International Scholars Journals

African Journal of Medical Case Reports ISSN 2756-3316 Vol. 9 (2), pp. 001-002, September, 2021.
Available online at www.internationalscholarsjournals.com © International Scholars Journals

Editorial

The mechanism of renal osteodystrophy

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Accepted 16 September,, 2021

EDITORIAL NOTE

A change in bone shape in patients with chronic kidney disease is now characterised as renal osteodystrophy bone disease. It is one marker of the skeletal component of chronic renal disease's systemic condition-mineral and bone disorder. CKD-MBD, on the other hand, is characterised as a systemic impairment of mineral and bone metabolism caused by CKD and manifested by one or more of the following symptoms and vascular or other soft-tissue calcification and abnormalities in bone turnover, mineralization, volume, linear growth, or strength (renal osteodystrophy) and abnormalities in calcium, phosphorus, PTH, or vitamin D metabolism (Hruska, 1995).

Renal osteodystrophy has traditionally been defined as the outcome of hyperparathyroidism caused by hypophosphatemia and hypocalcaemia, both of which are caused by decreased phosphate excretion by the injured kidney. Low levels of activated vitamin D3 come from the injured kidneys' failure to convert vitamin D3 to its active form, calcitriol, leading to additional hypocalcaemia. The most major reason of low calcitriol levels in CKD patients now appears to be high levels of fibroblast growth factor 23.

The increased production of parathyroid hormone in CKD accelerates bone resorption and leads to secondary hyperparathyroidism histological bone findings. In some cases, however, a variety of circumstances, such as age, ethnic origin, sex, and therapies such as vitamin D, adynamic bone disease, may cause the first increase in parathyroid hormone and bone remodelling to be unduly retarded (Moe, 2006).

In CKD patients on dialysis, both high and low bone turnover diseases are now seen equally, and all kinds of renal osteodystrophy are linked to an increased risk of skeletal fractures, a lower quality of life, and poor clinical outcomes. Renal osteodystrophy is normally discovered after end-stage renal disease treatment has begun; however, condition-mineral and bone disorder appears early in the course of CKD. Blood tests in the advanced phases will reveal lower calcium and calcitriol levels, as well as higher phosphate and parathyroid hormone levels. Serum calcium and phosphate levels are normal in the early stages, but parathyroid hormone levels are elevated (Moe, 2006).

Renal osteodystrophy is also visible on X-rays; however it can be difficult to distinguish from other disorders. Because existing clinical, biochemical, and imaging approaches cannot accurately diagnose these bone abnormalities, bone biopsy has been, and continues to be, the gold standard analysis for determining the kind of renal osteodystrophy. Renal osteodystrophy must be described by assessing bone turnover, mineralization, and volume to validate the diagnosis. If a mass-forming lesion is present, all forms of renal osteodystrophy should be distinguished from other bone diseases that can cause decreased bone density, such as osteoporosis, osteoopenia, and osteomalacia. If a mass-forming lesion is present, brown tumour should be considered the top-line diagnosis (Fukagawa, 2002).

Renal osteodystrophy has been seen to improve after kidney donation. Renal osteodystrophy is a chronic disorder that requires haemodialysis on a regular basis. However, it's crucial to remember that condition-mineral and bone disorder, which includes renal osteodystrophy, is linked to not only bone disease and increased fracture risk, but also cardiovascular calcification, poor quality of life, and higher morbidity and mortality in condition-mineral and bone disorder patients. In fact, bone is now thought to represent a novel endocrine organ at the centre of condition-mineral and bone disorder.

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