



Bone enumerates as a new endocrine organ interacted in chronic kidney disease and mineral and bone disorders

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Perturbation of mineral metabolism and bone disease are popular complications in chronic kidney disease patients (1-3). This condition is associated with increased morbidity and mortality and diminished quality of life. Additionally it has been detected that these disorders are causally related to numerous adverse clinical outcomes, especially cardiovascular disease and increased bone fracture risk. However, disturbed concentrations of humoral factors are not only epidemiologically connected to cardiovascular morbidity and mortality, but can also be causally implicated, particularly in chronic kidney disease (2-6).

In fact, the increased knowledge of the potential role played by mineral and bone disorder in the appearance of cardiovascular disease in renal disease patients has generated investigation efforts intended at discovering possible pathogenic links (2-5).

In chronic kidney disease, abnormalities in circulating parameters of mineral and bone metabolism such as serum phosphorous, vitamin D, calcium, parathyroid hormone and fibroblast growth factor 23 are frequently present and associated with adverse clinical consequences far beyond renal osteodystrophy (3-8). Chronic kidney disease progresses to more advanced stages in a small, but significant percentage of people. When chronic kidney disease stage 5 advances to end-stage renal disease, some people progress to renal replacement therapy. When renal dysfunction advances, there is an elevated hazard of mortality and various comorbidities become more serious (2-6). Hyperphosphatemia arises because of inadequate filtering of phosphate from the blood by poorly functioning kidneys. This denotes that a certain amount of the phosphate does not leave the body in the urine, instead staying in the blood at abnormally raised levels (5-8). High serum phosphate levels is able to directly and indirectly increase parathyroid hormone (PTH) secretion, resulting to the development of secondary hyperparathyroidism. However, abnormalities in bone and mineral metabolism commence to develop in the early stage of chronic kidney disease. Serum phosphorus, calcium, parathormone and alkaline phosphatase are recommended to be assessed from chronic kidney disease stage 3, and it is recommended

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

By definition, chronic kidney disease and mineral and bone disorders represent a synopsis of three closely related disease circumstances, laboratory abnormalities indicative of disturbed bone and mineral metabolism, renal osteodystrophy explaining the variety of bone lesion subtypes happening in chronic kidney disease, cardiovascular disease demonstrating left ventricular hypertrophy, accelerated arteriosclerosis and other pathologies in the vasculature and the heart in patients with chronic kidney disease.

that these markers be kept within the reference ranges of each facility. The management of phosphorus is principal and is combined of phosphorus restriction and phosphate binders (7-11).

Indeed, phosphorus retention plays a pivotal role in the beginning of mineral and bone disorders in chronic kidney disease. Phosphorus retention usually happens as a result of net intestinal absorption exceeding kidney excretion or dialysis removal. Applicable strategies to control dietary phosphorus intake contain restricting phosphorus-rich foods, preferring phosphorus sourced from plant origin, boiling as the preferred cooking procedure and avoiding foods with phosphorus-containing additives (4-12).

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Various investigations had detected that, fibroblast growth factor 23, which increases with progressing chronic renal failure, provokes left ventricular hypertrophy, and is possibly interacted in the process of vessel calcification.

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Additionally, Sclerostin and Dickkopf (DKK1), both secreted mainly by osteocytes, are important wnt-type (Wnt) inhibitors and as such can interfere with systems for biological signaling which operate in the vessel wall (10-14). Osteocalcin, generated by osteoblasts or released from mineralized bone, interferes with insulin concentrations and sensitivity, and its metabolism is perturbed in renal disease (11-15). These bone-derived humoral factors may place the bone at the center of cardiovascular disease accompanying with chronic renal failure (13-15). Most significantly, factors that dictate the regulation of these elements in bone and subsequent secretion into the circulation have not been investigated, and could provide entirely new opportunities for therapeutic intervention.

Authors' contributions

All authors contributed to paper equally.

Ethical considerations

Ethical issues (including plagiarism, informed consent, misconduct, double publication and redundancy) have been completely observed by authors.

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