

Opinion Article

Bone Mineral Metabolism in Kidney Transplantation Patients

Mark Boldin^{*}

Department of Molecular and Cellular Biology, Beckman Research Institute, California, USA

DESCRIPTION

A life-threatening consequence in people with Chronic Kidney Disease (CKD) is Mineral and Bone Disorder (MBD). Disturbances in mineral metabolism, osteodystrophy, and extraskeletal calcification are the three changes that make up MBD. The development of hyperphosphataemia, increases in the phosphatonins Fibroblast Growth Factor (FGF)-23 and Parathyroid Hormone (PTH), decreased levels of active 1,25dihydroxyvitamin D in the blood, and hypo or hypercalcemia are all abnormalities in mineral metabolism in CKD. Even with relatively little decreases in Glomerular Filtration Rate (GFR), these pertubations are visible. According to epidemiological data, mineral diseases including hyperparathyroidism and hyperphosphataemia are linked to higher rates of morbidity and mortality, particularly from Cardiovascular Disease (CVD). In reality, there is a direct connection between the development of MBD and CVD, and it is crucial to comprehend this nuanced interaction given that CVD continues to be the main cause of death in patients with CKD.

Alterations in bone shape and turnover that enhance the risk of fracture and may be a factor in extraskeletal calcification are the characteristics of skeletal diseases in CKD. Notably, extensive vascular and soft tissue calcifications are linked to CKD-MBD. The increased cardiovascular mortality seen in patients with renal impairment is mostly attributed to arterial calcification, which is directly related to arterial stiffness. Calcium and phosphate homeostasis, which is primarily controlled by vitamin D, PTH, FGF23, and klotho, is closely related to both skeletal health and extraskeletal mineralization. These bone mineral metabolism regulators respond to modifications in kidney function and engage in a variety of endocrine feedback loop interactions with calcium and phosphate concentrations as well as with one another. To help with the design of therapies to lessen the cardiovascular and skeletal consequences of CKD-MBD, it is crucial to identify the fundamental abnormality resulting from a decrease in eGFR and driving aberrant mineral metabolism.

In the early and late post-transplant period, mineral and bone disorders in kidney transplant patients is a significant cause of morbidity and mortality. Bone Mineral Density (BMD) evaluation is advised to predict fracture risk in kidney transplant recipients, particularly in the first three months following transplantation, according to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. This section specifically addresses the management of CKD-MBD in transplant patients. This assessment is crucial for identifying patients who can receive osteoporosis medication in an effort to avoid fractures. The National Osteoporosis Foundation advises using any of the following standards to establish treatment eligibility for the general public: T-scores of less than or equal to -2.5 at the femoral neck, total hip, or lumbar spine; prior vertebral or hip fractures; and 10-year Major Osteoporotic Fracture (MOF) or hip fracture probability of greater than or equal to 20% or 3%, respectively. While the efficacy of BMD evaluations in transplant patients is widely acknowledged, the significance of screening for vertebral fractures is still largely underappreciated in transplant practise. The most frequent osteoporotic fractures seen are in vertebral bones, and osteoporosis increases the risk of fractures in the entire population. Osteoporotic vertebral fractures not only show weakened bones but also raise the possibility of death and further fractures. The reported frequency of vertebral fractures in kidney transplant recipients ranges from 15% to 66.0%, depending on the diagnostic criteria, suggesting that the prevalence of these injuries is highly variable. Data on the relationship between lower BMD and the prevalence of fractures in transplant patients are scarce, and it is still unclear how valuable BMD and vertebral fracture assessments are beyond the first year following a kidney donation. The Fracture Risk Assessment Tool (FRAX) has not been validated in transplant recipients for the 10-year risk of hip and MOF, and clinical investigations are required to verify the efficacy of these tools. The Fracture Risk Assessment Tool (FRAX) of the World Health Organization determined the patient's fracture risk based on the patient's age, sex, and clinical risk factors. Based on the Turkish FRAX tool, the ten-year chance of a Major Osteoporotic Fracture (MOF) and hip fracture was determined (FRAX Web, version 4.1). High risk was defined as MOF risk above 20% and hip fracture risk over 3%. The fracture rates recorded in the overall Turkish population closely match FRAX forecasts.

Although there is ample evidence linking transplant recipients to general risk factors for low bone density and vertebral fractures,

Citation: Boldin M (2022) Bone Mineral Metabolism in Kidney Transplantation Patients. J Bone Res. 10:198.

Copyright: © 2022 Boldin M. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Correspondence to: Mark Boldin, Department of Molecular and Cellular Biology, Beckman Research Institute, California, USA, E-mail: boldinmark.res@gmail.com

Received: 01-Nov-2022; Manuscript No. BMRJ-22-20954; Editor assigned: 03-Nov-2022; PreQC. No. BMRJ-22-20954 (PQ); Reviewed: 17-Nov-2022; QC. No. BMRJ-22-20954; Revised: 24-Nov-2022; Manuscript No. BMRJ-22-20954 (R); Published: 02-Dec-2022, DOI: 10.35248/2572-4916.22.10.198.

Boldin M

the incidence of these risks and the risk factors that are unique to particular transplants can differ. Although vertebral fractures and low BMD are more frequently observed in women, numerous studies have found that men are also at a high risk of developing bone metabolism abnormalities.