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VITAMIN D DEFICIENCY: PATHOPHYSIOLOGY, HORMONAL REGULATION AND DIAGNOSTIC APPROACHES

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Annotation

This article examines the contemporary relevance of vitamin D deficiency, focusing on its pathophysiological mechanisms and its interaction with the endocrine system. The metabolism of vitamin D, as well as the role of its biologically active form in regulating gene expression, is discussed based on current scientific evidence. Particular attention is given to metabolic and hormonal alterations associated with vitamin D deficiency, including disturbances in calcium–phosphorus homeostasis, secondary hyperparathyroidism, and insulin resistance. In addition, modern approaches to diagnosis, treatment, and prevention are outlined.

Keywords

Vitamin D, calcitriol, vitamin D deficiency, endocrine system, calcium–phosphorus metabolism, parathyroid hormone, insulin resistance, metabolic syndrome, vitamin D receptor (VDR), osteomalacia

Vitamin D is a fat-soluble secosteroid that exerts hormone-like effects in the human body. Its active metabolite, calcitriol (1,25-dihydroxyvitamin D), interacts with intracellular receptors to regulate gene transcription. In recent years, vitamin D deficiency has been recognized as a global health issue due to its association not only with skeletal disorders but also with endocrine and immune dysfunctions.

Vitamin D Metabolism and Regulation

Vitamin D undergoes a series of metabolic transformations to become biologically active. Initially, it is synthesized in the skin under ultraviolet B (UVB) radiation as cholecalciferol (vitamin D3). It is then hydroxylated in the liver to form 25-hydroxyvitamin D [25(OH)D], followed by further hydroxylation in the kidneys to produce the active form, 1,25-dihydroxyvitamin D.

This process is tightly regulated by several factors:

- Parathyroid hormone (PTH), which enhances activation
- Serum phosphate levels
- Fibroblast growth factor-23 (FGF-23), which inhibits activation

Pathogenesis of Vitamin D Deficiency

Vitamin D deficiency leads to impaired intestinal absorption of calcium, resulting in hypocalcemia. This triggers compensatory secretion of parathyroid hormone, leading to

secondary hyperparathyroidism. Prolonged imbalance disrupts bone mineralization, contributing to osteomalacia in adults and rickets in children.

Endocrine Implications

Vitamin D plays a crucial role in endocrine regulation. It influences pancreatic β-cell function by enhancing insulin secretion and improving insulin sensitivity in peripheral tissues. Consequently, deficiency of vitamin D is associated with an increased risk of insulin resistance and metabolic syndrome.

Furthermore, vitamin D has been implicated in thyroid function and autoimmune endocrine disorders, including Hashimoto's thyroiditis.

Immunological Effects

Vitamin D acts as an immunomodulatory agent by regulating both innate and adaptive immune responses. It suppresses pro-inflammatory cytokine production while promoting antimicrobial peptide synthesis. Deficiency may therefore increase susceptibility to infections and autoimmune diseases.

Diagnosis and Management

Serum 25(OH)D concentration is the primary marker used to assess vitamin D status:

- Deficiency: <20 ng/mL
- Insufficiency: 20–30 ng/mL
- Sufficiency: 30–100 ng/mL

Treatment typically involves supplementation with cholecalciferol (vitamin D3), with dosage tailored to individual patient characteristics. Preventive strategies include adequate sun exposure, balanced nutrition, and screening of at-risk populations.

Conclusion

Vitamin D deficiency is associated with complex metabolic and hormonal disturbances affecting multiple organ systems. Its impact extends beyond skeletal health, influencing endocrine and immune functions. Early detection and appropriate management are essential to prevent long-term complications and improve overall health outcomes.

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