

## Renal Osteodystrophy

Renal osteodystrophy is defined by pathological bone features in a patient with chronic kidney disease/failure. 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.



PLAY PICMONIC

### Pathophysiology

#### Chronic Kidney Disease

##### [Crone and Dead Kidney](#)

Chronic kidney failure is characterized by the inability to excrete phosphate normally. Over time, this will result in a high phosphate level in the blood, which triggers the secretion of fibroblast growth factor (FGF) from the bone. FGF-23 can decrease calcitriol production and intestinal calcium absorption. Hypocalcemia then causes a compensatory PTH response.

#### Secondary Hyperparathyroidism

##### [\(2\) Tutu Hiker-para-thigh-droid](#)

Abnormalities found in renal osteodystrophy will trigger the parathyroid glands to secrete more parathyroid hormone to maintain homeostasis. This is also known as secondary hyperparathyroidism. If left untreated, secondary hyperparathyroidism can develop into tertiary hyperparathyroidism over many years. Tertiary hyperparathyroidism is a condition where hyperparathyroidism is refractory due to chronic renal disease. These will lead to high phosphate and calcium, resulting in deposits in the blood vessels and soft tissues.

#### Hyperphosphatemia

##### [Hiker-phosphate-P](#)

The inability of the kidney to excrete phosphate in renal failure causes hyperphosphatemia. This will initiate the onset of CKD-related mineral bone disorder.

#### 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](#)

Chronic kidney failure also impairs the hydroxylation of vitamin D into calcitriol, which decreases intestinal calcium absorption. Patients with have decreased 1,25-dihydroxyvitamin D, or calcitriol.

### Clinical Features

#### Osteitis Fibrosa Cystica

##### [Ostrich-in-flames Fibrous-sacks Sisters](#)

Osteitis fibrosa cystica can occur in up to 50% of renal osteodystrophy cases due to excess parathyroid hormone production. Increased osteoclast activity is the root of this process. It manifests as bone pain and is characterized by cystic bone spaces loaded with brown fibrous tissue. The brown color appears due to hemosiderin deposits from microhemorrhages. It can be present with subperiosteal bone resorption, which leads to cortical thinning.

### **Osteoporosis**

#### [Ostrich-with-porous-bones](#)

Renal osteodystrophy can lead to osteoporosis. This will increase the risk of fracture in patients. Osteoporosis is characterized by bone loss and trabecular thinning with fewer interconnections.

### **Osteomalacia**

#### [Ostrich-Malaysia](#)

Osteomalacia can be seen as a part of the spectrum of bone abnormalities in renal osteodystrophy. Osteomalacia is characterized by impaired bone mineralization, which can manifest as bone pain.

## **Management**

### **Treating Underlying Disorder**

#### [Treating Disorders Under the Tent](#)

Treatment of renal osteodystrophy is aimed at treating the underlying disorder. Dietary phosphate restriction is recommended in chronic kidney failure patients. Phosphate serum level should be below 5.5. mg/dL. A vegetarian diet helps restricts phosphate due to the low bioavailability of phosphate in vegetarian protein. If dietary phosphate restriction is not effective in reducing phosphate level, a phosphate binder may be indicated. Phosphate binders can be calcium-containing or non-calcium containing. Calcium-containing includes calcium carbonate and calcium acetate. Non-calcium containing agents include sevelamer and lanthanum. Vitamin D2 is recommended for patients with vitamin D below 30 ng/mL. Calcitriol is a potent vitamin D analog. Other options that can be considered are cinacalcet, sodium bicarbonate, bisphosphonates, and surgical parathyroidectomy.