

Osteoporosis in Chronic Kidney Disease: Mechanisms and Management

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ABOVE THE STUDY

Osteoporosis is a well-recognized complication of Chronic Kidney Disease (CKD), contributing significantly to morbidity through increased fracture risk, reduced quality of life, and higher mortality rates. The interplay between CKD and bone health is complex, involving disturbances in mineral metabolism, hormonal imbalances, and alterations in bone remodeling. This condition is often encompassed within the broader spectrum of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD), which includes abnormalities in calcium, phosphorus, Parathyroid Hormone (PTH), and vitamin D metabolism, along with changes in bone turnover and vascular calcification.

One of the central mechanisms underlying osteoporosis in CKD is disrupted calcium and phosphate homeostasis. As kidney function declines, phosphate excretion is impaired, leading to hyperphosphatemia. This triggers increased secretion of Fibroblast Growth Factor 23 (FGF23) and PTH, both of which aim to restore mineral balance but inadvertently contribute to bone demineralization. Elevated PTH levels, characteristic of secondary hyperparathyroidism, stimulate bone resorption, resulting in decreased Bone Mineral Density (BMD) and structural deterioration.

Vitamin D deficiency is another critical factor. The kidneys play a vital role in converting inactive vitamin D into its active form, calcitriol. In CKD, this conversion is impaired, leading to reduced intestinal calcium absorption and further stimulation of PTH secretion. This cascade exacerbates bone loss and weakens skeletal integrity. Additionally, metabolic acidosis, commonly observed in advanced CKD, promotes bone buffering of excess hydrogen ions, further contributing to bone mineral depletion.

Bone turnover abnormalities in CKD are heterogeneous, ranging from high-turnover bone disease (osteitis fibrosa) due to excessive PTH activity to low-turnover conditions such as adynamic bone disease. The latter is particularly concerning because it is associated with reduced bone formation and impaired repair mechanisms, increasing susceptibility to fractures. Differentiating between these conditions is essential, as management strategies differ significantly.

The diagnosis of osteoporosis in CKD presents unique challenges. Dual-Energy X-ray Absorptiometry (DEXA) scans are commonly used to assess BMD; however, they may not fully capture bone quality or turnover status in CKD patients. Bone biopsy remains the gold standard for evaluating bone histology but is invasive and not routinely performed. Emerging biomarkers and imaging techniques are being explored to improve diagnostic accuracy.

Management of osteoporosis in CKD requires a multifaceted approach tailored to the stage of kidney disease and underlying bone pathology. Controlling mineral metabolism is fundamental. Dietary phosphate restriction, phosphate binders, and vitamin D supplementation are commonly employed to maintain serum phosphate and calcium levels within target ranges. Active vitamin D analogs and calcimimetics may be used to suppress secondary hyperparathyroidism.

Pharmacological treatment of osteoporosis in CKD must be approached with caution. Bisphosphonates, widely used in the general population, are renally excreted and may accumulate in patients with reduced kidney function, raising concerns about safety and efficacy. Denosumab, a monoclonal antibody that inhibits osteoclast activity, has shown promise as it is not cleared by the kidneys, but careful monitoring for hypocalcemia is. Anabolic agents such as teriparatide may be beneficial in selected cases, particularly in low-turnover bone disease, but their use in CKD remains limited and requires further study.

Non-pharmacological interventions are equally important. Adequate nutrition, weight-bearing exercise, and fall prevention strategies play a crucial role in reducing fracture risk. Regular monitoring of biochemical parameters and bone health is essential to guide therapy and prevent complications.

In conclusion, osteoporosis in CKD is a multifactorial condition driven by complex metabolic and hormonal disturbances. Effective management requires an integrated approach that addresses both bone health and underlying kidney dysfunction. Continued research is needed to refine diagnostic tools and develop targeted therapies that can safely and effectively reduce fracture risk in this vulnerable population.

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