

MBD – CKD PATHWAY

Key Points in Managing Mineral Bone Disease in CKD –

(See references on last page)

Phosphorus retention causes:

- Parathyroid gland hyperplasia
- Increases PTH mRNA translation
- Decreases renal calcitriol production
- Increases PTH gland resistance to vitamin D

Management:

- Control phosphorus:
 - Diet, binders, dialysis
- Control calcium:
 - Intake, dialysis, cinacalcet
- Suppress PTH (but not too much):
 - Phosphorus control
 - Optimal use of vitamin D
 - Cinacalcet

Secondary Hyperparathyroidism:

It is a multi-system disease syndrome:

- Bone disease
- Tissue calcification
- Cardiac calcification
- Neurological dysfunction
- Myopathy
- Immune impairment

Consequences of sustained high PTH:

Skeleton:

- High bone matrix turnover:
 - Creates “woven” bone, not “lamellar” bone. (Woven bone is structurally inferior).
 - Risk of fracture: 48-fold higher in ESRD patients under age 45 compared to age-matched controls.

Extra- Skeletal consequences:

- Vascular calcification:
 - Calcification of arteries of all sizes.
 - Calcification of myocardium.
 - Increased risk of cardiac death.
 - Increased risk of central and peripheral vascular complications.

Promoters of vascular calcification:

- Hypercalcemia
- Hyperphosphatemia
- High calcium and phosphorus product
- Inflammation
- Warfarin
- Vitamin D3
- Corticosteroids
- Estrogen

Inhibitors of vascular calcification:

- Matrix Gla protein (*Gla is a protein found in numerous body tissues that requires vitamin K for its optimum function and it is present in bone*).
- Fetuin (*Fetuin is a blood protein that is made in the liver and secreted into the blood stream. It belongs to a large group of binding proteins mediating the transport and availability of a wide variety of cargo substances in the blood stream*).
- PTH
- Osteocalcin (*Osteocalcin is a noncollagenous protein found in bone and dentin. It is secreted by osteoblasts and thought to play a role in mineralization and calcium ion homeostasis*).
- Vitamin K

Objectives of treating Secondary hyperparathyroidism:

- Suppress PTH synthesis with vitamin D.
- Support bone mineralization and remodeling with vitamin D:
 - Avoid sustained PT gland oversuppression because this may result in adynamic bone disease.
- Avert soft tissue calcification, which is driven by high phosphorus and high calcium and phosphorus product.

Management of Secondary Hyperparathyroidism:

- Suppress and control PTH
- Control phosphate
- Control calcium
- Sustain bone turnover

Interdisciplinary Team (IDT) – POC (Individual patient)

- Review monthly results of phosphorus, calcium, PTH, calcium X phosphorus and alkaline phosphatase.
- Analyze trends within these lab results.
- Analyze correlations among these labs results.
- Analyze correlations of these lab results with clinical outcomes.
- Review monthly medication (dose, frequency etc).
- Review patient compliance (medication and dialysis)
- Establish short and long term goals (measurable) for individual patient improvement.
- If lab result is out of target range, identify causes/barriers and brainstorm solutions to correct causes and overcome barriers (by individual patient)

Interdisciplinary Team (IDT) – QAPI (Aggregated data)

- If MBD indicators are not meeting target goal:
 - Develop an improvement plan (using all components of the IP: root causes, interventions, goals, time frames, evaluation), using the model for improvement (PDSA).
 - Review IP during monthly QAPI meetings.



- Adjust IP as needed.
- Implement all successful, tested interventions.
- Close IP when all MBD indicators achieve target goal.
- If MBD indicators are meeting target goal:
 - Discuss measurable improvements during monthly QAPI meetings.
 - Review positive trends and current successful interventions.
 - Use benchmarking to motivate the team.

(Kidney International 58: 396-399, 2000).

(Seminars in Dialysis 17: 209-216, 2004).

(Kidney Int. 2009 May;75(10):1114; author reply 1114).

(Clin J Am Soc Nephrol. 2009 Nov;4(11):1805-10).