SECTION 1. CLINICAL MEDICINE: EXPERIENCE AND INNOVATIONS

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THE ROLE OF VITAMIN D IN DENTAL MINERALIZATION AND CARIES PREVENTION

РОЛЬ ВІТАМІНУ D У МІНЕРАЛІЗАЦІЇ ЗУБІВ ТА ПРОФІЛАКТИЦІ КАРІЄСУ

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Vitamin D is a multifunctional steroid hormone that is synthesized in the human body primarily under the influence of sunlight. Additional sources include dietary intake and nutritional supplements [1].

There are few natural foods rich in vitamin D. The highest concentrations are found in fatty fish (such as salmon, mackerel, and herring) and fish oils, particularly cod liver oil [2].

Vitamin D is a general term referring to a group of compounds. Two main forms are distinguished: vitamin D_2 (ergocalciferol) and vitamin D_3 (cholecalciferol). Vitamin D_2 is produced through ultraviolet irradiation of ergosterol (derived from yeast). Vitamin D_3 is synthesized via ultraviolet irradiation of 7-dehydrocholesterol in human skin and can also be derived from lanolin. Cholecalciferol represents the biologically active precursor form.

The accepted biomarker for assessing vitamin D status is serum 25-hydroxyvitamin D (25(OH)D).

Optimal synthesis and metabolism of vitamin D are essential for the physiological functioning of most body systems [1].

Primarily, vitamin D acts as a hormone regulating calcium and phosphate homeostasis by enhancing their intestinal absorption [3]. Consequently, it promotes growth and physiological remodeling of bones and teeth.

In addition, vitamin D functions as an autocrine and paracrine agent, regulating cell differentiation, cell maturation, and innate immune responses [4].

At the cellular level, vitamin D exerts its effects via the vitamin D receptor (VDR), an intracellular receptor that binds the active form of vitamin D [5]. Vitamin D modulates the expression of approximately 5–10% of the human genome [5].

Currently, there is a significant global increase in the prevalence of vitamin D deficiency (VDD) [6]. This represents a major public health concern, particularly in relation to children, pregnancy, certain malignancies, and infection prevention [7].

The primary cause of VDD is insufficient sun exposure and inadequate ultraviolet B (UVB) radiation [6]. Countries with limited annual sunlight are at higher risk.

Other contributing factors include inadequate dietary intake, inherited disorders affecting absorption or metabolism [6], and medication-induced alterations (e.g., phenytoin, carbamazepine, oxcarbazepine) [8].

Teeth are highly mineralized organs. Dental mineralization occurs in parallel with skeletal mineralization. Therefore, vitamin D plays a crucial role in bone and tooth formation.

Severe vitamin D deficiency (<10 ng/mL) leads to hypocalcemia and hypophosphatemia, which trigger secondary hyperparathyroidism [8]. As a result, serum calcium levels increase (due to bone resorption), while inorganic phosphate levels decline further. This imbalance disrupts normal mineralization and contributes to enamel and dentin defects.

The so-called “rachitic tooth” is characterized by hypomineralization, structural weakness, and increased susceptibility to caries and fractures [8].

Beyond mineral homeostasis, vitamin D signaling via VDR induces the synthesis of structural proteins, including calcium-binding proteins and extracellular matrix proteins (enamelins, amelogenins, dentin phosphoproteins, dentin sialoproteins).

The type and location of mineralization defects depend on the gestational timing of vitamin D deficiency, which may serve as a biological marker of prenatal deficiency. Monitoring vitamin D levels before conception may reduce the risk of enamel defects in primary teeth.

Dental caries in primary and permanent teeth is the most prevalent disease worldwide. According to the World Health Organization (WHO), dental caries is among the most costly chronic diseases in terms of treatment expenditures [9].

Current evidence suggests an association between low vitamin D levels and increased caries prevalence, although the exact mechanism remains unclear [10].

An optimal 25(OH)D concentration (≥75 nmol/L) is associated with a reduced risk of caries in children [11].

A systematic review of controlled clinical trials involving 2,827 children demonstrated that vitamin D supplementation reduced caries risk by approximately 47%. Children without caries were twice as likely to have optimal 25(OH)D levels (≥75 nmol/L), whereas children with severe early childhood caries were nearly three times more likely to have deficient levels (<35 nmol/L) [12].

However, serum vitamin D levels do not alter the structure of already formed teeth.

The preventive effect of vitamin D is mediated through: modulation of immune responses, stimulation of antimicrobial activity, induction of bactericidal peptides.

Vitamin D deficiency is also associated with enamel hypoplasia and salivary gland atrophy, further increasing caries susceptibility.

Additionally, deficiency may elevate the risk of low bone mineral density, osteopenia, osteoporosis, and inflammatory or infectious diseases [5].

In the mid-20th century, the American Medical Association and the US National Research Council concluded that vitamin D was beneficial in caries prevention. In contrast, the American Dental Association expressed an opposing view. In 1989, the US National Research Council described the relationship between vitamin D and caries as “unresolved.”

Despite historical controversy, current evidence supports the important role of vitamin D in dental development and oral health maintenance. Monitoring serum 25(OH)D levels may be considered as part of preventive oral health strategies.

Maintaining adequate vitamin D status is associated with improved dental development and lifelong oral health.

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