INDIAN HEALTH SERVICE
National Pharmacy and Therapeutics Committee
Formulary Brief: ***Pediatric Chronic Kidney Disease***
-February 2026-

Background:

The Indian Health Service (IHS) National Pharmacy and Therapeutics Committee (NPTC) provided a drug class review of pediatric chronic kidney disease agents. This marks the first NPTC review of medications specific to this condition. Medications listed on the NCF relevant to this condition include calcium, furosemide, hydrochlorothiazide, iron (oral), lisinopril, losartan, prednisone, sevelamer, and vitamin D. Following clinical review and analysis, the NPTC **made no modifications** to the National Core Formulary.

Discussion:

Kidney Disease: Improving Global Outcomes (KDIGO) defines chronic kidney disease (CKD) as structural or functional kidney damage that persists for at least 3 months. For children two years of age and older, GFR is used to stage CKD and stratify risk for progression and complications. Patients under two years of age are compared with age-specific standards for GFR to categorize reduction in kidney function. [1] Incidence and prevalence of pediatric CKD is difficult to ascertain as early stages of CKD are often asymptomatic resulting in underdiagnosis. Incidence of pediatric CKD in Europe is 11-12 per million of the age-related population and the National Health and Nutrition Examination Survey (NHANES) from 2005-2020 found a prevalence of 0.51% for children aged 12-17 in the United States.[2,3]

The etiology of pediatric CKD is vastly different than adult CKD with 60% of cases attributed to congenital anomalies of the kidney and urinary tract (CAKUT) including kidney aplasia/hypoplasia/dysplasia, reflux nephropathy, obstructive uropathy anomalies (e.g., posterior urethral valve), and polycystic kidney disease. Approximately 10-20% of pediatric CKD are from glomerular diseases such as focal segmental glomerulosclerosis, hemolytic uremic syndrome, and secondary glomerular disease (e.g., systemic lupus nephritis). Other causes of pediatric CKD include genetic disorders such as Alport syndrome, interstitial nephritis, or unknown etiology. Diabetic nephropathy and hypertension are rare causes of CKD in children.[4]

Routine health maintenance for children with CKD include frequent monitoring of growth and nutrition, monitoring of blood pressure, periodic lab evaluation, developmental surveillance and screening, and immunizations aside from live-attenuated vaccines in patients who are immunosuppressed.[1, 5]

To slow the progression of pediatric CKD, it is important to treat the underlying causes of kidney disease whether that be correcting obstructive uropathy or high-grade vesicoureteral reflux or using immunosuppressive or immune-modulating therapy for primary nephrotic syndrome and primary and secondary glomerulonephritis. Other measures to prevent or delay progression of kidney disease include avoiding subsequent kidney injury (e.g., avoiding kidney hypoperfusion, avoiding nephrotoxic drugs) and strict blood pressure control.[1, 5] The ESCAPE trial recommends blood pressure (BP) target <75th percentile in non-proteinuric and <50th percentile in proteinuric patients younger than 16 years of age and BP <130/80 mmHg in non-proteinuric and <125/75 mmHg in adolescents aged 16 and older.[6] The 2017 American Academy of Pediatrics (AAP) Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children recommends lowering BP to <50th percentile in children with CKD regardless of degree of proteinuria.[7] The ESCAPE trial, European Society of Hypertension, AAP, and KDIGO all recommend the use of renin-angiotensin-aldosterone system antagonists (angiotensin-converting enzyme inhibitors [ACEi] or angiotensin II receptor blockers [ARBs]) as first-line antihypertensive therapy.[8]

There are many complications of pediatric CKD including but not limited to: fluid and electrolyte abnormalities, chronic kidney disease-mineral and bone disorder (CKD-MBD), anemia, cardiovascular disease, malnutrition and poor growth, and uremia. Sodium and water homeostasis can be tenuous and require access to free water and salt supplementation in instances of salt-wasting and poor urinary concentration or sodium restriction and diuretics in instances of salt and fluid overload. Hyperkalemia can occur due to decreased potassium excretion due to reduced GFR and may require low potassium diets and/or potassium binders; breastmilk is recommended for infants due to low potassium content and close monitoring with a pediatric renal dietician is suggested if using formula. Sodium bicarbonate therapy may be necessary to treat metabolic acidosis.[9]

Biochemical abnormalities (calcium, phosphate, parathyroid hormone, 1,25-dihydroxyvitamin D), bone abnormalities (short stature, reduced mineralization, increased risk of fractures), and extra-skeletal calcification constitute CKD-MBD and is a result of secondary hyperparathyroidism. Regulating phosphorus and calcium balance is the primary treatment of

CKD-MBD; dietary regimen is first-line management and phosphate binders are used when dietary regimen is insufficient.[10]

Anemia is screened for annually and diagnosed when hemoglobin (Hb) is below the 2.5th percentile of normal for sex and age. Anemia of CKD is normocytic and normochromic. Microcytic anemia suggests iron deficiency and can be treated with iron therapy if confirmed. Target Hb levels are between 11 and 12 g/dL and use of erythropoiesis-stimulating agents is indicated when Hb <10 g/dL and iron stores are replete.[9]

Malnutrition and growth failure are common complications of pediatric CKD. Energy intake should follow recommendations for healthy children of the same chronological age or trend towards the higher end of standard dietary intake for patients with suboptimal weight gain or linear growth. Protein intake should be targeted at the upper end of standard dietary intake for children with CKD, higher than standard dietary intake for children on dialysis, and the lower end of standard dietary intake for children with high blood urea levels. Growth failure is height below the third percentile or height velocity below the 25th percentile for more than 3 months in infants or 6 months in children and adolescents. If catch-up growth is not induced within 3 months of attempted nutritional management, growth hormone therapy should be initiated.[11,12]

Findings:

Pediatric CKD is a lifelong complex disease with significant morbidity and mortality. Progression and potential adverse outcomes of CKD can be delayed by early detection and treatment. Frequent monitoring of blood pressure and strict blood pressure control can help slow progression of CKD; first-line therapy for hypertension in pediatric CKD are ACE inhibitors and ARBs. Complications of pediatric CKD include electrolyte disturbances, mineral and bone disease, cardiovascular disease, malnutrition, and growth failure; screening for complications should occur at regular intervals determined by CKD staging and treated appropriately.

If you have any questions regarding this document, please contact the NPTC at IHSNPTC1@ihs.gov . For more information about the NPTC, please visit the NPTC website.

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