Chronic kidney disease–mineral and bone disorder (CKD–MBD) is a recently acknowledged metabolic abnormality, proposed in 2006 by the Kidney Disease: Improving Global Outcomes (KDIGO) working group.The latter includes regular dialysis to remove blood phosphorus, which is considered to be of limited value in removal of phosphates, highlighting the importance of dietary restriction.The clinical features of CKD–MBD are primarily due to the compromised renal excretion of minerals, leading to abnormality in circulating calcium, phosphorous, parathyroid hormone (PTH), and vitamin D. The secondary effects of this renal insufficiency include abnormalities in bone turnover, mineralization, volume, linear growth, strength, and ectopic calcification (vascular and soft tissues).This distinct naming of varied clinical syndromes (CKD–MBD) that develop due to impaired mineral regulation in chronic kidney disease (CKD) was to differentiate it from the histologically well–defined renal osteodystrophy1.Hyperphosphatemia in patients with CKD, apart from inducing secondary hyperparathyroidism and renal osteodystrophy, CV calcification is also an important prognostic factor for morbidity and mortality in patients with end–stage renal disease (ESRD) undergoing dialysis.CKD–MBD is considered to be a progressive disorder, affecting the homeostasis among the renal, skeletal, and cardiovascular (CV) systems, thereby leading to a kidney–bone–vascular axis pathophysiological hypothesis.The prognostic role of serum phosphorous as a modifiable risk factor has also been reported in multiple observational studies, highlighting the potential effect of lowering blood phosphorous (Pi and phosphates) in CKD–MBD.