontitis, tooth loss, early implant failure, and peri-implantitis.

There is a widespread prevalence of varying degrees (50–90%) of vitamin D deficiency. It has been estimated that one billion people worldwide have vitamin D deficiency or insufficiency[2].

A chronically low intake of vitamin D and calcium may lead to a negative calcium balance, thus causing a secondary increase in calcium removal from bones, including the alveolar bone[3]. Such bone loss may contribute to weakening of the tooth-attachment and implant-attachment apparatus.

In addition to its action on skeletal homeostasis, vitamin D has anti-inflammatory and anti-microbial effects via modulation of inflammatory cytokine production by immune cells and stimulated secretion of peptides with anti-bacterial action by cells of the monocyte–macrophage lineage[4].

Vitamin D is a membrane antioxidant, that has the ability to inhibit iron-dependent lipid peroxidation in liposomes and protect membranes and LDL (low density lipoprotein) against oxidative damage[5].

The multiple actions of vitamin D in calcium homeostasis, immune response, and antioxidant effect make it a multi-edged sword that is potentially appealing for the management of patients with periodontal disease and peri-implantitis, whose pathogenesis is based on chronic bacterial-driven inflammation.

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The inflammatory response leads to tissue destruction by direct action of bacterial products, by activation of host defence cells and secretion of inflammatory mediators, or by increasing oxidative stresses[6]. These locally produced factors eventually result in connective tissue break-down and bone loss via activation of osteoclast-mediated bone resorption.

It is plausible that vitamin D deficiency affects the periodontal tissues. An association between serum concentrations of 25-hydroxy- vitamin D (25OHD) and gingival inflammation was found, possibly linked to vitamin D's anti-inflammatory effect[7]. A potential role of vitamin D in periodontal health is also supported by findings that polymorphisms of the vitamin D receptor gene are associated with periodontitis, alveolar bone loss, clinical attachment loss, and/or tooth loss. The human vitamin D receptor (VDR) gene is located in chromosome 12q12–q14, and it is an interesting candidate gene because of its association with periodontitis and because it affects both bone metabolism and immune functions[8].

In dental implantology, the role of vitamin D in the calcium economy is extremely important. During osteointegration, calcitriol affects the processes of activation and differentiation of osteoblasts and osteoclasts. Vitamin D has also been found to be essential for the maturation and proper functioning of bone cells by stimulating osteoclast precursor fusion and stimulation of osteoblast differentiation[8]. Vitamin D also increases osteoid mineralization, playing an important role in the stabilization phase of the implant[9].

Beyond modulation of bone formation, vitamin D has an impact on the innate and adaptive immune response in the field of osteoimmunology, influencing early implant healing.

Changes in cytokine secretion due to vitamin D deficiency can impair osteoclast activation and differentiation through VDR activation. The plausible reason for implant failure is considered to be bone necrosis during implant bed preparation or placement[10]. Furthermore, vitamin D deficiency might disrupt the sensitive balance between the immune system and bone metabolism during implant healing due to direct or indirect alteration of osteoclast function. Vitamin D controls osteoclast precursor monocyte migration,

therefore the removal of bone debris through osteoclasts could be hampered[11]

Implant osseointegration is not simply a wound healing phenomenon but rather a complex foreign body reaction with activation of the immune system. The titanium and metal particles released from the implant, may be a cause for implant failure, as metallic particles affect macrophages and lymphocytes and cause them to release inflammatory cytokines, leading to increased osteoclastogenesis and decreased osteoblastogenesis, which eventually results in peri-implant bone degeneration. This osteolytic effect could be enhanced by vitamin D deficiency[12].

Vitamin D is essential for the antibacterial response. It inhibits Porphyromonas gingivalis-induced pro-inflammatory cytokine expression and improves the expression of anti-inflammatory cytokines in macrophages[13].

Nevertheless, the high prevalence of vitamin D deficiency in the Indian population indicates that it is probably not the sole causative factor for early implant failure; otherwise, the failure rate would be significantly higher. However, a synergistic effect with other factors is conceivable.

As it has become an active factor in dental and implant surgery, it is imperative to examine vitamin D deficiency before implant and dental surgery, and start with supplements.

There is enough evidence pointing to a potential role of vitamin D on dental health and dental implant health, however, the possible effects of such dietary supplementation on periodontal disease parameters and outcomes have not been addressed. It is recommended to conduct comprehensive studies with larger sample sizes to determine the exact mechanism involved.

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