KDIGO CKD-MBD GUIDELINE UPDATE OVERVIEW

David Wheeler, KDIGO Co-Chair
DISCLOSURES:

• Honoraria and/or travel support from: Akebia, Amgen, AstraZeneca, Boehringer Ingelheim, Vifor Fresenius Medical Care, and Janssen.
Improve the care and outcomes of kidney disease patients worldwide through the development and implementation of clinical practice guidelines.
Diagnosis and Management of CKD  January 2013
Lipid Management  November 2013
CKD-MBD Update  July 2017
Living Kidney Donors  August 2017
Hepatitis C  April 2008
Mineral Bone Disorder  July 2009
Transplant Recipient  Oct 2009

Acute Kidney Injury  March 2012
Glomerulonephritis  June 2012
Anemia  August 2012
Blood Pressure in CKD  November 2012
Overview of proposed changes

• **Selective update in red**
• **Minor changes in dark grey**
• **No changes in black and white**
CKD-MBD GUIDELINE UPDATE 2017

Guideline Chairs
Markus Ketteler (Germany)
Mary B Leonard (USA)

Work Group
• Geoffrey A. Block (USA)
• Pieter Evenepoel (Belgium)
• Masafumi Fukagawa (Japan)
• Charles A. Herzog (USA)
• Linda McCann (USA)
• Sharon M. Moe (USA)
• Rukshana Shroff (UK)
• Marcello A. Tonelli (Canada)
• Nigel D. Toussaint (Australia)
• Marc G. Vervloet (Netherlands)
KDIGO CKD-MBD GUIDELINE UPDATE

August 2009

July 2017
Summary and comparison of 2017 updated and 2009 KDIGO CKD-MBD recommendations

2017 revised KDIGO CKD-MBD recommendations

4.1.6. In pediatric patients with CKD G3a-G5, we recommend restricting the dose of calcium-based phosphate binders and/or the dose of calcitriol or vitamin D analogs in the presence of persistent or recurrent hypercalcemia (CII). New evidence from ICTs supports a more general recommendation to restrict calcium-based phosphate binders in phosphate-naïve patients across all severities of CKD.

4.1.7. In patients with CKD G3a-G5 and hyperphosphatemia, we recommend restricting the dose of calcium-based phosphate binders in the presence of abnormal calcium (CII) and alendronate (bone disease) (CII) and if serum PTH levels are persistently low (CII).

Brief rationale for updating

4.1.8. In patients with CKD G3d-G5, we suggest limiting dietary phosphate intake in the treatment of hyperphosphatemia alone or in combination with other treatments (CII). The primary motivation for this revision was the growing experience with cephalosporin medications in patients with CKD G3d-G5, and a high risk of failure. The inability to perform a bone biopsy may not justify withholding antiresorptive therapy from patients at high risk of fracture.

This new recommendation is provided in order to emphasize the necessity and interaction of KDIGO MBD Therapy parameters.

4.1.9. In patients with CKD G3a-G5, we suggest minimizing serum calcium in the age-appropriate normal range (CII).

4.2.2. In adult patients with CKD G4-G5 on dialysis, we suggest that calcitriol and vitamin D analogs are used for patients with CKD G4-G5 with severe and progressive hyperparathyroidism (CII).

4.2.3. In adult patients with CKD G4-G5 on dialysis, we suggest that calcium and vitamin D analogs are used for patients with CKD G4-G5 with severe and progressive hyperparathyroidism (CII).

New data on phosphate sources were derived from ICTs and required an update to the previous recommendation.

The Work Group felt that modest increases in PTH may represent an appropriate adaptive response to declining kidney function and has revised this statement to include "persistently" above the upper normal limit as well as "progressively rising" PTH levels, rather than "showing the upper normal limit." This treatment should not be based on a single elevated value.

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GUIDELINE TOPICS

Topic 1: Bone Quality
Topic 2: Serum phosphate
Topic 3: Serum calcium
Topic 4: Dialysate calcium
Topic 5: Phosphate binders
Topic 6: Dietary phosphate intake
Topic 7: Vitamin D and PTH
Topic 8: Vascular calcification
Topic 9: Parathyroid hormone range
OLD 3.2.2: In patients with CKD G3a–G5D with evidence of CKD–MBD, we suggest that BMD testing not be performed routinely, because BMD does not predict fracture risk as it does in the general population, and BMD does not predict the type of renal osteodystrophy (2B).

NEW 3.2.1: In patients with CKD G3a–G5D with evidence of CKD-MBD and/or risk factors for osteoporosis, we suggest BMD testing to assess fracture risk if results will impact treatment decisions (2B).
Bone mineral density (hip)

Lower BMD associated with fracture
Higher BMD associated with fracture

Bucur RC et al, Osteoporosis 2015;26: 449-458
**TOPIC 1: BONE QUALITY**

**OLD 3.2.1:** In patients with CKD G3a–G5D, it is reasonable to perform a bone biopsy in various settings including, but not limited to: unexplained fractures, persistent bone pain, unexplained hypercalcemia, unexplained hypophosphatemia, possible aluminum toxicity, and prior to therapy with bisphosphonates in patients with CKD-MBD (*Not Graded*).

**NEW 3.2.2:** In patients with CKD G3a–G5D, it is reasonable to perform a bone biopsy if knowledge of the type of renal osteodystrophy will impact treatment decisions (*Not Graded*).
**TOPIC 2: SERUM PHOSPHATE**

**OLD 4.1.1:** In patients with CKD G3a–G5, we suggest maintaining serum phosphate in the normal range (2C). In patients with CKD G5D, we suggest lowering elevated phosphate levels toward the normal range (2C).

**NEW 4.1.1:** In patients with CKD G3a–G5D, treatments of CKD-MBD should be based on serial assessments of phosphate, calcium, and PTH levels, considered together (**Not Graded**).

**NEW 4.1.2:** In patients with CKD G3a–G5D, we suggest lowering elevated phosphate levels toward the normal range (2C).
Population: 128 patients with eGFR 20-45 ml/min/1.73 m²
Intervention: Calcium acetate, lanthanum carbonate, sevelamer carbonate
Comparator: Placebo
Primary endpoint: Change in mean phosphorus from baseline to the average of 3, 6 and 9 months
PHOSPHATE NORMALIZATION TRIAL (PNT): RESULTS

Block GA et al, JASN 2012;23:1407-15
OLD 4.1.2: In patients with CKD G3a–G5D, we suggest maintaining serum calcium in the normal range \((2D)\).

NEW 4.1.3: In adult patients with CKD G3a–G5D, we suggest avoiding hypercalcemia \((2C)\).

In children with CKD G3a–G5D, we suggest maintaining serum calcium in the age-appropriate normal range \((2C)\).
**EVOLVE**: BIOCHEMICAL PARAMETERS (ITT)

- **Median iPTH**: Blue line represents Placebo (n = 1935), magenta line represents Cinacalcet (n = 1948).

- **Median Serum Calcium**: Blue line represents Placebo, magenta line represents Cinacalcet.

- **Median Serum Phosphate**: Blue line represents Placebo, magenta line represents Cinacalcet.

- **Median Ca x P Product**: Blue line represents Placebo, magenta line represents Cinacalcet.

*EVOLVE did not meet its primary end point.*
**TOPIC 4: DIALYSATE CALCIUM**

**OLD: 4.1.3** In patients with CKD G5D, we suggest using a dialysate calcium concentration between 1.25 and 1.50 mmol/l (2.5 and 3.0 mEq/l) (2D).

**NEW: 4.1.4:** In patients with CKD G5D, we suggest using a dialysate calcium concentration between 1.25 and 1.50 mmol/l (2.5 and 3.0 mEq/l) (2C).
# Grading System

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Recommendation grading</th>
<th>Quality of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>“We recommend”</td>
<td></td>
</tr>
<tr>
<td>Level 2</td>
<td>“We suggest”</td>
<td></td>
</tr>
<tr>
<td>Not graded</td>
<td>Based on common sense</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>Very Low</td>
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</tr>
</tbody>
</table>
TRIAL OF 2 DIALYSIS CALCIUM CONCENTRATIONS

Population: 431 HD patients with PTH < 300 pg/ml
Intervention: 1.25 mmol/l calcium bath vs. 1.75 mmol/l calcium bath
Primary Outcome: Coronary calcification (CAC) scores
Secondary Outcome: Bone histomorphometry
Follow-up: 24 months

"At 24 months, bone formation rate, trabecular thickness, and bone volume were higher in the 1.25 Calcium group than in the 1.75 Calcium group."

**TOPIC 5: PHOSPHATE BINDERS**

**OLD 4.1.4** In patients with CKD G3a–G5 (2D) and G5D (2B), we suggest using phosphate-binding agents in the treatment of hyperphosphatemia. It is reasonable that the choice of phosphate binder takes into account CKD stage, presence of other components of CKD–MBD, concomitant therapies, and side-effect profile (not graded).

**OLD 4.1.5:** In patients with CKD G3a–G5D and hyperphosphatemia, we recommend restricting the dose of calcium-based phosphate binders … in the presence of persistent or recurrent hypercalcemia (1B). In patients with CKD G3a–G5D and hyperphosphatemia, we suggest restricting the dose of calcium-based phosphate binders in the presence of arterial calcification (2C) and/or adynamic bone disease (2C) and/or if serum PTH levels are persistently low (2C).
NEW 4.1.5: In patients with CKD G3a-G5D, decisions about phosphate-lowering treatment should be based on progressively or persistently elevated serum phosphorus. (Not Graded)

NEW 4.1.6: In adult patients with CKD G3a–G5D receiving phosphate-lowering treatment, we suggest restricting the dose of calcium-based phosphate binders. (2B)

In children with CKD G3a–G5D, it is reasonable to base the choice of phosphate-lowering treatment on serum calcium levels. (Not Graded)
**Binder Choice in HD: Independent Trial**

**Population:** 466 incident haemodialysis patients  
**Intervention:** Sevelamer  
**Comparator:** Calcium binder  
**Outcome:** CV death due to cardiac arrhythmia  
**Timeline:** 24 months (36 months follow-up)

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**Arrhythmias**

**CV mortality**

Di Iorio et al., AJKD 2013; 62:771-78
**Binder choice in non-dialysed patients**

**Population:** 212 G3a-G4 CKD patients  
**Intervention:** Sevelamer  
**Comparator:** Calcium carbonate  
**Outcome:** All cause mortality  
**Timeline:** 24 months (36 months follow-up)

Di Iorio et al., CJASN 2012; 7:487-93
META ANALYSIS OF BINDER TRIALS IN CKD

Data from 11 RCTs.
Patients taking Sevelamer had 22% lower mortality RR 0.78 (95% CI 0.61 – 0.98)

Favors non-calcium Favors calcium

META ANALYSIS OF BINDER TRIALS IN CKD

Data from 25 studies
Patients taking Sevelamer had 46% lower mortality RR 0.54 (95% CI 0.32 – 0.93)

Patel L et al CJASN 2016;11:232-44
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Patel L et al CJASN 2016;11:232-44
**META-ANALYSIS OF BINDER TRIALS IN CKD**

<table>
<thead>
<tr>
<th>Sevelamer</th>
<th>Lanthanum</th>
<th>Calcium</th>
<th>Iron</th>
<th>Colestilan</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.50 (0.09, 2.65)</td>
<td>0.78 (0.16, 3.72)</td>
<td>2.67 (0.63, 11.4)</td>
<td>0.68 (0.07, 6.40)</td>
<td>0.66 (0.10, 4.29)</td>
<td></td>
</tr>
<tr>
<td><strong>0.39 (0.21, 0.74)</strong></td>
<td>2.08 (0.26, 16.5)</td>
<td>1.82 (0.23, 14.7)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1.04 (0.27, 3.97)</td>
<td>1.42 (0.12, 17.4)</td>
<td>1.20 (0.21, 6.77)</td>
<td>0.45 (0.08, 2.66)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.71 (0.09, 5.46)</td>
<td>0.93 (0.11, 8.05)</td>
<td></td>
<td>0.66 (0.10, 4.29)</td>
<td></td>
<td></td>
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<tr>
<td>0.47 (0.08, 2.59)</td>
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<td></td>
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</tr>
</tbody>
</table>

Network estimated odds ratios of phosphate binders on all-cause mortality

OLD 4.1.7: In patients with CKD G3a–G5D, we suggest limiting dietary phosphate intake in the treatment of hyperphosphatemia alone or in combination with other treatments (2D).

NEW 4.1.8: In patients with CKD G3a–G5D, we suggest limiting dietary phosphate intake in the treatment of hyperphosphatemia alone or in combination with other treatments (2D).

It is reasonable to consider phosphate source (e.g., animal, vegetable, additives) in making dietary recommendations. (Not Graded)
**TOPIC 6: DIETARY PHOSPHATE INTAKE**

9 patients, eGFR 30 ml/min/1.73 m²
7-day crossover trial

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Meat (casein) diet</th>
<th>Vegetarian (grain) diet</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td><strong>Daily (PO_4) (mg/day)</strong></td>
<td>810 ± 27</td>
<td>795 ± 51</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Plasma (PO_4) (mg/day)</strong></td>
<td>3.5 ± 0.6</td>
<td>3.7 ± 0.6</td>
<td>3.5 ± 0.6</td>
</tr>
<tr>
<td><strong>Plasma iPTH (pg/ml)</strong></td>
<td>58 ± 31</td>
<td>46 ± 29</td>
<td>58 ± 39</td>
</tr>
<tr>
<td><strong>Plasma FGF23 (pg/ml)</strong></td>
<td>72 ± 39</td>
<td>101 ± 83</td>
<td>84 ± 65</td>
</tr>
<tr>
<td><strong>Plasma Ca (mg/dl)</strong></td>
<td>9.2 ± 0.4</td>
<td>9.4 ± 0.7</td>
<td>9.3 ± 0.4</td>
</tr>
<tr>
<td><strong>Urine CA exc. (mg/day)</strong></td>
<td>66 ± 69</td>
<td>77 ± 48</td>
<td>60 ± 59</td>
</tr>
<tr>
<td><strong>Urine (PO_4) exc. (mg/day)</strong></td>
<td>836 ± 187</td>
<td>583 ± 216</td>
<td>778 ± 190</td>
</tr>
</tbody>
</table>

**Phosphate/protein ratio (mg/g) in processed vs unprocessed meat products**

OLD 4.2.2: In patients with CKD G3a–G5 not on dialysis, in whom serum PTH is progressively rising and remains persistently above the upper limit of normal for the assay despite correction of modifiable factors, we suggest treatment with calcitriol or vitamin D analogs (2C).

NEW 4.2.2: In adult patients with CKD G3a–G5 not on dialysis, we suggest that calcitriol and vitamin D analogs not be routinely used (2C). It is reasonable to reserve the use of calcitriol and vitamin D analogs for patients with CKD G4–G5 with severe and progressive hyperparathyroidism (Not Graded).
At 48 weeks, the change in left ventricular mass index did not differ between treatment groups.
Episodes of hypercalcaemia were more frequent in the paricalcitol group compared with the placebo group.

Thadhani et al, JAMA 2012
**OPERA STUDY: PARICALCITOL VS. PLACEBO**

- **Population:** 60 CKD patients (LVH)
- **Intervention:** Paricalcitol 1 µg/day
- **Comparator:** Placebo
- **Outcome:** LVMI by CMR
- **Timeline:** 52 weeks

### Table 3. Changes in cardiac MRI and echocardiographic parameters from baseline to 52 weeks

<table>
<thead>
<tr>
<th>Cardiac Parameters</th>
<th>Paricalcitol (n=30)</th>
<th>Placebo (n=30)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass index by body surface area, g/m²</td>
<td>81.2 (14.8)</td>
<td>79.5 (14.7)</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 52</td>
<td>79.0 (15.1)</td>
<td>75.2 (17.7)</td>
<td></td>
</tr>
<tr>
<td>Change from baseline to 52 wk</td>
<td>-2.59 (-6.13 to +0.32)</td>
<td>-4.85 (-9.89 to -1.10)</td>
<td>0.40</td>
</tr>
</tbody>
</table>

- At 52 weeks, the change in left ventricular mass index did not differ between treatment groups.
- PTH levels lower in patients receiving paricalcitol.
- Serum calcium increased in paricalcitol group but not in placebo group.

Wang A et al, JASN 2014; 25:125
OLD 4.2.4: In patients with CKD G5D and elevated or rising PTH, we suggest calcitriol, or vitamin D analogs, or calcimimetics, or a combination of calcimimetics and calcitriol or vitamin D analogs be used to lower PTH (2B).

NEW 4.2.4: In patients with CKD G5D requiring PTH-lowering therapy, we suggest calcimimetics, calcitriol, or vitamin D analogs, or a combination of calcimimetics with calcitriol or vitamin D analogs (2B).
No changes
KDIGO CKD-MBD 2017 Guideline: Key Messages

1. Prospective studies evaluating BMD testing in adults with CKD represent a substantial advance since the original guideline from 2009, making a reasonable case for BMD testing if the results will impact future treatment.

2. It is important to emphasize the interdependency of serum Ca, P, and PTH for clinical therapeutic decision-making.

3. Phosphate-lowering therapies may only be indicated in the case of “progressive or persistent hyperphosphatemia”.

4. New evidence suggests that excess exposure to exogenous calcium in adults may be harmful in all stages of CKD, regardless of other risk markers.
5. It is reasonable to limit dietary P intake, when considering all sources of dietary P (including “hidden” sources).

6. The PRIMO and OPERA trials failed to demonstrate improvements in clinically relevant outcomes but did demonstrate increased risk of hypercalcemia. Accordingly, routine use of calcitriol or its analogs in CKD G3a–G5 is no longer recommended.

7. No consensus was reached to recommend cinacalcet as first-line therapy for lowering PTH in all patients with SHPT and CKD G5D.