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Cinacalcet in patients with secondary hyperparathyroidism; a review

Hamid Reza Hemmati^{*}

Abstract

Secondary hyperparathyroidism is a common complication of chronic kidney disease characterized by excessive secretion of parathyroid hormone from the parathyroid glands. Cinacalcet is a calcimimetic medication that reduces parathormone levels by increasing the sensitivity of the calcium-sensing receptors on the parathyroid glands. This review aims to summarize the existing literature on the use of cinacalcet in patients with secondary hyperparathyroidism.

Keywords: Cinacalcet, End-stage renal disease, Parathyroid hormone, Secondary hyperparathyroidism, Chronic kidney disease, Calcimimetic agents, Parathormone

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Introduction

Secondary hyperparathyroidism occurs due to a disruption in the normal feedback loop that helps regulate calcium levels in the body (1). When calcium levels in the blood are low, the parathyroid glands release parathyroid hormone (PTH; parathormone), which stimulates calcium release from bones and increases the absorption of calcium from the intestines (2,3). However, in patients with chronic kidney disease or end-stage renal disease, the kidneys cannot properly regulate calcium and phosphorus levels, leading to low calcium levels. This triggers an overproduction of PTH by the parathyroid glands, which results in secondary hyperparathyroidism (4,5). This mini-review paper summarizes the current data on cinacalcet therapy in individuals with secondary hyperparathyroidism.

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 including: cinacalcet, end-stage renal disease, parathyroid hormone, secondary hyperparathyroidism, chronic kidney disease, calcimimetic agents and parathormone

Administration of cinacalcet in secondary hyperparathyroidism

Cinacalcet is a calcimimetic medication that is commonly used to treat secondary hyperparathyroidism. It works

by mimicking the action of calcium on the parathyroid glands. Cinacalcet binds to calcium-sensing receptors on the parathyroid glands, which increases their sensitivity to calcium. As a result, the parathyroid glands are less stimulated to produce and release parathormone, decreasing PTH levels (5,6).

Multiple studies have investigated the efficacy of cinacalcet in patients with secondary hyperparathyroidism. These studies have consistently shown that cinacalcet can effectively lower parathormone levels in patients with chronic kidney disease or end-stage renal disease (7,8). By reducing PTH levels, cinacalcet helps normalize the body's calcium and phosphorus balance. One study included in the review compared cinacalcet to placebo in patients with chronic kidney disease and secondary hyperparathyroidism. It found that cinacalcet significantly reduced PTH levels compared to placebo (9,10). Another study in the review also evaluated the long-term use of cinacalcet in patients with end-stage renal disease and secondary hyperparathyroidism. It demonstrated that cinacalcet effectively maintained control of PTH levels over an extended period and also led to improvements in calcium and phosphorus levels (11-13). Cinacalcet is generally well-tolerated by patients with secondary hyperparathyroidism. The most commonly reported side effects include nausea, vomiting, and hypocalcemia. These side effects are usually mild and can be managed with appropriate dosage adjustments or discontinuation if necessary (14,15).

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Clinical Research Development Unit, Kowsar Educational, Research and Therapeutic Hospital, Semnan University of Medical Sciences, Semnan, Iran.

***Corresponding author:** Hamid Reza Hemmati, Email: dr.hemmati2007@yahoo.com, dr.hhemmati@semums.ac.ir

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

Cinacalcet is an effective treatment option for patients with secondary hyperparathyroidism, particularly in those with chronic kidney disease or end-stage renal disease. Cinacalcet helps restore the body's calcium and phosphorus balance by lowering parathormone levels. It is important to consult with a healthcare professional for appropriate dosing and management of side effects.

Conclusion

Overall, cinacalcet has effectively reduced PTH levels in patients with secondary hyperparathyroidism. It works by activating the calcium-sensing receptors on the parathyroid glands, leading to decreased PTH secretion. This reduction in PTH levels helps to normalize calcium and phosphorus metabolism.

Conflicts of interest

The author declares that he has no competing interests.

Ethical issues

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