



Chronic kidney disease and mineral bone disorder (CKD-MBD)

Bahareh Marghoob .MD
Assistant professor of Nephrology
HKC-IUMS

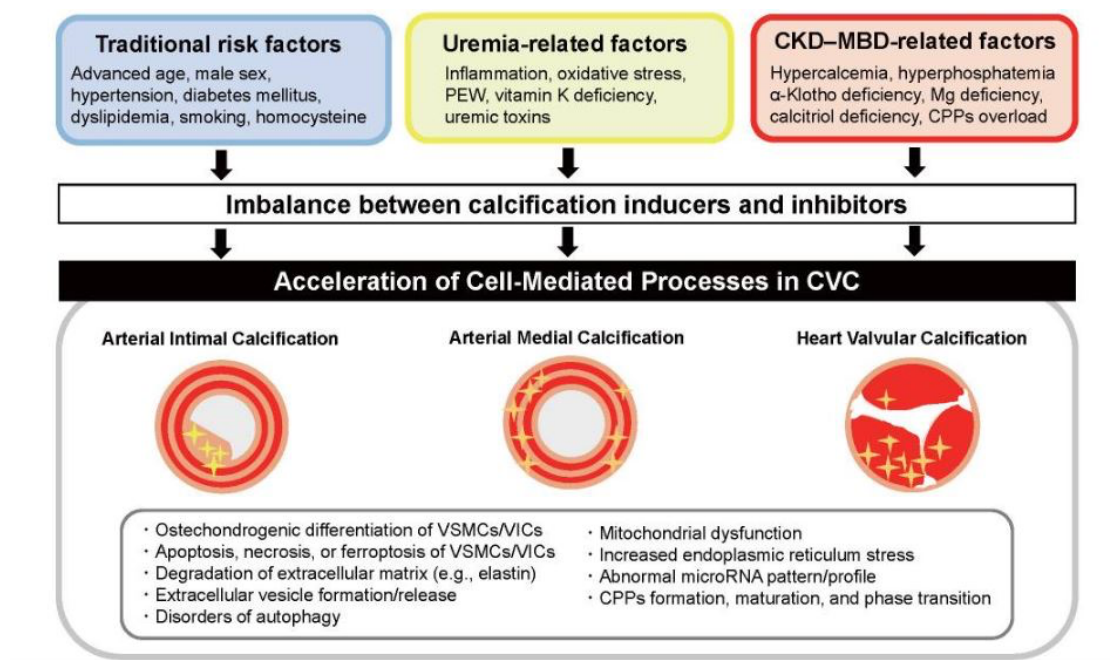
- A 47 y/o woman CKD with unknown cause admitted because of uremic symptoms. HD was started
- Cr: 8.4 mg/dl
- Urea : 184
- K : 6.1 mg/dl
- Ca : 7.1 mg/dl
- P : 8.5 mg/dl
- PTH : 874

- 67 y/o man known case of DM admitted with hyperkalemia and underwent maintenance HD
- Cr :6.3 mg /dl
- BUN : 104
- K : 6.9 mg/dl
- Ca : 8.2 mg /dl
- P: 6.7 mg/dl
- PTH : 118

- 27 y/o man known case of Alport syndrome admitted because of edema and hypertension
- Cr :8.3 mg /dl
- BUN : 124
- K : 5.9 mg/dl
- Ca : 9.2 mg /dl
- P: 5.7 mg/dl
- PTH : 980

- 48 y/o woman known case of ADPKD referred for routine exam:
- Cr :3.3 mg /dl
- BUN : 54
- K : 4.9 mg/dl
- Ca : 9.1 mg /dl
- P: 4.5 mg/dl
- PTH : 82
- Vit D: 16

- The term CKD–MBD was first introduced to the field of nephrology in exchange for the term renal osteodystrophy
- The use of the term renal osteodystrophy is limited to indicate bone disorders in CKD–MBD
- CKD–MBD is a **systemic disorder** that causes cardiovascular calcification (CVC), bone disorder, and other complications, all of which together **increase mortality**
- CKD–MBD comprises the following three components: laboratory abnormalities, bone disorders, and CVC.

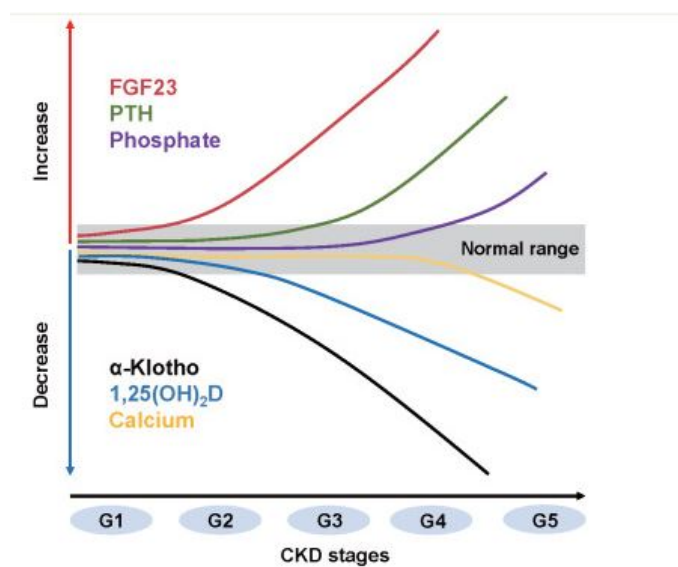


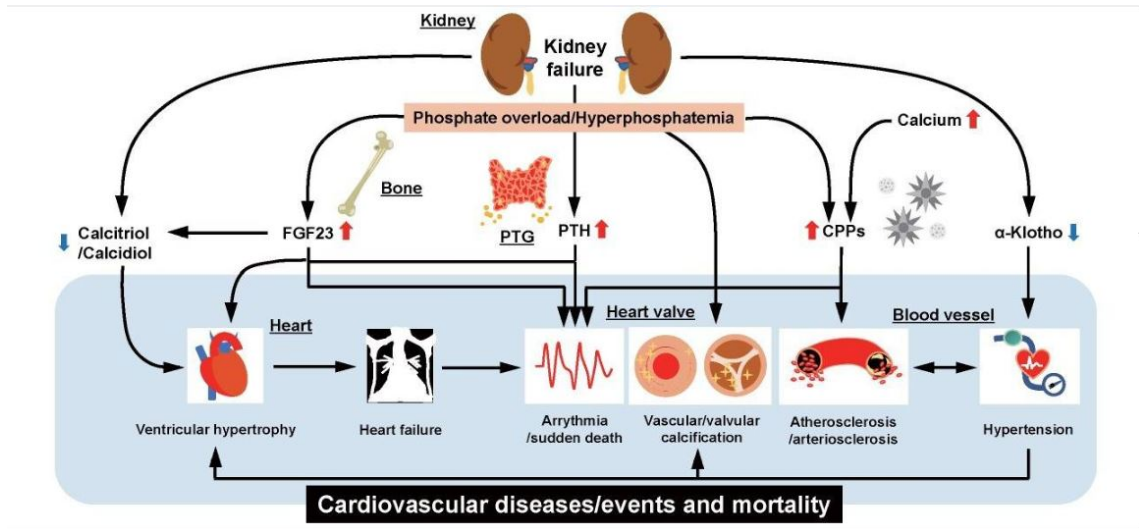
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- The initial step in CKD-MBD involves decreased phosphate excretion in the urine, followed by increased circulating concentrations of fibroblast growth factor 23 (FGF23) and parathyroid hormone (PTH), which increase urinary phosphate excretion
- FGF23 and PTH cause **left ventricular hypertrophy**, **arrhythmia**, and **cardiovascular calcification**
- CKD-MBD has become one of the critical therapeutic targets for the prevention of cardiovascular events

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GFR category	GFR (mL/min/1.73 m ²)	Terms
G1	≥ 90	Normal or high
G2	60–89	Mildly decreased*
G3a	45–59	Mildly to moderately decreased
G3b	30–44	Moderately to severely decreased
G4	15–29	Severely decreased
G5	< 15	Kidney failure





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2-4x

Increased Fracture Risk

CKD patients face higher fracture rates than general population

33%

Dialysis Patients

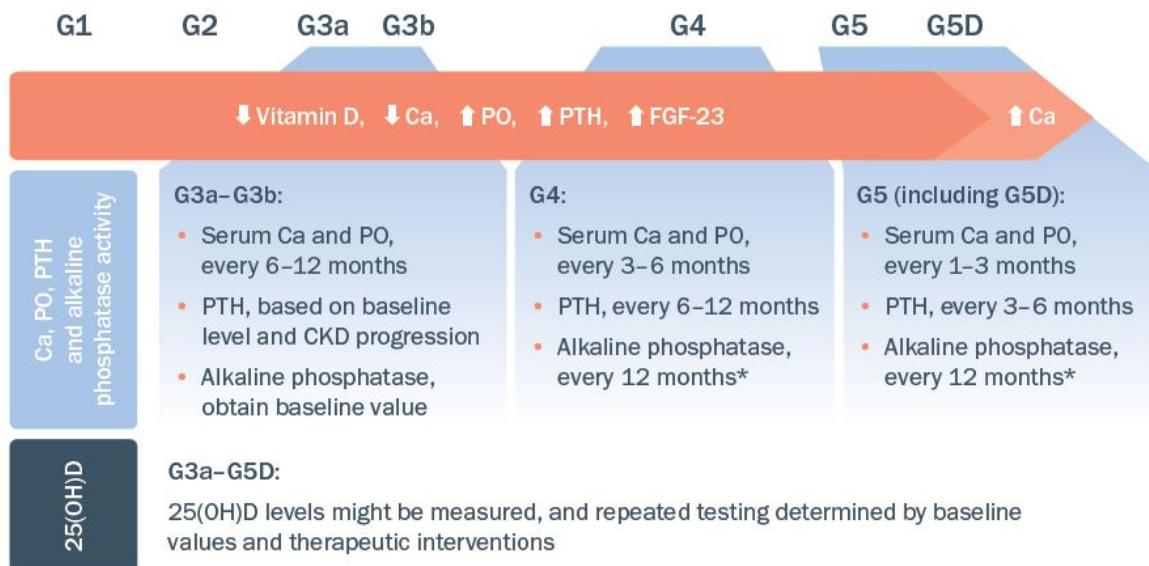
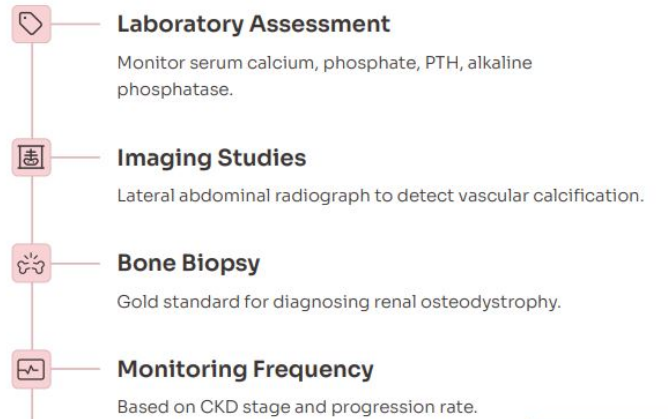
Percentage with hip fracture who die within one year

11-14%

Annual Mortality

For CKD patients suffering from any fragility fracture

KDIGO suggests BMD testing if results will impact treatment decisions. Consider fracture risk assessment tools for CKD stages 3a-5D.



Multidisciplinary Approach

Involve nephrologists, dietitians, pharmacists, and nurses in care planning.

Patient Education

Ensure patients understand dietary restrictions and medication importance.

Regular Monitoring

Implement systematic lab monitoring schedules based on CKD stage.

Local Protocol Development

Adapt guidelines to local resources while maintaining core principles.



Dietary Restriction

Limit dietary phosphate intake to 800-1000 mg/day.



Phosphate Binders

Calcium-based or non-calcium-based binders with meals.

3

Dialysis Management

Optimize phosphate removal through dialysis prescription.



Calcium Balance

Maintain normal calcium levels, avoid hypercalcemia.

Nutritional Vitamin D

Correct 25(OH)D deficiency with cholecalciferol or ergocalciferol.

- Target levels > 30 ng/mL
- Monitor serum calcium and phosphate

Active Vitamin D

Consider calcitriol or vitamin D analogs for elevated PTH.

- Individualize dosing based on PTH levels
- Avoid hypercalcemia and hyperphosphatemia

Monitoring Requirements

Regular assessment of calcium, phosphate, and PTH.

- Adjust therapy based on trends
- Consider bone-specific markers

Phosphate Binders

- Calcium-based: carbonate, acetate
- Non-calcium: sevelamer, lanthanum
- Iron-based: ferric citrate

Limit calcium-based binders in calcification cases.

Vitamin D Therapy

- Nutritional: ergocalciferol, cholecalciferol
- Active: calcitriol, paricalcitol, alfacalcidol

Individualize based on PTH, calcium, and phosphate levels.

Calcimimetics & Others

- Cinacalcet, etelcalcetide
- Bisphosphonates (with caution)
- Denosumab (with monitoring)

Reserve for specific indications with close monitoring.

- Treatment decisions should be based on serial assessments and trends of phosphate, calcium, and PTH levels considered together rather than isolated values (Ungraded recommendation).
- Lowering **elevated serum phosphate** toward the normal range is suggested to reduce complications (2C recommendation)
- **Avoidance of hypercalcemia** is emphasized due to its association with increased mortality and cardiovascular events; mild asymptomatic hypocalcemia can be tolerated, especially with calcimimetic therapy (2C recommendation)
- The guideline shifts away from strict biochemical targets to a more **individualized**, pragmatic approach tailored to patient preferences, baseline values, and treatment tolerance

- Mild hypocalcemia treat with correction of Vitamin D deficiency
- Vitamin D supplement not indicated in:
 - Patients with elevated serum phosphate level
 - Patients with hypercalcemia
- In nondialysis CKD patients goal of PTH is > 2.3 to 3 times the upper limit (< 150 pg/ml)
- In nondialysis CKD patients we do not use Cinacalcet

- KDOQI guideline:
 - Serum level of phosphate should be maintained between 3.5 to 5.5 mg/dl
 - Serum level of calcium should be maintained < 9.5 mg/dl
 - PTH level should be maintained less than two to nine times the normal upper limit

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Calcium supplement

Phosphate binder like sevelamer carbonate

Avoid starting calcitriol

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Calcium level is acceptable

Use calcium or non calcium based phosphate binder

Avoid calcitriol or calcimimetics

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Best choice for treatment of PTH is Cinacalcet in combination with non calcium based phosphate binder

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Vit D analog is the best treatment
Avoid calcitriol

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Thanks for your attention

