DOI: https://doi.org/10.4038/jccp.v56i2.8165

Rational use of vitamin D analogues and calcium carbonate in chronic kidney disease-mineral bone disorder (CKD-MBD): a Sri Lankan perspective

Rajakrishna PN [1]

Journal of the Ceylon College of Physicians, 2025, **56**, 179-182

Abstract

The empirical use of vitamin D analogues and calcium carbonate remains common among patients with chronic kidney disease in Sri Lanka. These agents are often prescribed without biochemical evidence of secondary hyperparathyroidism or hyperphosphataemia, which can cause hypercalcaemia, vascular calcification, adynamic bone disease and increased cardio-vascular morbidity and mortality. This article summarises a practical guideline for managing chronic kidney disease-mineral bone disorder in Sri Lanka, based on the Kidney Disease: Improving Global Outcomes (KDIGO) 2017 Guideline, and highlights a rational, step-by-step approach that uses monitoring of alkaline phosphatase, calcium, and phosphate, rather than unavailable parathyroid hormone assays.

Introduction

Chronic kidney disease-mineral bone disorder (CKD-MBD) is a systemic condition affecting calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism, characterised by biochemical abnormalities, bone disease, and vascular calcification. CKD-MBD arises from impaired phosphate excretion, decreased renal activation of vitamin D, and secondary hyperparathyroidism. Elevated phosphate and fibroblast growth factor-23 (FGF-23) suppress calcitriol synthesis, leading to hypocalcaemia and a compensatory increase in PTH, which contributes to abnormal bone remodelling and vascular calcification. Phosphate binders, such as calcium carbonate, help regulate serum phosphate levels by reducing intestinal absorption, thereby limiting phosphate-induced vascular toxicity. Nutritional vitamin D (cholecalciferol) corrects deficiency and supports calcium balance, while active vitamin D analogues (e.g., alfacalcidol, calcitriol and paricalcitol) directly inhibit excessive PTH secretion.

Calcitriol (1,25-dihydroxyvitamin D_3) acts directly on the vitamin D receptor without needing renal activation. Alfacalcidol (1-α-hydroxyvitamin D_3) is a synthetic vitamin D analogue that requires activation in the liver to become calcitriol (1,25-dihydroxyvitamin D_3). Because alfacalcidol does not require renal 1-α-hydroxylation, it is helpful in patients with CKD, in whom renal activation is impaired. Paricalcitol is similar to calcitriol but is more selective for vitamin D receptors on the parathyroid gland than those in the intestine, thereby reducing the risk of hypercalcaemia and hyperphosphataemia.

According to KDIGO, calcium-based phosphate binders and active vitamin D analogues-although intended to improve CKD-MBD – may actually increase the risk of vascular calcification and cardiovascular events, the leading cause of death in CKD, if they are used routinely without clear indications. However, in local practice, many CKD patients – regardless of biochemical status – receive daily alfacalcidol (or calcitriol) and calcium carbonate as 'renal vitamins,' reflecting historical habits rather than evidence-based care.

[1]*Consultant Nephrologist, Teaching Hospital, Kurunegala, Sri Lanka.*

Correspondence: e-mail: premil3nadeekanth@gmail.com

https://orcid.org/0009-0009-1375-1886

Received 23 October 2025, accepted 28 November 2025

Local barriers and practical adaptation

In Sri Lanka, access to PTH testing is limited because samples require immediate plasma separation and freezing. Furthermore, it is not widely available in the public sector, and cost also poses a barrier. As an alternative, alkaline phosphatase (ALP) is a practical surrogate marker of bone turnover when hepatic disease is ruled out by a normal γ-glutamyl transferase (γ-GT) level. KDIGO (2017 CKD-MBD update and 2022 quick reference) recognises that either serum PTH or bone-specific alkaline phosphatase can be used to evaluate bone turnover in CKD. However, specific treatment thresholds or cut-offs for these markers are not provided. This article introduces a practical algorithm using phosphate, calcium, and ALP to guide the initiation or discontinuation of therapy with calcium carbonate and alfacalcidol, a vitamin D analogue, when PTH testing is unavailable. Although ALP may not replace PTH, in our resource-limited setting it provides a safer alternative to blind prescribing, helping to reduce potential harm from inappropriate use of calcium carbonate and vitamin D analogues.

When to use calcium carbonate

- Begin only if serum phosphate exceeds 4.5 mg/dL (2.5-4.5 mg/dL) on two or more occasions, repeated in a 1-3 month period, despite dietary restriction in non dialysis CKD patients.
- Stop or switch to sevelamer or lanthanum carbonate (non-calcium-based phosphate binders) if albumin-corrected calcium is >10.5 mg/dL (8.5-10.5 mg/dL) or phosphate remains uncontrolled.
- Avoid blanket use in all CKD patients; excessive calcium load promotes positive calcium balance, vascular calcification, and accelerated atherosclerosis.

When to use vitamin D analogues

- Start treatment only when there is evidence of high bone turnover, indicated by persistently elevated PTH; if PTH is not available, use a rising alkaline phosphatase (with normal γ-GT) as a surrogate indicator.
- Consider vitamin D analogues only if alkaline phosphatase remains persistently elevated despite correcting serum phosphate with calcium carbonate and after repleting nutritional vitamin D, indicating ongoing secondary hyperparathyroidism.
- Because vitamin D deficiency is common in CKD and testing can be expensive, empirical supplementation with cholecalciferol (vitamin D_3) can be recommended before using vitamin D analogues.
- Discontinue therapy if serum calcium exceeds 10.2 mg/dL or if alkaline phosphatase falls below 40 IU/L, as these findings indicate over-suppression of bone turnover and possible adynamic bone disease.

Stepwise practical approach

Step 1: Correct modifiable factors

- Limit dietary phosphate, mainly by prioritising fresh foods over processed products.
- Treat vitamin D deficiency empirically with cholecalciferol supplementation.

Step 2: Assess serum phosphate

- If serum phosphate remains elevated, introduce calcium carbonate 500 mg with meals up to three times daily for maximum phosphate-binding effect. The maximum dose of calcium carbonate is 3g per day; 1g, taken three times daily.
- Total elemental calcium intake from diet and binders should not exceed about 2,000 mg/day.[1] Reassess serum calcium and phosphate after three months.

Step 3: Evaluate bone turnover

- If alkaline phosphatase continues to rise (suggesting high bone turnover) commence alfacalcidol or calcitriol 0.25 µg daily or on alternate days.

Step 4: Monitor for treatment safety

- Continue monitoring serum calcium, phosphate, and PTH (or ALP) every three months.
- Discontinue vitamin D analogue therapy if serum calcium exceeds 10.2 mg/dL or if alkaline phosphatase falls below 40 IU/L, as this indicates possible adynamic bone disease.

Avoidable malpractices

Inappropriate practice	Potential consequence
Empirical calcium carbonate and active vitamin D analogues in all CKD patients	Hypercalcaemia causes vascular calcification with increased risk of cardiovascular disease.
Failure to check γ-GT before interpreting ALP	Misclassification of bone turnover
Lack of follow-up biochemistry after starting therapy	Delayed detection of under- or overtreatment
Continuing vitamin D analogues with suppressed ALP	Increased risk of adynamic bone disease

Discussion

The shift from routine supplementation to investigation-driven prescribing is both clinically sound and cost-effective. Overreliance on calcium-based binders and activated vitamin D analogues contributes to morbidity without improving survival, whereas using ALP as a surrogate allows safe, population-level implementation even when PTH is unavailable.

The economic implications are substantial: calcium carbonate and vitamin D analogues constitute a large share of the national renal drug budget. Rationalising their use can save money for dialysis consumables and non-calcium-based phosphate binders.

Educational outreach to non-nephrology prescribers, especially primary care doctors, is essential. Teaching that normal calcium levels do not justify calcium supplements and that rising ALP, rather than CKD stage, determines the need for vitamin D analogues will standardise practices across centres.

Future directions

- Broader introduction of regional PTH testing and quality-assured ALP standardisation.
- Continued surveillance of calcium carbonate and vitamin D analogues utilisation in state hospitals.
- Integration of CKD-MBD modules into national CME programmes.

Conclusion

Indiscriminate prescription of vitamin D analogues and calcium carbonate remains a major therapeutic pitfall in CKD care across Sri Lanka. Simple laboratory investigation-guided algorithms using serum calcium, phosphate, and ALP can replace empirical treatment, thus preventing vascular calcification, cardiovascular disease, and low-turnover bone disease while making the best use of limited healthcare resources. Clinicians should emphasise the importance of investigation-guided treatment initiation rather than applying blanket treatments.

Learning points

- Do not prescribe vitamin D analogues (alfacalcidol, calcitriol) or calcium carbonate routinely to all CKD patients.
- Use ALP (with normal γ-GT) as a surrogate for PTH when facilities for PTH measurement are unavailable.
- Start calcium carbonate only for persistent hyperphosphataemia > 4.5 mg/dL.
- Start vitamin D analogues only if ALP is rising or PTH is high; stop if serum calcium is > 10.2 mg/dL or ALP < 40 IU/L.
- Repeat serum calcium, phosphate, PTH (or ALP) every 3 months, especially if there has been a change in therapy.
- Rational prescribing reduces vascular calcification, cardiovascular morbidity, adynamic bone disease, and drug wastage.

Conflict of interests

None.

Further reading

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