Complete Summary

GUIDELINE TITLE

K/DOQI clinical practice guidelines for bone metabolism and disease in children with chronic kidney disease.

BIBLIOGRAPHIC SOURCE(S)

KDOQI, National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in children with chronic kidney disease. Am J Kidney Dis 2005 Oct;46(4 Suppl 1):S1-121. [557 references]

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

- Chronic kidney disease
- Mineral and bone metabolism and disease
- Hyperparathyroidism

GUIDELINE CATEGORY

Diagnosis
Management
Prevention
Treatment

CLINICAL SPECIALTY

Family Practice
Internal Medicine
Nephrology
Pediatrics

INTENDED USERS

Advanced Practice Nurses
Allied Health Personnel
Health Care Providers
Nurses
Physician Assistants
Physicians

GUIDELINE OBJECTIVE(S)

To develop a set of clinical practice guidelines that would improve diagnoses and treatment of bone disease in chronic kidney disease in the pediatric population and serve as a clinical action plan for the health care practitioner

TARGET POPULATION

Children with chronic kidney disease

INTERVENTIONS AND PRACTICES CONSIDERED

Evaluation

1. Measurement and monitoring of serum levels of calcium, phosphorus, total carbon dioxide (CO_2), and parathyroid hormone (PTH)
2. Assessment of bone disease, including:
    - Iliac crest bone biopsy with double tetracycline labeling and bone histomorphometric analysis
    - Bone radiographs
3. Measurement of serum 25-hydroxyvitamin D and serum levels for carbon dioxide

Treatment/Management

1. Surgical management of osteodystrophy
    - Surgical correction of lower extremity angular deformity
    - Surgical stabilization of symptomatic proximal femoral slipped epiphyses
2. Maintenance/monitoring of serum phosphorus levels
    - Dietary phosphorus restriction
    - Use of calcium-based phosphate binders, noncalcium-, nonmetal containing phosphate binders, and aluminum-based phosphate binders
3. Maintenance of corrected serum calcium levels and calcium-phosphorus product (CaXP) within normal range

    - Hypercalcemia
        - Discontinued use of calcium-based phosphate binders
        - Discontinued use of vitamin D sterols
        - Dialysis using lower dialysate calcium
        - Control serum phosphorus levels to control CaXP
    - Hypocalcemia
        - Calcium salts and/or oral vitamin D sterols

4. Prevention and treatment of vitamin D insufficiency and vitamin D deficiency
    - Vitamin D_2 supplementation with monitoring of serum calcium, phosphorus, and PTH
    - Active vitamin D sterol therapy in stage 2-5 chronic kidney disease (CKD) with monitoring of serum calcium, phosphorus, and PTH
5. Maintenance of dialysate calcium concentration levels
6. Growth hormone (GH) therapy
7. Prevention of aluminum overload and toxicity
    - Avoidance of regular administration of aluminum and regulation of dialysate aluminum concentration
    - Measurement of serum aluminum at recommended intervals
    - Desferrioxamine (DFO) testing
    - Bone biopsy
8. Treatment of aluminum toxicity
    - Identifying and removing source of aluminum
    - DFO treatment, as indicated
9. Treatment of bone disease
    - Therapeutic management based on specific type of bone disease (hyperparathyroid, rickets/osteomalacia, and adynamic bone disease) as outlined in the guideline
10. Parathyroidectomy
11. Supplemental alkali salts for metabolic acidosis
12. Monitoring and treatment of bone disease following kidney transplant

MAJOR OUTCOMES CONSIDERED

- Accuracy of assessment of bone disease in pediatric patients with chronic kidney disease
- Incidence of and morbidity and mortality from serum levels of phosphorus, calcium, parathyroid hormone (PTH), and vitamin D
- Incidence of and morbidity and mortality from aluminum toxicity
- Treatment-related changes in serum levels of phosphorus, calcium, parathyroid hormone (PTH), vitamin D, and aluminum
- Incidence and severity of deformity, level of function and mobility, and level of self-esteem in patients with osteodystrophy
- Rate of linear growth
- Incidence and morbidity from:
    - Bone disease
    - Bone deformities
    - Deranged mineral ion homeostasis
    - Neurological abnormalities
    - Bone fractures
    - Hyperparathyroidism
- Incidence, severity, and duration of metabolic acidosis

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Strength of Evidence

Each rationale statement was graded according to the level of evidence on which it was based. The overall guideline was then graded according to the strength of evidence supporting the rationale statements.

Rationale Statements

1. Analysis of controlled trials, generalizable studies of high methodological quality
2. Analysis of lower quality studies
3. Vote count analysis of evidence tables
4. Review of reviews and selected original articles
5. Opinion

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Members of the committee for the preparation and publication of the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQI) Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease (CKD) in adults recognized that the subject in that population was filled with enough controversy that recommendations for children could not properly be placed within it. Therefore, a K/DOQI Work Group met in October 2002, to begin the process of sorting through the literature to produce a pediatric specific set of K/DOQI guidelines for bone metabolism and disease in CKD with colleagues and committee members.

The task differed initially from the resultant guidelines seen in the original guideline document; but with very limited resources, and no external review of extant literature by a third party, the Work Group decided late in 2003 to follow the structure of the adult-based bone metabolism and disease guidelines and adapt it where indicated for the aspects unique to pediatric osteodystrophy.

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

In formulating the guidelines, the rationale and evidentiary basis of each recommendation was made explicit.

- When all components of the rationale for a guideline were based on published evidence, the guidelines were labeled **"Evidence."**
- When no definite evidence existed or the evidence was considered inconclusive, and either the guideline or steps in its rationale were based on judgment they were labeled **"Opinion."**

As such, it is the available literature that determined the labeling of each guideline.

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Not stated

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Evidentiary Basis for Recommendations

- When all components of the rationale for a guideline were based on published evidence, the guidelines were labeled **"Evidence."**
- When no definite evidence existed or the evidence was considered inconclusive, and either the guideline or steps in its rationale were based on judgment they were labeled **"Opinion."**

Guideline 1. Evaluation of Calcium and Phosphorus Metabolism

1.1 Serum levels of calcium, phosphorus, alkaline phosphatase, total carbon dioxide (CO_2), and parathyroid hormone (PTH)—measured by a first-generation immunometric parathyroid hormone assay—should be measured in all patients with chronic kidney disease (CKD) Stages 2 through 5. The frequency of these measurements should be based on the stage of CKD (see Table below). **(OPINION)**

Frequency of Measurement of Parathyroid Hormone (PTH), Calcium, Phosphorus, Total Carbon Dioxide (CO_2), and Alkaline Phosphatase by Stage of CKD

Stage	Glomerular Filtration Rate (GFR)	Calcium, Phosphorus, Total CO_2	PTH & Alkaline Phosphatase ($mL/min/1.73m^2$)
2	60-89	At least yearly	At least yearly
3	30-59	At least every 6 months	At least every 6 months
4	15-29	At least every 3 months	At least every 3 months
5	<15 or dialysis	At least every month	At least every 3 months

1.1.a Patients with known tubulopathies in Stage 1 CKD should have serum phosphorus levels measured at least yearly.

1.2 These measurements should be made more frequently if the patient is receiving concomitant therapy for the abnormalities in the serum levels of calcium, phosphorus, or PTH (see Guidelines 4-10), is a transplant recipient (see Guideline 17), or is a patient being treated with recombinant growth hormone (rhGH) (see Guideline 11).

1.2.a The target range of serum PTH, in the various stages of CKD, are denoted in the following table. **(OPINION)**

Target Range of Serum Parathyroid Hormone (PTH) by Stage of Chronic Kidney Disease (CKD)

CKD Stage	GFR Range ($mL/min/1.73m^2$)	Target Serum PTH
2	60-89	35-70 pg/mL **(OPINION)**
3	30-59	35-70 pg/mL **(OPINION)**
4	15-29	70-110 pg/mL **(OPINION)**
5	<15 or dialysis	200-300 pg/mL (**EVIDENCE)**

Guideline 2. Assessment of Bone Disease Associated with CKD

2.1 The most accurate diagnostic test for determining the type of bone disease associated with CKD is iliac crest bone biopsy with double tetracycline labeling and bone histomorphometric analysis. **(EVIDENCE)**

2.2 It is not necessary to perform bone biopsy for most situations in clinical practice. However, a bone biopsy should be considered in patients with kidney failure (Stage 5) who have:

2.2.a Fractures with minimal or no trauma (pathological fractures) **(OPINION)**

2.2.b Suspected aluminum bone disease, based upon clinical symptoms or history of aluminum exposure **(OPINION)** (see Guideline 12)

2.2.c Persistent hypercalcemia with PTH levels between 400-600 pg/mL

2.3 Bone radiographs are indicated in patients with clinical manifestations suggestive of avascular necrosis, symptomatic proximal femoral slipped epiphyses (SCFE), rickets, or for the assessment of skeletal maturation. **(EVIDENCE)**

2.4 Dual-energy X-ray absorptiometry (DXA) should not be used to monitor bone mineral density in children with CKD. **(OPINION)**

Guideline 3. Surgical Management of Osteodystrophy

3.1 Lower extremity angular deformity should be surgically corrected if the deformity is progressive or severe as defined by interference with gait, or by the presence of a mechanical axis deviation of more than 10 degrees between the femur and tibia. Control of secondary hyperparathyroidism (HPT) is recommended prior to surgical correction. **(OPINION)**

3.2 SCFE should be surgically stabilized if Kidney Disease Outcomes Quality Initiative (K/DOQI) target values for PTH are not achieved within 3 months of the diagnosis of SCFE. **(OPINION)**

Guideline 4. Target Serum Phosphorus Levels

4.1 In CKD patients (Stages 1-4), the serum level of phosphorus should be maintained at or above the age-appropriate lower limits **(EVIDENCE)** and no higher than the age-appropriate upper limits. **(OPINION)**

4.2 For children with kidney failure (CKD Stage 5), including those treated with hemodialysis or peritoneal dialysis, the serum levels of phosphorus should be maintained between 3.5-5.5 mg/dL (1.13-1.78 mmol/L) during adolescence and between 4-6 mg/dL for children between the ages of 1-12 years. **(EVIDENCE)**

4.3 In children with renal tubular phosphate wasting, or other causes of hypophosphatemia, hypophosphatemia should be corrected via dietary

modification, enteral supplementation, or reduction in the use of phosphate binders. **(EVIDENCE)**

Representative Normal Values for Serum Phosphorus, Total Calcium, Blood Ionized Calcium, and Alkaline Phosphatase Concentrations

Age (years)	Serum Phosphorus (mg/dL)	Serum Total Calcium (mg/dL)	Blood Ionized Calcium (mM)	Alkaline Phosphatase (IU)
0-0.25	4.8-7.4	8.8-11.3	1.22-1.40	
1-5	4.5-6.5	9.4-10.8	1.22-1.32	100-350
6-12	3.6-5.8	9.4-10.3	1.15-1.32	60-450
13-20	2.3-4.5	8.8-10.2	1.12-1.30	40-180

Guideline 5. Management of Dietary Phosphorus Intake in Children With CKD

5.1 Dietary phosphorus should be decreased to the Dietary Reference Intake (DRI) for age (see Table below) when the serum PTH concentration is above the target range for the stage of CKD and serum phosphorus is within the target range for age (see Table above, Guideline 4). **(OPINION)**

5.2 Dietary phosphorus should be decreased to 80% of the DRI for age (see Table below) when the serum PTH concentration is above the target range for the stage of CKD and serum phosphorus is above the target range for age (see Table above, Guideline 4). **(OPINION)**

Dietary Reference Intakes (DRI) of Phosphorus in Children

Age (Years)	Phosphorus DRI (mg/day)
0-0.5	100
0.5-1.0	275
1-3	460
4-8	500
9-18	1,250

5.3 After initiation of phosphorus restriction, serum phosphorus concentrations should be monitored at least every 3 months in patients with CKD Stages 3-4, and monthly in patients with CKD Stage 5. Serum phosphorus values below the target range for age should be avoided.

Guideline 6. Use of Phosphate Binders In CKD

In Patients with CKD Stages 2-4:

6.1 If serum phosphorus levels cannot be controlled within the target range (see Guideline 4), despite dietary phosphorus restriction (see Guideline 5), phosphate binders should be prescribed. **(OPINION)**

6.2 Calcium-based phosphate binders are effective in lowering serum phosphorus levels **(EVIDENCE)** and should be used as the initial binder therapy. **(OPINION)**

In Patients with CKD Stage 5 (Dialysis):

6.3 Both calcium-based phosphate binders and the non-calcium, non-metal-containing phosphate binders, such as sevelamer HCl, are effective in lowering serum phosphorus levels. **(EVIDENCE)** As of this writing, calcium-based phosphate binders should be used as primary therapy in infants and young children. In older children and adolescents, either drug may be used. **(OPINION)**

6.4 In dialysis patients who remain hyperphosphatemic (above the upper target value) despite the use of either calcium-based phosphate binders or other noncalcium, non-metal-containing phosphate binders, the dialysis prescription should be modified to control hyperphosphatemia. **(OPINION)**

6.5 The total dose of elemental calcium provided by the calcium-based phosphate binders and dietary calcium should not exceed up to 2X DRI for calcium, based on age **(OPINION)**, and the total intake of elemental calcium (including dietary calcium) should not exceed 2,500 mg/day. **(OPINION)**

6.6 The dosage of calcium-based phosphate binders should be lowered in dialysis patients with corrected serum calcium of >10.2 mg/dL (2.54 mmol/L), or with serum PTH levels <150 pg/mL (150 ng/L) on two consecutive measurements. **(EVIDENCE)**

6.7 In adolescent patients with serum phosphorus levels >7.0 mg/dL (2.26 mmol/L), aluminum-based phosphate binders may be used as a short-term therapy (up to 4-6 weeks), and for one course only, to be replaced thereafter by other phosphate binders. **(EVIDENCE)**

6.8 In children receiving aluminum-based phosphate binders, concurrent use of citrate-based products should be avoided, due to the risk of increasing aluminum absorption and potential toxicity. **(EVIDENCE)**

Guideline 7. Serum Calcium and Calcium-Phosphorus Product

In CKD Patients Stages 2-4:

7.1 The serum levels of corrected total calcium should be maintained within the normal range for the laboratory used. **(EVIDENCE)**

In CKD Patients with Kidney Failure (Stage 5):

7.2 Serum levels of corrected total calcium should be maintained within the normal range for the laboratory used (8.8-9.7 mg/dL [2.20-2.37 mmol/L]), and preferably toward the lower end. **(OPINION)**

7.3 In the event that the corrected total serum calcium level exceeds 10.2 mg/dL (2.54 mmol/L), therapies that increase serum calcium should be adjusted as follows:

7.3.a In patients taking calcium-based phosphate binders, the therapy should be discontinued and the use of non-calcium, non-metal based phosphate binders should be considered. **(OPINION)** (See Guideline 6.)

7.3.b In patients taking active vitamin D sterols, the therapy should be discontinued until the serum levels of corrected total calcium return to the target range (8.8-9.5 mg/dL [2.20- 2.37 mmol/L]). **(OPINION)** (See Guideline 8b.)

7.3.c If hypercalcemia (serum levels of corrected total calcium >10.2 mg/dL [2.54 mmol/L]) persists despite discontinuation of therapy with vitamin D and/or modification of calcium-based phosphate binders, dialysis using lower dialysate calcium may be used for 3-4 weeks. **(OPINION)** (See Guideline 10.)

In CKD Patients Stages 3-5:

7.4 The total dose of elemental calcium provided by the calcium-based phosphate binders should not exceed up to 2X DRI for calcium based on age, **(OPINION)** and the total intake of elemental calcium (including dietary calcium) should not exceed 2,500 mg/day. **(OPINION)**

7.5 The serum calcium-phosphorus product (CaXP) should be maintained at <55 mg^2/dL^2 in adolescents >12 years, and <65 mg^2/dL^2 in younger children. **(OPINION)** This is best achieved by controlling serum levels of phosphorus within the target range. **(OPINION)** (See Guidelines 4-6.)

7.6 Patients whose serum levels of corrected total calcium are below the lower limit (<8.8 mg/dL [2.20 mmol/L]) should receive therapy to increase serum calcium levels:

7.6.a Therapy for hypocalcemia should include calcium salts such as calcium carbonate or calcium acetate orally, or calcium gluconate or calcium chloride parenterally **(EVIDENCE)**, and/or oral vitamin D sterols. **(EVIDENCE)** (See Guideline 9.)

Guideline 8. Prevention and Treatment of Vitamin D Insufficiency And Vitamin D Deficiency in CKD Patients

In CKD Stages 2-4:

8.1 If serum PTH is above the target range for the stage of CKD (see Guideline 1), serum 25-hydroxyvitamin D (25[OH]D) should be measured. **(EVIDENCE)** Periodic assessment is warranted thereafter if dietary or lifestyle changes have occurred in the patient. **(OPINION)**

8.2 If the serum level of 25-hydroxyvitamin D is <30 ng/mL, supplementation with vitamin D_2 (ergocalciferol) should be initiated (see Table below). **(OPINION)**

Recommended Supplementation for Vitamin D Deficiency/Insufficiency in Patients with CKD Stages 3-4

Serum 25(OH)D (ng/mL)	Definition	Ergocalciferol Dose (Vitamin D_2)	Duration (months)	Comment
<5	Severe vitamin D deficiency	8,000 IU/day orally x 4 weeks or (50,000 IU per week X 4 weeks); then 4,000 IU/day or, (50,000 IU 2X per month for 2 months) X 2 months	3 months	Measure 25(OH)D levels after 3 months
5-15	Mild vitamin D deficiency	4,000 IU/day orally x 12 weeks or (50,000 IU every other week, for 12 weeks)	3 months	Measure 25(OH)D levels after 3 months
16-30	Vitamin D insufficiency	2,000 IU daily or (50,000 IU every 4 weeks)	3 months	Measure 25(OH)D levels after 3 months

8.3 Following initiation of vitamin D supplementation:

8.3.a The use of ergocalciferol therapy should be integrated with the serum calcium and phosphorus levels (see Algorithm 1 in the original guideline document).

8.3.b The serum levels of corrected total calcium and phosphorus should be measured after 1 month, and then at least every 3 months. **(OPINION)**

8.3.c If the serum levels of corrected total calcium exceed 10.2 mg/dL (2.54 mmol/L), discontinue ergocalciferol therapy and all forms of vitamin D therapy. **(OPINION)**

8.3.d If the serum phosphorus exceeds the upper limits for age, initiate dietary phosphate restriction (see Guidelines 4 and 5) or, if hyperphosphatemia persists but the 25(OH)D is <30 ng/mL, initiate oral phosphate binder therapy. If the 25(OH)D is normal, discontinue vitamin D therapy. **(OPINION)**

8.3.e Once patients are replete with vitamin D, continued supplementation with a vitamin D-containing multivitamin preparation should be used with annual reassessment of serum levels of 25(OH)D, as should the continued assessment of corrected total calcium and phosphorus per stage of CKD (see Table above). **(OPINION)**

In CKD Stage 5:

8.4 Therapy with an active vitamin D sterol (calcitriol) should be provided if the serum levels of PTH are >300 pg/mL. **(OPINION)** (See Guideline 9.)

Guideline 9. Active Vitamin D Therapy in CKD Patients

This Guideline encompasses two parts: Guideline 9A, which is specific for CKD Stages 2-4, and Guideline 9B, which refers to CKD Stage 5.

Guideline 9a. Active Vitamin D Therapy in Patients with CKD Stages 2-4

9A.1 In patients with CKD Stages 2-4, therapy with an active oral vitamin D sterol (calcitriol) should be initiated when serum levels of 25(OH)D are >30 ng/mL (75 nmol/L), and serum levels of PTH are above the target range for the CKD stage (see Guideline 1). **(EVIDENCE)**

9A.1.a An active vitamin D sterol should be administered only in patients with serum levels of corrected total calcium <10 mg/dL (2.37 mmol/L) and serum levels of phosphorus less than age-appropriate upper limits (see Table below). **(OPINION)**

Serum Levels of PTH, Calcium, and Phosphate Required for Initiation of Oral Vitamin D Sterol Therapy, and Recommended Initial Doses in Patients with CKD Stages 2-4

Serum PTH (pg/mL or ng/L)	Serum Calcium (mg/dL)[mmol/L]	Serum Phosphate (mg/dL)	Dose Oral Calcitriol
>70 (CKD stage 2, 3)			
>110 (CKD stage 4)	<10 [2.37]	≤ age appropriate levels	<10kg: 0.05 micrograms every other day 10-20kg: 0.1-0.15 micrograms/day >20kg: 0.25 micrograms/day

9A.2 After initiation of active vitamin D sterols, serum levels of calcium and phosphorus should be measured at least monthly for the first 3 months, and at least every 3 months thereafter. Serum PTH levels should be measured at least every 3 months. **(OPINION)**

9A.3 The dosage of active vitamin D sterols should be adjusted as follows:

9A.3.a If serum levels of PTH decrease to values below the target range for the CKD stage (see Table, Guideline 1), active vitamin D sterol therapy should be held until serum levels of PTH increase to above the target range; treatment should then be resumed at half the previous dose of active vitamin D sterols. If the dosage is below a 0.25 microgram capsule or 0.05 microgram dose as liquid, alternate-day dosing should be used. **(OPINION)**

9A.3.b If serum levels of corrected total calcium exceed 10.2 mg/dL (2.37 mmol/L), active vitamin D sterol therapy should be held until serum calcium decreases to <9.8 mg/dL (2.37 mmol/L); treatment should then be resumed at half the previous dose. If the lowest daily dose of the active vitamin D sterol is being given, alternate-day dosing should be used. If the dosage is below a 0.25 microgram capsule or

0.05 microgram dose as liquid, alternate day dosing should be used. **(OPINION)** See algorithm 2 in the original guideline document.

9A.3.c The dosage of active vitamin D sterols should be adjusted downward as follows: If serum levels of phosphorus increase to greater than age-appropriate upper limits, active vitamin D therapy should be held; the dose of phosphate binders should be increased or initiated until the levels of serum phosphorus decrease to age-appropriate levels; then, treatment at half the prior dose of active vitamin D sterol should be resumed. **(OPINION)**

9A.4 The dosage of active vitamin D sterols should be adjusted upward as follows: If serum levels of PTH fail to decrease by at least 30% after the initial 3 months of therapy, and the serum levels of calcium and phosphorus are within the target ranges based on CKD stage, the dose of active vitamin D sterols should be increased by 50%. Serum levels of PTH, calcium, and phosphorus must be measured monthly for 3 months thereafter.

Guideline 9b. Active Vitamin D Therapy in Patients on Dialysis (CKD Stage 5)

9B.1 In patients with CKD Stage 5 and serum PTH levels >300 pg/mL, an active vitamin D sterol (calcitriol; see table below) should be administered to reduce the serum levels of PTH to a target range of 200-300 pg/mL. **(EVIDENCE)**

Serum PTH (pg/mL)	Serum Calcium (mg/dL)[mmol/L]	Serum Phosphate (mg/dL) [mmol/L]	CaXP*	Calcitriol Dose per HD Session**	Calcitriol Dose for Patients Receiving PD (TIW)***
300-500	<10 [2.37]	<5.5 [1.78] for adolescents <6.5 [2.10] for infants and children	<55 for adolescents <65 for infants and children	0.0075 micrograms/kg (maximum = .25 micrograms) qd	0.0075 micrograms/kg (maximum = .25 micrograms) qd
>500-1000	<10 [2.37]	<5.5 [1.78] for adolescents <6.5 [2.10] for infants and children	<55 for adolescents <65 for infants and children	0.015 micrograms/kg (maximum = 0.5 micrograms) qd	0.015 micrograms/kg (maximum = 0.5 micrograms) qd
>1000	<10 [2.50]	<5.5 [1.78] for adolescents <6.5 [2.10] for infants and children	<55 for adolescents <65 for infants and children	0.025 micrograms/kg (maximum = 1 microgram) qd	0.025 micrograms/kg (maximum = 1 microgram) qd

*<65 in children below 12 years of age

** HD: Hemodialysis, thrice weekly (TIW)

*** PD: Peritoniel dialysis, thrice weekly (TIW)

9B.2 The intermittent administration of calcitriol by intravenous or oral routes is more effective than daily oral calcitriol in lowering serum PTH levels. **(EVIDENCE)**

9B.3 When therapy with vitamin D sterols is initiated or the dose is increased, serum levels of calcium and phosphorus should be measured at least every 2 weeks for 1 month and then monthly thereafter. The serum PTH level should be measured monthly for at least 3 months and then at least every 3 months once target levels of PTH are achieved. **(OPINION)**

9B.4 For patients treated with peritoneal dialysis, initial oral doses of calcitriol (0.5-1.0 micrograms) can be given three times weekly. Alternatively, an equivalent lower dose of calcitriol (0.25 micrograms) can be administered daily. **(OPINION)**

9B.5 The dosage of active vitamin D sterols should be adjusted upward as follows: If serum levels of PTH fail to decrease by at least 30% after the initial 3 months of therapy, and the serum levels of calcium and phosphorus are within the target ranges based on CKD stage, increase the dose of active vitamin D sterols by 50%. Serum levels of PTH, calcium, and phosphorus must be measured monthly for 3 months thereafter.

9B.6 Treatment with active vitamin D sterols should be integrated with the changes in serum calcium, phosphorus, and PTH. A separate algorithm is shown for each of these three variables with suggested interventions based on the values obtained. **(OPINION)**

Guideline 10. Dialysate Calcium Concentrations

10.1 In patients receiving calcium-based phosphate binders, the dialysate calcium concentration should be targeted to 2.5 mEq/L (1.25 mM). **(OPINION)**

10.2 In patients not receiving calcium-containing phosphate binders, the dialysate calcium should be targeted to 2.5-3.0 mEq/L (1.25-3 mM) based on serum calcium levels and the need for therapy with active vitamin D sterols. **(OPINION)**

Guideline 11. Recommendations for the Use of Growth Hormone for Children with CKD

11.1 Children should have hip X-rays and a wrist bone age performed prior to initiation of growth hormone (GH) therapy. Children with active rickets or a slipped capital femoral epiphysis should not begin GH therapy until these problems have been resolved. **(EVIDENCE)**

11.2 Growth hormone therapy should not be initiated until the PTH level is no greater than 2X the target upper limit for CKD Stages 2-4 or 1.5X (450 pg/mL) the target upper limit in CKD Stage 5 (dialysis) (see Guideline 1). **(OPINION)**

11.3 Growth hormone therapy should not be initiated until the phosphorus is no greater than 1.5X the upper limit for age (see Guideline 4). **(OPINION)**

11.4 Children receiving GH therapy in Stages 2-4 CKD should have calcium, phosphorus, PTH, and alkaline phosphatase monitored at least every 3 months during the first year of therapy. Children receiving GH therapy in CKD Stage 5 should have calcium, phosphorus, PTH, and alkaline phosphatase monitored at least every month during the first 6 months of therapy. Thereafter, interval measurements should be made according to stage of CKD (see Guideline 1). **(OPINION)**

11.5 Children receiving GH therapy should have a wrist bone age performed yearly. Hip X-rays should be performed when clinically indicated.

11.6 Growth hormone therapy should be stopped temporarily:

11.6.a CKD Stages 2-4: If the patient has a PTH level >400 pg/mL; GH should not be restarted until the PTH level is $\leq$200 pg/mL **(EVIDENCE AND OPINION)**

11.6.b CKD Stage 5: If the patient has a PTH level >900 pg/mL; GH should not be restarted until the PTH level is $\leq$450 pg/mL **(EVIDENCE AND OPINION)**

11.6.c In all stages of CKD, if the patient develops a slipped capital femoral epiphysis or symptomatic high turnover renal osteodystrophy **(EVIDENCE)**

11.7 Growth hormone therapy should be stopped permanently when the epiphyses are closed.

Guideline 12. Aluminum Overload and Toxicity in CKD

12.1 To prevent aluminum toxicity, the regular administration of aluminum should be avoided and the dialysate concentration of aluminum should be maintained at <10 micrograms/L. **(EVIDENCE)**

12.1.a CKD patients ingesting aluminum should not receive citrate salts simultaneously. **(EVIDENCE)**

12.2 In CKD Stage 5, to assess aluminum exposure and the risk of aluminum toxicity, serum aluminum levels should be measured at least yearly and every 3 months in those receiving aluminum containing medications. **(OPINION)**

12.2.a In children with CKD prior to Stage 5, serum levels of aluminum should be measured yearly if children have been exposed to aluminum for 3 months or more in the prior year. **(OPINION)**

12.2.b Baseline levels of serum aluminum should be <20 micrograms/L. **(OPINION)**

12.2.c If levels of serum aluminum are between 20-60 micrograms/L, a search for and elimination of all sources of aluminum should be performed. **(OPINION)**

12.3 A desferrioxamine (DFO) test should be performed if there are elevated serum aluminum levels (60-200 micrograms/L) or clinical signs and symptoms of aluminum toxicity (see table below), or prior to parathyroidectomy if the patient has had aluminum exposure for at least 4 months or more. **(OPINION)** (See Algorithm 6 and Algorithm 7 in the original guideline document.)

12.3.a The test is performed by infusing 5 mg/kg of DFO during the last hour of the dialysis session with a serum aluminum measured both before DFO infusion and 2 days later, before the next dialysis session. **(OPINION)**

12.3.b The test is considered positive if the increment of serum aluminum is $\geq$50 micrograms/L. **(OPINION)**

12.3.c A DFO test should not be performed if the serum levels of aluminum are >200 micrograms/L to avoid DFO-induced neurotoxicity. **(OPINION)**

12.4 The presence of aluminum bone disease can be predicted by a rise in serum aluminum of $\geq$50 micrograms/L following DFO challenge combined with serum PTH levels of <150 pg/mL (150 ng/L). **(OPINION)** However, the gold standard for the diagnosis of aluminum bone disease is a bone biopsy showing increased aluminum staining of the bone surface (>15%-25%) using an aluminum-specific stain, and often the presence of adynamic bone or osteomalacia. **(EVIDENCE)**

12.5 Asymptomatic patients receiving maintenance hemodialysis, with elevated levels of serum aluminum between 60-200 micrograms/L, should be treated with removal of aluminum based gels and intensive dialysis. Treatment with DFO is optional unless desired serum aluminum levels are not achieved. **(OPINION)**

Aluminum-Related Disorders: Features, Causes, and Considerations for Therapy

Condition	Features (Clinical Diagnosis)	Causes	Management	Special Treatment
Acute Aluminum Neurotoxicity	Acute neurological syndrome with: Altered consciousness Seizures	1. Dialysate Al>200 micrograms/L 2. HD patients with marked Al-loading (P_{Al}>200 micrograms/L) treated	Measure serum Al Stop all Al intake Dialysate Al<5 micrograms/L	Standard management plus: Cause 2: Stop DFO until P_{Al}<200 micrograms/L Cause 3: Withdraw all citrate*

Condition	Features (Clinical Diagnosis)	Causes	Management	Special Treatment
	Coma Usually progresses to death	with DFO, 20 to 40 mg/kg 3. Stage 4 & 5 CKD patients who ingest both Al-drugs plus a salt containing citrate*	Daily dialysis High-flux dialyzer Follow algorithms for DFO testing & therapy	
Dialysis Encephalopathy	Subacute syndrome with: Speech abnormalities Defective spatial orientation Altered consciousness Seizures Often intermittent and worsens transiently after dialysis Usually slowly progressive	1. Dialysate Al>30 to 40 micrograms/L (with dialysate Al levels of 100 to 200 micrograms/L, symptoms appear sooner and progress more rapidly) 2. Rarely arises from Al ingestion alone, but ingestion of Al-containing agents can hasten its appearance	Measure serum Al Stop all Al intake Dialysate Al<5 micrograms/L High flux dialyzer Follow algorithms regarding need for daily dialysis, DFO testing & therapy	Know level of serum Al before doing DFO test Specific electroencephalographic features can aid in the diagnosis (see text in original guideline document)
Aluminum Bone Disease	Insidious appearance of: Bone pain Fractures Proximal muscle weakness	1. Dialysate Al>30 to 40 micrograms/L (with higher Al levels, symptoms appear sooner) 2. Ingestion of Al-	Measure serum Al Stop all Al intake Dialysate Al<5 micrograms/L High flux dialyzer	Know level of serum Al before DFO test is done May coexist with dialysis encephalopathy, particularly when dialysate Al>30-40 micrograms/L

Condition	Features (Clinical Diagnosis)	Causes	Management	Special Treatment
	(Diagnosis by bone biopsy; prediction from DFO test result and intact PTH level)	containing agents may hasten its development	Follow algorithms regarding need for daily dialysis, DFO testing & therapy	
Hypercalcemia	Search may reveal other features of Al toxicity when hypercalcemia appears in the absence of either: High intact PTH levels (e.g. when intact PTH>500 pg/mL), or Vitamin D therapy	1. Dialysate Al>30 to 40 micrograms/L 2. Ingestion of Al-containing drugs Serum Ca can rise rapidly with use of Ca-based phosphate binders—probable manifestation of low bone turnover	Measure serum Al Stop all Al intake Dialysate Al<5 micrograms/L Follow algorithms regarding need for daily dialysis, high-flux dialyzer use, DFO testing & therapy	Hypercalcemia can dissipate rapidly with use of lower dialysate Ca (2.0-2.5 mEq/L)
Microcytic Anemia	When microcytosis presents with: No evidence of iron deficiency No response to iron therapy	1. Dialysate Al>30 to 40 micrograms/L 2. Ingestion of Al-containing drugs (uncommonly the only source of Al loading)	Measure serum Al Stop all Al intake Dialysate Al<5 micrograms/L Follow algorithms regarding need for daily dialysis, high-flux dialyzer use, DFO testing & therapy	Aluminum loading may increase requirements for erythropoietin but magnitude of this effect is not well documented
Aluminum Overload	Asymptomatic (by definition)	1. Dialysate Al>20 to 40	Measure serum Al	May be subtle abnormalities on CNS

Condition	Features (Clinical Diagnosis)	Causes	Management	Special Treatment
	(Defined by analysis of bone Al content or by a specific but arbitrary rise of P_{Al} after a DFO test)	micrograms/L 2. Ingestion of Al-containing drugs	Stop all Al intake Dialysate Al<5 micrograms/L Follow algorithms regarding need for high-flux dialyzer use, DFO testing & therapy	testing May respond to withdrawal of all exposure to Al DFO treatment rarely needed

*Citrate source may be Bictra™ Shohl's solution, AlkaSeltzer™, calcium citrate, or excess intake of citrate-containing juices.

Abbreviations: Al, aluminum; P_{Al}, serum aluminum, DFO, desferrioxamine; CNS central nervous system; HD, hemodialysis; PTH, parathyroid hormone

Guideline 13. Treatment of Aluminum Toxicity

13.1 In all patients with baseline serum aluminum levels between 20-60 micrograms/L, a positive DFO test, or clinical symptoms consistent with aluminum toxicity (see guideline 12 table), the source of aluminum should be identified and eliminated. **(OPINION)**

13.2 In symptomatic patients with serum aluminum levels >60 micrograms/L but <200 micrograms/L or increase in aluminum after DFO >50 micrograms/L, DFO should be given to treat the aluminum overload. (See Algorithm 8 and Algorithm 9 in the original guideline document.) **(OPINION)**

13.3 To avoid DFO-induced neurotoxicity in patients with serum aluminum >200 micrograms/L, DFO should not be given until the predialysis serum aluminum level has been reduced to <200 micrograms/L, which can be achieved by intensive dialysis with high-flux dialysis membrane and a dialysate aluminum level of <5 micrograms/L. **(OPINION)**

Guideline 14. Treatment of Bone Disease in CKD

The treatment of bone disease in CKD is based on its specific type. This Guideline encompasses three parts: Guideline 14A deals with high-turnover bone disease; Guideline 14B with rickets/osteomalacia; and Guideline 14C with adynamic bone disease.

Guideline 14a. Hyperparathyroid (High-Turnover) Bone Disease

14A.1 In CKD patients who have serum PTH levels >70 pg/mL (Stages 2-3) or >110 pg/mL (Stage 4), dietary phosphate intake should be modified according to Guidelines 5 and 6, and dietary calcium should be modified according to Guideline 7. Nutritional vitamin D insufficiency or deficiency should be corrected according to Guideline 8. If a repeat first-generation immunometric parathyroid hormone assay (1^{st} PTH-IMA) after 3 months of dietary intervention shows that PTH levels remain elevated, then patients should be treated with calcitriol or 1-alpha-vitamin D_2 **(EVIDENCE)**, to prevent or ameliorate high-turnover bone disease.

14A.2 In CKD Stage 5 patients who have elevated serum PTH levels (>300 pg/mL) despite modification of dietary phosphate intake according to Guidelines 5 and 6, calcitriol or 1-alpha-vitamin D_2 **(EVIDENCE)** should be used to reverse the bone features of hyperparathyroidism (i.e., high-turnover bone disease).

Guideline 14b. Rickets/Osteomalacia

14B.1 Rickets and osteomalacia due to aluminum toxicity should be prevented in dialysis patients by maintaining aluminum concentration in dialysate fluid at <10 micrograms/L and by avoiding the use of aluminum-containing compounds. **(OPINION)**

14B.2 Rickets and osteomalacia due to vitamin D deficiency should be treated according to Guideline 7. **(EVIDENCE)**

14B.3 Rickets and osteomalacia due to hypophosphatemia should be treated with neutral sodium phosphate salts. Concomitant active vitamin D therapy should be considered. See Guidelines 7 and 8. **(EVIDENCE)**

Guideline 14c. Adynamic Bone Disease

14C.1 In CKD Stage 5, adynamic bone disease not related to aluminum (as determined either by bone biopsy or suggested by PTH <150 pg/mL) should be treated by allowing serum levels of PTH to rise in order to increase bone turnover. **(OPINION)**

> 14C.1.a The increase in PTH levels can be accomplished by discontinuing treatment with activated vitamin D analogs, decreasing or eliminating the use of calcium-based phosphate binders, reducing the dialysate calcium concentration (see Guideline 8) **(EVIDENCE)**, and/or using a metal-free phosphate binder. **(OPINION)**

Guideline 15. Parathyroidectomy in Patients with CKD

15.1 Parathyroidectomy should be considered in patients with severe hyperparathyroidism (persistent serum levels of PTH >1,000 pg/mL [1,000 ng/L]), and disabling bone deformities associated with hypercalcemia and/or hyperphosphatemia that are refractory to medical therapy. **(OPINION)**

15.2 Effective surgical therapy of severe hyperparathyroidism can be accomplished by subtotal parathyroidectomy or total parathyroidectomy with parathyroid tissue autotransplantation. **(EVIDENCE)**

15.2a Total parathyroidectomy probably is not the procedure of choice in patients who may subsequently receive a kidney transplant, since the subsequent control of serum calcium levels may be problematic.

15.3 In patients who undergo parathyroidectomy the following should be done:

15.3.a In the 72 hours prior to parathyroidectomy, consideration should be given to administration of calcitriol or other active vitamin D sterols, to lessen postoperative hypocalcemia.

15.3.b The blood level of ionized calcium should be measured every 4-6 hours for the first 24 hours after surgery, and then less frequently until less stable. **(OPINION)**

15.3.c If the level of ionized calcium falls below normal (<1 mM or <4 mg/dL, corresponding to corrected total calcium of 7.2 mg/dL [1.80 mmol/L]), a calcium gluconate infusion should be initiated at a rate of 1-2 mg elemental calcium per kilogram body weight per hour and adjusted to maintain an ionized calcium in the normal range (1.15-1.36 mM or 4.6-5.4 mg/dL). **(OPINION)** A 10-mL ampule of 10% calcium gluconate contains 90 mg of elemental calcium.

15.3.d The calcium infusion should be gradually reduced when the level of ionized calcium attains the normal range and remains stable. **(OPINION)**

15.3.e When oral intake is possible, the patient should receive elemental calcium 1-2 g, three times a day, as well as calcitriol 1-2 micrograms/day, and these therapies should be adjusted as necessary to maintain the level of ionized calcium in the normal range. **(OPINION)**

15.3.f If the patient was receiving phosphate binders prior to surgery, this therapy may need to be discontinued or reduced as dictated by the levels of serum phosphorus. **(OPINION)**

15.4 Imaging of parathyroid glands with ^{99}Tc-Sestamibi scan, ultrasound, CT scan, or magnetic resonance imaging (MRI) should be done prior to re-exploration parathyroid surgery. **(OPINION)**

Guideline 16. Metabolic Acidosis

16.1 In CKD Stages 1-5, the serum level of total carbon dioxide (CO_2) should be measured.

16.1.a The frequency of these measurements should be based on the stage of CKD as shown in the table below. **(OPINION)**

Frequency for Measurement of Serum Levels of Total CO_2

CKD Stage	GFR Range mL/min/1.73m^2	Frequency of Measurement
1	>90	At least every 12 months
2	60-89	At least every 12 months
3	30-59	At least every 6 months
4	15-29	At least every 3 months
5	<15 Dialysis	At least every 3 months At least every month

Abbreviations: CO_2, Carbon Dioxide; CKD, Chronic Kidney Disease, GFR, Glomerular Filtration Rate

16.2 In patients >2 years of age, serum levels of total CO_2 should be maintained at $\geq$22 mEq/L (22 mmol/L); in neonates and young infants below age two, serum levels of total CO_2 should be maintained at $\geq$20 mEq/L (20 mmol/L). **(EVIDENCE)** If necessary, supplemental alkali salts should be given to achieve this goal. Elevation of the bicarbonate concentration in the hemodialysis bath is an additional or alternative strategy. **(OPINION)**

Guideline 17. Bone Disease in the Pediatric Kidney Transplant Recipient

17.1 Serum levels of calcium, phosphorus, total CO_2 and PTH should be monitored following kidney transplantation. **(OPINION)**

17.1.a The frequency of these measurements should be at least as often as shown in the table below. **(OPINION)**

Frequency of Measurement of Calcium, Phosphorus, PTH, and Total Carbon Dioxide (CO_2) after Kidney Transplantation

Parameter	First Week	First 2 Months	From 2-6 Months
Calcium	Daily	Weekly	Monthly
Phosphorus	Daily	Weekly	Monthly
PTH	Optional	Once at one month, thereafter optional	Optional if normal initially
Total CO_2	Daily	Every 1 week	Monthly

17.1.b Six months after transplantation, the frequency of measurements should follow the recommendations of Guideline 1, depending on the stage of CKD.

17.2 The care of osteodystrophy in kidney transplant patients reaching CKD Stage 2 and below should follow the guidelines established for native CKD. **(OPINION)**

17.3 Kidney transplant recipients who develop persistent hypophosphatemia (below the age-appropriate lower limits) should be treated with phosphate supplementation. **(OPINION)**

17.4 To minimize bone mass loss and osteonecrosis, the lowest effective dose of glucocorticoids should be used. **(OPINION)**

CLINICAL ALGORITHM(S)

The following algorithms are provided in the original guideline document:

- Vitamin D Supplementation in Chronic Kidney Disease (CKD) Stages 2-4
- Management of CKD Patients (Stages 2-4) with Active Vitamin D Sterols
- Managing Vitamin D Sterols Based on Serum Calcium Levels
- Managing Vitamin D Sterols Based on Serum Phosphorus Levels
- Managing Vitamin D Sterols Based on Parathyroid Hormone (PTH) Levels in Children Not Receiving Growth Hormones
- Evaluation of Aluminum Neurotoxicity
- Evaluation of Aluminum-Related Disorders: Considerations for desferrioxamine (DFO) Test and Subsequent DFO Treatment
- DFO Treatment after Serum Aluminum (P_{Al}) Rise between 50-300 micrograms/L
- Subsequent DFO Treatment after P_{Al} Rise $\geq$300 micrograms/L

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of evidence supporting the recommendations is not specifically stated.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

- Accurate assessment of bone disease in pediatric patients with chronic kidney disease
- Maintenance of appropriate serum levels of phosphorus, calcium, parathyroid hormone (PTH), vitamin D
- Reduced incidence and severity of deformity, restored function and mobility, and improved self-esteem in patients with osteodystrophy
- Reduced incidence and shortened duration of aluminum toxicity
- Improved linear growth
- Reduced incidence of and morbidity and mortality from:
    - Bone disease
    - Bone deformities
    - Deranged mineral ion homeostasis
    - Neurological abnormalities
    - Bone fractures
    - Impaired overall mineral ion homeostasis
    - Hyperparathyroidism
- Prevention and correction of metabolic acidosis

POTENTIAL HARMS

- In children receiving aluminum-based phosphate binders, concurrent use of citrate-based products should be avoided, due to the risk of increasing aluminum absorption and potential toxicity.

- A desferrioxamine (DFO) test should not be performed if the serum levels of aluminum are >200 micrograms/L to avoid DFO-induced neurotoxicity.
- These guidelines recommend close monitoring of mineral metabolism during recombinant growth hormone (rhGH) therapy in children with CKD. A patient who has poorly controlled secondary hyperparathyroidism, active rickets, or a slipped capital femoral epiphysis may be at increased risk for more severe bone disease if rhGH therapy is continued. Given this potential, it is prudent not to use rhGH in children with poorly controlled osteodystrophy.
- Total parathyroidectomy probably is not the procedure of choice in patients who may subsequently receive a kidney transplant, since the subsequent control of serum calcium levels may be problematic.

CONTRAINDICATIONS

CONTRAINDICATIONS

- Children with active rickets or a slipped capital femoral epiphysis should not begin growth hormone therapy until these problems have been resolved.
- Chronic kidney disease patients ingesting aluminum should not receive citrate salts simultaneously.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

See the "Limitations" sections for each guideline in the original guideline document for information on limitations of the available evidence.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Clinical Algorithm

For information about availability, see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Living with Illness

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

KDOQI, National Kidney Foundation. K/DOQI clinical practice guidelines for bone metabolism and disease in children with chronic kidney disease. Am J Kidney Dis 2005 Oct;46(4 Suppl 1):S1-121. [557 references]

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2005 Oct

GUIDELINE DEVELOPER(S)

National Kidney Foundation - Disease Specific Society

SOURCE(S) OF FUNDING

The development of the K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease in Children with Chronic Kidney Disease is supported by an unrestricted educational grant from Abbott Renal Care. Additional support was received from Amgen Inc. and Genzyme Therapeutics.

GUIDELINE COMMITTEE

NKF-K/DOQI (National Kidney Foundation-Kidney Disease Outcomes Quality Initiative) Bone Metabolism and Disease in Children with Chronic Kidney Disease Work Group

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Work Group Members: Craig B. Langman, MD (*Co-Chair*) Northwestern University Feinberg School of Medicine, Children's Memorial Hospital, Chicago, IL; Isidro B. Salusky, MD (*Co-Chair*) David Geffen School of Medicine at UCLA, Los Angeles, CA; Larry Greenbaum, MD, PhD, Medical College of Wisconsin, Milwaukee, WI; Harald Jueppner, MD, Massachusetts General Hospital, Harvard Medical School, Boston, MA; Mary Leonard, MD, Children's Hospital of Philadelphia, Philadelphia, PA; Pauline Nelson, RD, UCLA Medical Center, Los Angeles, CA; Anthony Portale, MD, University of California San Francisco, San Francisco, CA; Bradley A. Warady, MD, The Children's Mercy Hospital, Kansas City, MO

Consultants to the Work Group: Richard E. Bowen, MD, Assistant Clinical Professor, David Geffen School of Medicine at UCLA, Los Angeles, CA; William L. Oppenheim, MD, David Geffen School of Medicine at UCLA, Los Angeles, CA

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

The National Kidney Foundation makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the Work Group.

Specifically, all members of the Work Group are required to complete, sign, and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest. All affiliations are published in their entirety at the end of the original guideline document in the biographical sketches of the Work Group Members.

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available from the National Kidney Foundation (NKF) Web site.

Print copies: Available from the National Kidney Foundation (NKF), 30 East 33rd St., New York, NY 10016. These guidelines are also available on CD-ROM from NKF.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This NGC summary was completed by ECRI on September 11, 2006. The information was verified by the guideline developer on November 29, 2006.

COPYRIGHT STATEMENT

DISCLAIMER

NGC DISCLAIMER

The National Guideline Clearinghouse™ (NGC) does not develop, produce, approve, or endorse the guidelines represented on this site.

All guidelines summarized by NGC and hosted on our site are produced under the auspices of medical specialty societies, relevant professional associations, public or private organizations, other government agencies, health care organizations or plans, and similar entities.

Guidelines represented on the NGC Web site are submitted by guideline developers, and are screened solely to determine that they meet the NGC Inclusion Criteria which may be found at http://www.guideline.gov/about/inclusion.aspx .

NGC, AHRQ, and its contractor ECRI Institute make no warranties concerning the content or clinical efficacy or effectiveness of the clinical practice guidelines and related materials represented on this site. Moreover, the views and opinions of developers or authors of guidelines represented on this site do not necessarily state or reflect those of NGC, AHRQ, or its contractor ECRI Institute, and inclusion or hosting of guidelines in NGC may not be used for advertising or commercial endorsement purposes.

Readers with questions regarding guideline content are directed to contact the guideline developer.

Date Modified: 11/3/2008