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Perspective

Phosphate Homoeostasis in Individuals with Chronic Renal Disease's Risk of Bone Fracture

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DESCRIPTION

Phosphate homoeostasis, along with low serum calcium and high levels of parathyroid hormone, fibroblast growth factor 23, and sclerostin, are key factors in the development of Mineral and Bone Disorders (MBD) in Chronic Kidney Disease (CKD).

Although phosphorus is necessary for life, both high and low serum phosphate levels are linked to unfavorable consequences. The homoeostatic equilibrium between dietary intake, intestinal absorption, and renal excretion is what controls the phosphate pool in humans. The severe cellular and organ dysfunction seen in people with hereditary abnormalities of phosphate metabolism suggests that the clinical effects of chronic hypophosphatemia may be significant. On the other hand, if the phosphate balance is positive, reducing dietary phosphate intake might be the simplest method to influence it through preventive and therapeutic interventions. However, according to the Kidney Disease Improving Global Outcomes (KDIGO) recommendations, reducing dietary phosphate intake is still rated as having low evidence (2D).

A 70 kg healthy man typically stores 700 mg of phosphorus, which is mostly distributed as hydroxyapatite in skeletal tissues (85%) and organic phosphorus in the intracellular space (14%), including nucleic acid, ATP, phospholipids, creatine phosphate, and phosphoproteins. Because it only makes up 1% of the body's total phosphate, circulating phosphorus serves as a poor indicator of both the body's phosphate pool and its compartmental distribution. When there are high amounts of calcitriol in the blood, up to 80% of the dietary phosphate is absorbed in the intestine as inorganic phosphate. Importantly, the KDIGO guidelines highlight in a not-graded consideration for phosphate source in generating dietary recommendations that phosphorus contained as an addition in commercial food has 100% bioavailability.

The key factors affecting intestinal phosphorus absorption are vitamin D activity, the amount of phosphate in the food, phosphate bioavailability, and the usage of phosphate binders. 1,25-dihydroxy vitamin D (calcitriol), Parathyroid Hormone (PTH), Fibroblast Growth Factor 23 (FGF-23), and Klotho are some of the hormones that control phosphorus homoeostasis. The way these factors interact varies depending on the degree of Chronic Kidney Disease (CKD). Particularly, when stages 4 and 5 of CKD develop, normal phosphate levels can be maintained because FGF-23 and PTH levels rise while calcitriol and Klotho levels fall. Observational studies have shown that very high serum phosphate levels in people with CKD Stage 5D (dialysis patients) are linked to increased vascular calcification, bone disease, and shorter survival. Additionally, compared to those who weren't receiving treatment, CKD and dialysis patients who took oral phosphate binders demonstrated a decreased mortality risk. It should be stressed that the general population also follows the same rule reduced mortality with lower phosphate levels within the normal range.

The evaluation of bone turnover, mineralization, and volume is necessary for the classification of Renal Osteodystrophy (ROD). ROD's clinical aftereffects include bone discomfort and fractures. The risk of fracture rises as kidney function decreases until it reaches the rates seen in dialysis patients with Stage 4 CKD. ROD is associated with a number of histological abnormalities, and it has been shown that these lesions change as CKD progresses through the various phases.

Bone quality and mass affect bone strength (and hence fracture risk). Dual-energy X-ray absorptiometry (DXA) is used to determine the bone mass (or density). The most recent KDIGO guidelines, which were updated in 2017, propose using DXA to predict fracture in patients with CKD since low Bone Mineral Density (BMD) evaluated by DXA at the forearm, hip, and spine indicates future fracture. DXA, however, is unable to determine the type of ROD that is present or the quality of the bone tissue.

Measures of bone quality reflect bone's material characteristics, such as bone turnover, microarchitecture, mineralization, and collagen properties. The KDIGO recommendations support the use of bone histomorphometry to rule out unusual or unusual bone pathology, identify a mineralization deficit that could influence therapy choices, and assess if the patient has a high or low-turnover condition. As a result, the gold standard for assessing bone quality is a tetracycline double-labeled trans-iliac crest bone sample. The classic histological abnormalities of

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CKD-MBD include mixed uraemic osteodystrophy, Adynamic Bone Disease (ABD), osteomalacia, and hyperparathyroid bone disease. Unfortunately, due to the invasive nature of bone biopsies and the specific expertise needed to administer them, they are not routinely carried out in clinical practice. Additionally, bone histomorphometric analysis takes time and requires specialized knowledge. Another thing to keep in mind is that a bone biopsy only tells us about the quality of bone at one specific location (the iliac crest), at one specific time, and tells us very little about cortical bone. It has been shown that when kidney function decreases, the type of ROD alters, indicating the intricacy of CKD-MBD and its progression across the stages of CKD.

However, aberrant bone turnover has been the main focus of bone quality in the majority of studies, despite the fact that it is well-established that CKD also directly impacts the composition and quality of bone through altered mineral and vitamin D metabolism. In healthy bone, osteoclasts and osteoblasts work together to select portions of the aged or damaged bone for turnover as part of an ongoing repair process. Disruption of bone turnover in CKD might show up as high or low turnover.

Limited research has been done on the independent function that phosphate plays in determining bone fractures. Phosphate levels are typically higher in hemodialysis patients who have experienced fragility fractures in the past. In patients with normal renal function and, most crucially, even when blood phosphate levels are within the normal range, there is a relationship between greater serum phosphate levels and a higher risk of bone fracture. After adjusting for FGF-23 and PTH levels in the MrOS, the link between phosphate levels and fracture risk was still significant in CKD patients, indicating that elevated phosphate levels alone and no other mechanisms may account for the higher fracture risk in this population.