



Secondary hyperparathyroidism and chronic kidney disease

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One of the chronic kidney disease complications, is secondary hyperparathyroidism. The development of secondary hyperparathyroidism results from various factors, including deficiency of calcitriol, retention of phosphorus, a decrease in the activation of the calcium-sensing receptor in the parathyroid gland, and skeletal resistance to the calcemic effects of parathormone. As kidney function declines, so does phosphorus excretion, thus causing plasma phosphorus levels to rise while plasma calcium and calcitriol levels decrease. A reduction in calcitriol also contributes to a reduction in intestinal calcium absorption. All of these factors contribute to the development of hypocalcemia, which is the motivation for an increased production of parathormone (1).

Several studies have assessed the relation of hyperparathyroidism in dialysis patients with other risk factors. In these studies, the relationship between malnutrition and inflammatory processes and cardiovascular complications in dialysis patients are discussed. The results suggest a positive association between parathormone and C-reactive protein and the inverse relationship between parathormone and body mass index. It was recommended that the hyperphosphatemia should be managed and parathormone levels did not exceed more than 1.5 times the normal level to prevent from secondary hyperparathyroidism complications (2,3). Parathormone may be responsible for long-term consequences that include renal osteodystrophy, severe vascular calcifications, changes in cardiovascular structure and function, anemia and immune dysfunction. These unfavorable effects may be related to an increased risk of cardiovascular morbidity and mortality among end-stage renal failure patients (1-4).

In a study, there was a significant inverse association between $\text{Ca} \times \text{PO}_4$ product and the age and significant positive correlation of logarithm of serum C-reactive protein with $\text{Ca} \times \text{PO}_4$ product. This study indicated to further attention to hyperphosphatemia in hemodialysis patients (5).

Progression of chronic kidney disease is associated with

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

Secondary hyperparathyroidism is a frequently encountered problem in the management of patients with chronic kidney disease. Its pathophysiology is mainly due to hyperphosphatemia and vitamin D deficiency and resistance. This situation has a high impact on the mortality and morbidity of dialysis patients. Prompt diagnosis of secondary hyperparathyroidism is crucial in the management of patients with chronic kidney disease. The treatment remains a challenge for patients and their clinicians. It would comprise a combination of dietary phosphorus restriction, phosphate binders, calcimimetics and vitamin D analogues.

a number of serious complications, including increased incidence of cardiovascular disease, hyperlipidemia, anemia and metabolic bone disease. Chronic kidney disease patients should be assessed for the presence of these complications and receive optimal treatment to reduce their morbidity and mortality (6).

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Dietary approaches and the use of phosphorus chelating agents, either alone or in combination, do not appear to be particularly promising for secondary hyperparathyroidism in uremic patients with chronic kidney disease stages 3 and 4. Pending the publication of statistically well-structured works on chronic kidney disease stages 3 and 4, experience

with calcium mimetic agents in chronic kidney disease stages 3 and 4 seems promising, even if there is a necessity to decrease the side effects most affecting medication compliance and as well safety calcium-mimetic agents seem to be more useful, particularly in association with vitamin D derivatives. Additional promising results seem to be provided by the latest generations of vitamin D derivatives such as paricalcitol which produces appropriate secondary hyperparathyroidism control (8).

Author's contribution

MRT is the single author of the manuscript.

Conflict of interests

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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