



Diseases of the parathyroid glands in chronic kidney disease

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Abstract

Chronic kidney disease (CKD) is a prevalent health condition that affects millions of people worldwide. One of the common complications of CKD is CKD-mineral and bone disorder (CKD-MBD), which involves the disturbances in mineral metabolism and skeletal health due to complications of CKD. The parathyroid glands, which is a small gland located near the thyroid glands in the neck, plays 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 disorder (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 the 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 (3,4). These changes can lead to various 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 the formation of a parathyroid adenoma or hyperplasia, which can further aggravate the CKD-MBD and require surgical intervention (5,6). This paper aims to discuss the different diseases of the parathyroid glands that occur in CKD.

Search strategy

For this mini-review, I conducted a comprehensive search using various databases: PubMed, Google Scholar, Directory of Open Access Journals (DOAJ), Web of

Science, EBSCO, Scopus, and Embase. We utilized different keywords such as Parathyroid glands, CKD-MBD, bone pain, Chronic kidney disease, parathyroid hormone, osteoporosis, cardiovascular disease, parathyroid hyperplasia, chronic kidney disease-mineral and bone disorders, parathormone, kidney and vitamin D.

Parathyroid Hormone

The parathyroid glands produce PTH, which plays a crucial role in calcium and phosphorus metabolism. PTH regulates the serum calcium and phosphorus levels by enhancing calcium reabsorption and inhibiting phosphorus reabsorption in the kidneys (7). In the early stages of CKD, the parathyroid glands compensate for the loss of calcium 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 an increase in phosphorus levels in the blood (4,8).

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, including alterations in mineral metabolism, changes in bone function, and the effects of multiple CKD-related comorbidities (9). CKD-MBD often develops earlier and

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

Chronic kidney disease-mineral and bone disorder 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.

progresses more rapidly in patients with CKD than the general population, and its prevalence increases as kidney function declines (3,10).

One of the primary causes of CKD-MBD is the dysfunction of the parathyroid glands, which produces the hormone 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 are unable to excrete phosphate as effectively, leading to hyperphosphatemia and hypocalcemia, which stimulates the release of PTH (3,9,11).

Moreover, in the early stages of CKD, the parathyroid glands compensate for the loss of calcium through the kidneys by overproducing PTH. However, over time, the glands become resistant to PTH, leading to a drop in calcium levels and an increase in phosphorus levels in the blood. The resulting mineral imbalances can cause various complications of CKD, including osteoporosis, osteopenia, and vascular calcification (4,8).

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 (12,13).

Hyperparathyroidism

Hyperparathyroidism is a common disorder that occurs in CKD due to increased PTH secretion from 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 (4,14,15).

Clinical features of hyperparathyroidism include bone pain, fractures, muscle weakness, fatigue, and renal osteodystrophy. Treatment options include medical management with phosphate binders and vitamin D analogs or surgical removal of enlarged glands (16).

Hypoparathyroidism

Hypoparathyroidism is a rare disorder that occurs due to decreased PTH secretion from the parathyroid glands. It can occur as a result of 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 (17,18).

Pathophysiology

In CKD, there is a decrease in renal function, leading to an increase in serum phosphate levels and a decrease 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 stimulation of PTH secretion leads to various disorders of the parathyroid glands (3,19).

Management of CKD-MBD

Management of CKD-MBD involves controlling the serum levels of calcium, phosphorus, and PTH. The therapeutic goals include preventing fractures, reducing cardiovascular morbidity and mortality, and improving patient quality of life. Treatment options include dietary modifications, oral medications, intravenous therapy, and surgical interventions (13,20). The first-line therapy for CKD-MBD often involves dietary modifications and oral medications, including calcium-containing phosphate binders, vitamin D analogs, and calcimimetics. In refractory cases, invasive therapies, including parathyroidectomy, may be necessary (21,22).

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 need to be aware of the risk factors, screening, monitoring, and treatment options available for CKD-MBD to ensure optimal patient management.

Conflicts of interest

The author declares that he has no competing interests.

Ethical issues

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

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