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Interactions of vitamin D with parathyroid glands; an updated mini-review

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Abstract

Vitamin D is essential for regulating calcium and phosphate metabolism, and its deficiency or excess can significantly affect parathyroid function. The deficiency or insufficiency of vitamin D can lead to increased parathyroid hormone (PTH) production, resulting in secondary hyperparathyroidism. Conversely, adequate vitamin D levels suppress PTH synthesis and maintain normal calcium levels. Dysregulation of this delicate balance can contribute to various disorders such as osteoporosis, renal stones, and skeletal abnormalities. Hence, understanding the complex interactions between vitamin D and the parathyroid glands is important for diagnosing and managing disorders such as primary and secondary hyperparathyroidism.

Keywords: Vitamin D, Parathyroid glands, Parathyroid hormone, Calcium homeostasis, Secondary hyperparathyroidism

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Introduction

Vitamin D is crucial in maintaining calcium and phosphate homeostasis, primarily through its interactions with the parathyroid glands (1,2). Vitamin D acts on parathyroid glands through multiple mechanisms. It suppresses parathyroid hormone (PTH) synthesis and secretion by directly inhibiting its gene expression (3,4). Vitamin D also enhances calcium-sensing receptor expression in parathyroid cells, decreasing PTH release. Additionally, vitamin D promotes calcium absorption from the intestines and reabsorption from renal tubules, further modulating PTH secretion (1,5). This review article aims to provide an overview of the relationship between vitamin D and the parathyroid glands, including the synthesis and metabolism of vitamin D, the regulation of PTH secretion, and the effects of vitamin D deficiency or excess on parathyroid function.

Search strategy

For this review, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ), and Embase, using different keywords such as vitamin D, parathyroid glands, parathyroid hormone, calcium homeostasis, and secondary hyperparathyroidism.

Results

The parathyroid glands, located behind the thyroid

gland in the neck, secrete a hormone called PTH. PTH is responsible for regulating blood calcium levels. When blood calcium levels drop, the parathyroid glands increase PTH secretion to stimulate a series of events that help elevate circulating calcium levels (6,7).

Vitamin D and PTH secretion

Vitamin D deficiency leads to decreased calcium absorption from the intestine, lowering blood calcium levels. As a compensatory mechanism, the parathyroid glands secrete more PTH to stimulate calcium release from bones and increase calcium reabsorption in the kidneys. Vitamin D deficiency can therefore cause an increase in PTH secretion (4,6,8,9).

Vitamin D supplementation and PTH secretion

Vitamin D supplementation can suppress PTH secretion in individuals with vitamin D deficiency. Adequate vitamin D levels can reduce PTH synthesis and release in the parathyroid glands, thus maintaining calcium homeostasis (10,11).

Parathyroid gland growth

Vitamin D deficiency can lead to the development of parathyroid gland enlargement, known as secondary hyperparathyroidism. This occurs due to increased PTH secretion needed to maintain calcium levels. Vitamin D supplementation helps prevent parathyroid gland

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■ Implication for health policy/practice/research/medical education

Vitamin D is a fat-soluble vitamin that plays a crucial role in maintaining calcium and phosphorus levels in the body. It is obtained from exposure to sunlight, certain foods, and dietary supplements. Once in the body, vitamin D converts to its active form, calcitriol. Calcitriol then acts on the intestines, bones, and kidneys to regulate calcium and phosphorus homeostasis..

enlargement by suppressing PTH secretion (4,12).

Feedback loop

The relationship between vitamin D and the parathyroid glands involves a feedback loop. When blood calcium levels are low, vitamin D synthesis is stimulated, leading to increased intestinal calcium absorption and decreased PTH secretion. Conversely, vitamin D synthesis is reduced when blood calcium levels are high, resulting in decreased intestinal calcium absorption and increased PTH secretion (2,13).

Discussion

Vitamin D is synthesized in the skin through exposure to sunlight and can also be obtained from dietary sources or supplements. It undergoes hydroxylation in the liver and kidneys to form its active metabolite, 1,25-dihydroxy vitamin D (calcitriol) (14-17). Calcitriol acts on the parathyroid glands to inhibit PTH synthesis and secretion, thereby reducing calcium release from bone and enhancing calcium reabsorption in the kidneys (18,19). Vitamin D deficiency increases PTH secretion, secondary hyperparathyroidism and subsequent bone loss (20). On the other hand, excessive vitamin D levels can suppress PTH secretion, leading to hypocalcemia and hyperphosphatemia. The optimal range of vitamin D levels for maintaining parathyroid function and bone health is still a subject of debate (21,22).

Vitamin D deficiency leads to reduced intestinal absorption of calcium, resulting in lower blood calcium levels. As a compensatory mechanism, the parathyroid glands respond by increasing PTH secretion. Higher PTH levels stimulate the release of calcium from bones and increase calcium reabsorption in the kidneys. This process helps raise blood calcium levels back to normal (4,23,24).

On the other hand, when individuals with vitamin D deficiency receive vitamin D supplementation, it helps overcome the calcium absorption problem in the intestines. This leads to increased blood calcium levels, and as a result, the parathyroid glands reduce their secretion of PTH. Adequate vitamin D levels, therefore, suppress PTH synthesis and release in the parathyroid glands, maintaining calcium homeostasis (25,26).

Moreover, vitamin D deficiency can have long-term effects on parathyroid gland function. When the body consistently lacks vitamin D, the parathyroid glands

undergo a process known as glandular hyperplasia. This means that the parathyroid glands become enlarged due to the constant need for increased PTH secretion to maintain calcium levels (27,28). Vitamin D supplementation, however, can prevent and reverse this condition by suppressing PTH secretion and preventing parathyroid gland growth. Notably, the relationship between vitamin D and the parathyroid glands involves a feedback loop (10,29). When blood calcium levels drop, the body stimulates vitamin D synthesis to help increase calcium absorption from the intestine and reduce PTH secretion. Conversely, the body reduces vitamin D when blood calcium levels are high (15,30).

In clinical practice, healthcare professionals may consider monitoring both vitamin D levels and assessing parathyroid gland function in patients with calcium and bone disorders. Additionally, maintaining optimal vitamin D levels through sunshine exposure, dietary sources, or supplementation is important for overall bone health and calcium regulation (31,32).

Conclusion

The interactions between vitamin D and the parathyroid glands are crucial in maintaining calcium and phosphate homeostasis. Clinicians should consider vitamin D levels when evaluating patients with disorders of parathyroid function and ensure appropriate supplementation or treatment to optimize bone health. Further research is needed better to understand these interactions' complex mechanisms and clinical implications.

Conflicts of interest

The author declares that he has no competing interests.

Ethical issues

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