Renal Osteodystrophy

Renal osteodystrophy is defined by pathological bone features in a patient with chronic kidney disease. It's a component of the spectrum of chronic kidney disease - mineral bone disorders (CKD-MBD). The diseased kidney results in a decreased ability to excrete phosphate. As phosphate builds up, it triggers growth factors to be released from the bone that inhibit calcitriol. Inhibited calcitriol leads to hypocalcemia and a compensatory parathyroid response, hence secondary hyperparathyroidism. Hyperphosphatemia and hypocalcemia are characteristic, as is decreased calcitriol. Clinical features include osteitis fibrosa cystica, osteoporosis, and osteomalacia. Management is aimed at treating the underlying kidney disease.



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Pathophysiology

Chronic Kidney Disease

Crone and Kidney

CKD results in decreased phosphate excretion and reduced calcitriol production. Hyperphosphatemia stimulates FGF-23 release, which inhibits 1?-hydroxylase in the kidney, decreasing calcitriol synthesis. Low calcitriol reduces intestinal calcium absorption, leading to hypocalcemia. Hypocalcemia and phosphate retention together drive secondary hyperparathyroidism.

Secondary Hyperparathyroidism

(2) Tutu Hiker-para-thigh-droid

Secondary hyperparathyroidism is a compensatory increase in parathyroid hormone (PTH) secretion in response to CKD-induced hypocalcemia and hyperphosphatemia. Chronic stimulation may progress to tertiary hyperparathyroidism, where parathyroid glands become autonomous, causing hypercalcemia and soft tissue calcifications.

Hyperphosphatemia

Hiker-phosphate-P

Due to phosphate retention and decreased calcitriol, serum calcium is low, which triggers secondary hyperparathyroidism.

Hypocalcemia

Hippo-calcified-cow

Hyperphosphatemia induces the release of fibroblast growth factor 23 from the bone. FGF-23 is an important mediator of phosphate homeostasis, which helps increase the excretion of phosphate via urine in hyperphosphatemia. It also reduces the production and function of active vitamin D, Calcitriol, which decreases calcium absorption, resulting in hypocalcemia.

Decreased Calcitriol

Down-arrow Calcified-troll

CKD decreases conversion of vitamin D to active 1,25-dihydroxyvitamin D, reducing intestinal calcium absorption and contributing to secondary hyperparathyroidism.

Clinical Features

Osteitis Fibrosa Cystica

Ostrich-in-flames Fibrous-sacks Sisters

This is the classic bone manifestation of secondary hyperparathyroidism in CKD. PTH increases osteoclast activity, causing subperiosteal bone resorption (especially in the phalanges), cortical thinning, and brown tumors (collections of fibrous tissue + hemosiderin from microhemorrhages). Patients present with bone pain and are at increased risk of fractures.

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Osteoporosis

Ostrich-with-porous-bones

Renal osteodystrophy can lead to osteoporosis due to high bone turnover and impaired mineralization, characterized by trabecular thinning and increased fracture risk.

Osteomalacia

Ostrich-Malaysia

Decreased calcitriol causes impaired mineralization of bone matrix, leading to osteomalacia, which presents as diffuse bone pain and skeletal weakness.

Management

Treating Underlying Disorder

Treating Disorders Under the Tent

Management of renal osteodystrophy in chronic kidney disease focuses on correcting mineral imbalances through a combination of strategies: controlling phosphate levels via dietary restriction (maintaining serum phosphate <5.5 mg/dL) and using phosphate binders (calcium-based such as calcium acetate or carbonate, or non-calcium agents like sevelamer and lanthanum); supplementing vitamin D with calcitriol or analogs (e.g., paricalcitol) to enhance calcium absorption; addressing secondary hyperparathyroidism with calcimimetics such as cinacalcet or, in refractory cases, parathyroidectomy; and providing additional supportive care, including sodium bicarbonate for metabolic acidosis and bisphosphonates when osteoporosis is severe.

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