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Mini-Review

Diseases of the parathyroid glands in chronic kidney disease

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Abstract

Chronic kidney disease (CKD) is a prevalent health condition affecting millions worldwide. One of the common complications of CKD is CKD-mineral and bone disorder (CKD-MBD), which involves disturbances in mineral metabolism and skeletal health due to complications of CKD. The parathyroid glands, a small gland located near the thyroid glands in the neck, play a vital role in regulating calcium and phosphorus metabolism in the body. CKD can result in the dysfunction of the parathyroid glands, leading to a range of complicated disorders.

Keywords: Parathyroid glands, CKD-MBD, Chronic kidney disease, Parathyroid hormone, Chronic kidney disease-mineral and bone disorders, Parathormone, Kidney, Vitamin D

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Introduction

Chronic kidney disease (CKD) can cause abnormalities in the parathyroid glands' function, leading to various disorders collectively known as CKD-mineral and bone disorders (CKD-MBD). CKD-MBD is a common complication of CKD that affects up to 90% of patients with advanced CKD (1, 2). In the early stages of CKD, the parathyroid glands may overproduce parathyroid hormone (PTH; parathormone) to compensate for the increased losses of calcium and phosphorus through the kidneys. However, as CKD progresses, the parathyroid glands may become resistant to the effects of parathormone, leading to a decrease in calcium levels and an increase in phosphorus levels in the blood (3). These changes can lead to complications such as osteoporosis, bone pain, fractures, and cardiovascular disease. In addition, high levels of PTH can cause the parathyroid glands to enlarge, resulting in a parathyroid adenoma or hyperplasia, which can further aggravate the CKD-MBD and require surgical intervention (4, 5). This paper aims to discuss the different diseases of the parathyroid glands that occur in CKD.

Search strategy

For this mini-review, we conducted a comprehensive literature search using multiple databases, including PubMed, Google Scholar, Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus and Embase. We used a variety of relevant keywords to retrieve articles related to the topic, such as parathyroid glands, Chronic kidney disease-mineral and bone disorders, CKD-MBD, bone pain, osteoporosis, cardiovascular disease, parathyroid hyperplasia and Vitamin D

Parathyroid Hormone

The parathyroid glands produce PTH, which plays a critical role in regulating calcium and phosphorus metabolism. PTH regulates serum calcium and phosphorus levels by enhancing calcium reabsorption and inhibiting kidney phosphorus reabsorption (6).

In the early stages of CKD, the parathyroid glands compensate for calcium loss through the kidneys by overproducing parathormone. However, as CKD progresses, the parathyroid glands become resistant to parathormone, leading to a drop in calcium levels and increased phosphorus levels in the blood (7).

Chronic kidney disease-mineral and bone disorder

Chronic kidney disease-mineral and bone disorder is a broad term used to describe a group of disorders that result from the complex interplay of several factors,

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Implication for health policy/practice/research/medical education

Chronic kidney disease-mineral and bone disorders is a complex and challenging condition to manage, and it requires close collaboration between nephrologists, endocrinologists, and surgeons. Early detection and appropriate management can help improve patient outcomes and prevent complications.

including alterations in mineral metabolism, changes in bone function, and the effects of multiple CKD-related comorbidities (8). CKD-MBD often develops earlier and progresses more rapidly in patients with CKD than in the general population, and its prevalence increases as kidney function declines (3, 9).

One of the primary causes of CKD-MBD is parathyroid gland dysfunction, which produces PTH. PTH regulates serum calcium and phosphorus levels, and when its production is altered, CKD-MBD can manifest in multiple ways. For example, as kidney function declines, the kidneys cannot excrete phosphate as effectively, leading to hyperphosphatemia and hypocalcemia, which stimulates the release of PTH (3,8,10).

Moreover, in the early stages of CKD, the parathyroid glands compensate for calcium loss through the kidneys by overproducing PTH. However, over time, the glands become resistant to PTH, leading to a drop in calcium levels and increased phosphorus levels in the blood. The resulting mineral imbalances can cause various complications of CKD, including osteoporosis, osteopenia, and vascular calcification (7).

The diagnosis of CKD-MBD often involves a combination of blood tests, measurements of the PTH levels, bone density assessments, and imaging studies. Treatment depends on the underlying cause of CKD-MBD, and interventions can include dietary modifications, oral medications, intravenous therapy, and surgical interventions. Treatment aims to restore the balance of calcium and phosphorus metabolism, control the adverse effects of elevated PTH levels, and prevent complications (11, 12).

Hyperparathyroidism

Hyperparathyroidism is a potential complication in patients with CKD due to an overproduction of PTH by the parathyroid glands. It is classified into two types: primary hyperparathyroidism and secondary hyperparathyroidism. Primary hyperparathyroidism occurs due to an autonomous overproduction of PTH by one or more enlarged parathyroid glands. Secondary hyperparathyroidism occurs due to decreased renal function leading to increased PTH secretion (13,14).

Clinical features of hyperparathyroidism include bone pain, fractures, muscle weakness, fatigue, and renal osteodystrophy. Treatment options include medical management with phosphate binders, vitamin D analogs, or surgical removal of enlarged glands (15).

Hypoparathyroidism

Hypoparathyroidism is a rare disorder due to decreased PTH secretion from the parathyroid glands. It can occur due to surgical removal of the parathyroid glands or autoimmune destruction of the glands. Clinical features include hypocalcemia, tetany, seizures, and neuromuscular irritability. Treatment involves calcium and vitamin D supplementation (16).

Pathophysiology

In CKD, there is a decrease in renal function, leading to an increase in serum phosphate levels and a reduction in serum calcium levels. This stimulates the secretion of PTH from the parathyroid glands, which acts on bone, kidney, and intestine to maintain calcium and phosphorus homeostasis. However, prolonged PTH secretion stimulation leads to various parathyroid gland disorders (3,17).

Management of CKD-MBD

Management of CKD-MBD involves balancing the levels of serum calcium, phosphorus, and PTH. The therapeutic goals include preventing fractures, educing cardiovascular morbidity and mortality, mproving patient quality of life.

Treatment options include dietary modifications, oral medications, intravenous therapy, and surgical interventions (11, 18). The first-line treatment for CKD-MBD often involves nutritional changes and oral medications, including calcium-containing phosphate binders, vitamin D analogs, and calcimimetics. In refractory cases, invasive therapies, including parathyroidectomy, may be necessary (19, 20).

Conclusion

CKD-MBD due to parathyroid gland dysfunction is a complex health condition that requires comprehensive management. The appropriate management of CKD-MBD may improve patient outcomes, reduce morbidity and mortality, and enhance patients' quality of life. Clinicians must be aware of the risk factors, screening tools, monitoring techniques, and treatment options for CKD-MBD to provide optimal patient care and prevent complications.

Authors' contribution

Conceptualization: AI.
Validation: MN.
Investigation: HS.
Resources: MA.
Data curation: AE and HS.
Visualization: MN.
Supervision: MA.
Funding acquisition: All authors.
Writing-original draft: AE, HS and AI.
Writing-review and editing: MN and MA.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

The authors have carefully adhered to ethical standards and avoided any potential ethical issues, such as plagiarism, data fabrication, or double publication, during the conduct and reporting of their study.

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