



Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care

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Chronic Kidney Disease-Mineral Bone Disorder in South East Asia: a Narrative Review

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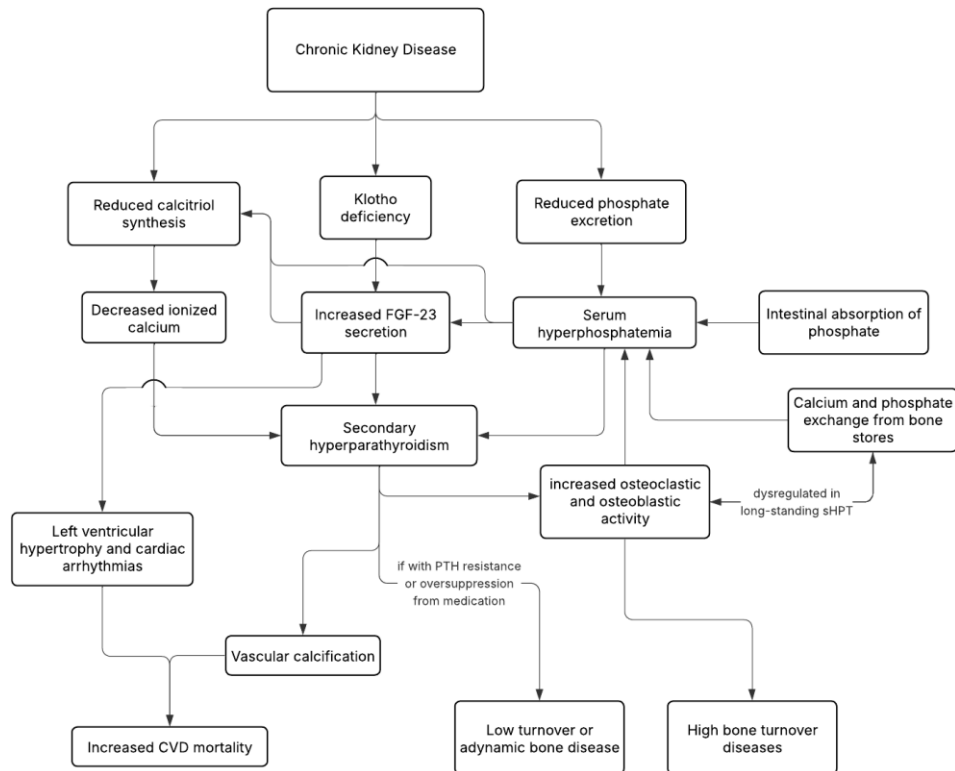
Objectives : Chronic kidney disease–mineral bone disorder (CKD-MBD) is a systemic complication of CKD characterized by dysregulated mineral metabolism, abnormal bone turnover, and vascular or soft tissue calcification, collectively increasing fracture risk, impairing functional capacity, and raising cardiovascular mortality. CKD burden in Southeast Asia (SEA) is high and growing, yet CKD-MBD remains under-recognized due to limited access to biochemical testing, imaging, and the heterogeneous socioeconomic conditions across the region. This narrative review synthesizes current evidence on the prevalence, pathophysiology, and management of CKD-MBD, contextualized with SEA data.

Methods : A structured literature search of international and regional databases using predefined terms was narratively synthesized.

Results : CKD-MBD arises from progressive phosphate retention, reduced calcitriol production, and impaired Klotho expression, driving secondary hyperparathyroidism, high- or low-turnover bone disease, and extra-skeletal calcification. Diagnosis relies on serial serum calcium, phosphate, parathyroid hormone (PTH), and vitamin D measurements, supplemented by bone density imaging and vascular calcification assessment. Access to intact PTH assays, DXA, and advanced imaging remains variable, particularly in low- and lower-middle-income settings. Management centers on maintaining near-normal serum calcium and phosphate through dietary phosphate restriction, phosphate binders, vitamin D sterols and analogues, and parathyroidectomy in refractory cases. Delivery of standard therapy is frequently constrained by inadequate health financing. Prognosis correlates with severity of mineral disturbances; geographic and financial barriers in SEA likely contribute to more pronounced biochemical derangements and higher CKD-MBD–related morbidity.

Conclusions : This review highlights critical gaps in early CKD detection, CKD-MBD monitoring, and access to cost-effective therapies. Region-specific implementation of guideline-based care and local economic evaluations are urgently needed. Future research should define CKD-MBD prevalence in SEA, evaluate non-imaging prognostic tools for low-resource settings, and conduct pragmatic trials comparing local interventions against global recommendations.

CKD-MBD Pathophysiology Concept Map.png



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Table 1. Summary of Treatment Modalities

Treatment Modality	Mechanism	Representative Interventions	Local Availability
Diet restriction	Avoidance of most common sources of phosphate	Individualized low-phosphate diet such as restriction of processed foods, cola beverages, dairy, meats	Low-cost intervention, best implemented with renal dietitians
Phosphate Binders	Binds enterally to dietary phosphate preventing absorption	Calcium carbonate, sevelamer, lanthanum carbonate, aluminum hydroxide	Calcium-based binders widely available and affordable with sevelamer variably available
Vitamin D Receptor Activators	Binds the vitamin D receptor (VDR) in the parathyroid gland and intestine, suppressing PTH transcription and increasing intestinal calcium absorption	Calcitriol, Paricalcitol, alfacalcidol	Calcitriol widely available and affordable
Calcimimetics	Allosteric activation of the calcium-sensing receptor (CaSR) on the parathyroid gland, suppressing PTH secretion	Cinacalcet, etelcalcetide	Cinacalcet available but costly
Parathyroidectomy	Directly mitigates the source of secretion of parathyroid hormone	Subtotal or total parathyroidectomy with autotransplantation	Mostly available in tertiary and academic institutions