World Kidney Day

11 March 2021

LIVING WELL WITH KIDNEY DISEASE

Chronic Kidney Disease and I

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INTRODUCTION

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- Decreased kidney function shown by glomerular filtration rate (GFR) of less than 60 mL/min per 1·73 m2, or markers of kidney damage, or both, of at least 3 months duration, regardless of the underlying cause.

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INTRODUCTION…

- If duration is >3 months, CKD is confirmed. Follow recommendations for CKD.
- If duration is not>3 months or unclear, CKD is not confirmed. Patients may have CKD or acute kidney diseases (including AKI) or both and tests should be repeated accordingly.

KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease

INTRODUCTION…

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- Diabetes and hypertension are the main causes of CKD in all high-income and middle-income countries, and also in many low-income countries
- Incidence, prevalence, and progression of CKD also vary within countries by ethnicity and social determinants of health, possibly through epigenetic influence. Many people are asymptomatic or have non-specific symptoms such as lethargy, itch, or loss of appetite.

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RISK FACTORS

- Small for gestation birth weight
- Childhood obesity
- Hypertension
- Diabetes mellitus
- Autoimmune disease
- Advanced age
- African ancestry
- Family history of kidney disease
- A previous episode of acute kidney injury, and
- The presence of proteinuria, abnormal urinary sediment, or structural abnormalities of the urinary tract.

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STAGINGOF CKD

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- To stage CKD, it is necessary to estimate the GFR rather than relying on serum creatinin concentration.
- Measurement of albuminuria is also helpful for monitoring nephron injury and the response to therapy in many form of CKD.

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Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012

GFR categories (ml/min/1.73 m²) description and range				Persistent albuminuria categories description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60–89			
	G3a	Mildly to moderately decreased	45–59			
	G3b	Moderately to severely decreased	30–44			
	G4	Severely decreased	15–29			
	G5	Kidney failure	<15			

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GENERAL MANAGEMENT OF CHRONIC KIDNEY DISEASE

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- Treatment of reversible causes of kidney failure
- Preventing or slowing the progression of kidney disease
- Treatment of the complications of kidney failure
- Adjusting drug doses when appropriate for the level of estimated glomerular filtration rate (eGFR)
- Identification and adequate preparation of the patient in whom kidney replacement therapy will be required

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Reversible causes of kidney failure

- Decreased renal perfusion
- Administration of nephrotoxic drugs
- Urinary tract obstruction

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Blood pressure control

- Hypertension is present in approximately 80 to 85 percent of patients with CKD.
- Treating hypertension can both slow the progression of proteinuric CKD and reduce the rate of cardiovascular complications

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Blood pressure control…

- We recommend that in both diabetic and non-diabetic adults with CKD and urine albumin excretion <30 mg/24 hours whose office BP is consistently >140mm Hg systolic or >90mm Hg diastolic be treated with BP-lowering drugs to maintain a BP that is consistently <140mm Hg systolic and <90mm Hg diastolic.

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Blood pressure control…

- We suggest that in both diabetic and non-diabetic adults with CKD and with urine albumin excretion of >30 mg/24 hours whose office BP is consistently >130mm Hg systolic or >80mm Hg diastolic be treated with BP-lowering drugs to maintain a BP that is consistently <130mm Hg systolic and <80mm Hg diastolic.

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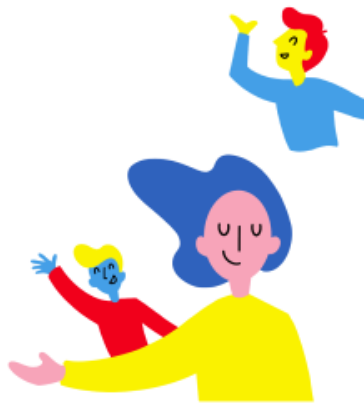
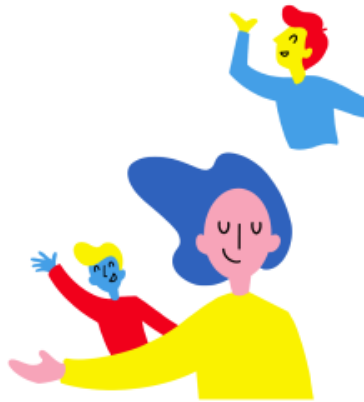
Choice of antihypertensive therapy

- Antihypertensive therapy in proteinuric CKD
- Angiotensin-converting enzyme (ACE) inhibitor or angiotensin II receptor blocker (ARB)



Choice of antihypertensive therapy…

- Antihypertensive therapy in nonproteinuric CKD
- In patients with edema initial therapy with a loop diuretic. Once the edema is controlled, an angiotensin inhibitor or a dihydropyridine calcium channel blocker (eg, amlodipine) can be added in either order if hypertension persists.
- In patients without edema, we start with an angiotensin inhibitor and then add a dihydropyridine calcium channel blocker (eg, amlodipine) as second-line therapy.



Other targets for renal protection

- Protein restriction
- Smoking cessation – Stopping smoking is associated with a slower rate of progression of CKD
- Treatment of chronic metabolic acidosis with supplemental bicarbonate may slow the progression to end-stage kidney disease (ESKD).
- Glycemic control

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Lifestyle

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- People with CKD be encouraged to undertake physical activity compatible with cardiovascular health and tolerance (aiming for at least 30 minutes 5 times per week),
- Achieve a healthy weight (BMI 20 to 25, according to country specific demographics
- Stop smoking.

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Protein intake

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- Protein intake
  - We suggest lowering protein intake to 0.8 g/kg/day in adults with diabetes or without diabetes and GFR <30 ml/min/ 1.73 m2 (GFR categories G4-G5), with appropriate education.
  - We suggest avoiding high protein intake (>1.3 g/kg/day) in adults with CKD at risk of progression.

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Salt intake

- We recommend lowering salt intake to <90mmol (<2 g) per day of sodium (corresponding to 5 g of sodium chloride) in adults

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Treatment of the complications of kidney failure

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- Volume overload
- Hyperkalemia
- Metabolic acidosis
- Mineral and bone disorders (MBD)
- Hypertension
- Anemia
- Dyslipidemia
- Sexual dysfunction

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Disorders of calcium and phosphate metabolism

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- Bone Manifestations of CKD:
- High bone turnover with increased PTH levels (including osteitis fibrosa cystica, the classic lesion of secondary hyperparathyroidism), osteomalacia due to reduced action of the active forms of vitamin D.
- Low bone turnover with low or normal PTH levels (adynamic bone disease)
- Most often combinations of the foregoing.

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The pathophysiology of secondary hyperparathyroidism

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- Declining GFR leads to reduced excretion of phosphate
- The retained phosphate stimulates increased synthesis of both FGF-23 by osteocytes and PTH and stimulates growth of parathyroid gland mass
- Decreased levels of ionized calcium, resulting from suppression of calcitriol production by FGF-23 and by the failing kidney, as well as phosphate retention, also stimulate PTH production.

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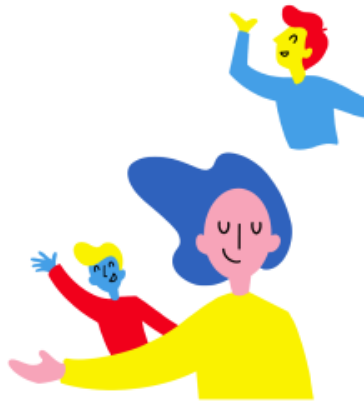
The pathophysiology of secondary hyperparathyroidism

- FGF-23 may defend normal serum phosphorus in at least three ways:
- Increased renal phosphate excretion.
- Stimulation of PTH, which also increases renal phosphate excretion.
- Suppression of the formation of 1,25(OH)2D3, leading to diminished phosphorus absorption from the GI tract.



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The pathophysiology of secondary hyperparathyroidism

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- Clinical manifestations of severe hyperparathyroidism include bone pain and fragility, brown tumors, compression syndromes, and erythropoietin (EPO) resistance in part related to the bone marrow fibrosis.

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Adynamic bone disease

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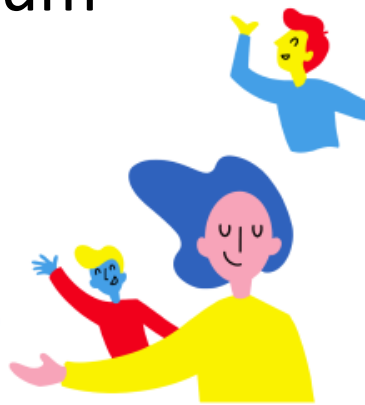
- Characterized by reduced bone volume and mineralization and may result from excessive suppression of PTH production, chronic inflammation, or both.
- Suppression of PTH can result from the use of vitamin D preparations or from excessive calcium exposure in the form of calcium-containing phosphate binders or high-calcium dialysis solutions.



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Disorders of Calcium and Phosphate Metabolism

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- TREATMENT
- The optimal management of secondary hyperparathyroidism and osteitis fibrosa is prevention.
- Low-phosphate diet
- Phosphate-binding agents.
- Calcitriol
- Calcimimetic agents

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Cardiovascular abnormalities

- Ischemic Vascular Disease
- Heart Failure
- Hypertension and Left Ventricular Hypertrophy
- Pericardial Disease

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Cardiovascular Abnormalities

- Management of hypertension
- Management of cardiovascular disease
- Management of pericardial disease

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Management of pericardial diseas

- Uremic pericarditis is an absolute indication for the urgent initiation of dialysis or for intensification of the dialysis prescription in those already receiving dialysis.
- Because of the propensity to hemorrhage in pericardial fluid, hemodialysis should be performed without heparin.

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Hematologic abnormalities

- Anemia:
  - A normocytic, normochromic anemia is observed as early as stage 3 CKD and is almost universal by stage 4.
  - The primary cause is insufficient production of EPO by the diseased kidneys.
- Abnormal Hemostasis

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Anemia

- TREATMENT
- Target a hemoglobin concentration of 10–11.5 g/dL.
- Erythropoietic-stimulating agents (ESA)
- Oral iron supplementation

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REFERRAL TO NEPHROLOGISTS

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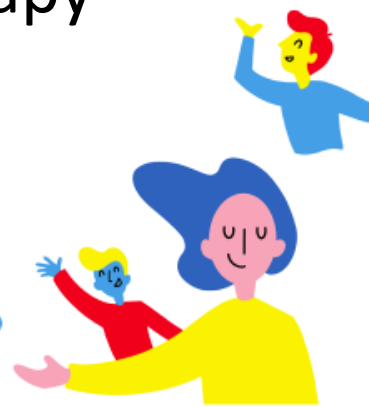
- Patients with CKD should be referred to a nephrologist when the estimated glomerular filtration rate (eGFR) is <30 mL/min/1.73 m2 in order to discuss and potentially plan for kidney replacement therapy.
- referral to the nephrologist is considered late if it is within one to six months of the requirement for kidney replacement therapy



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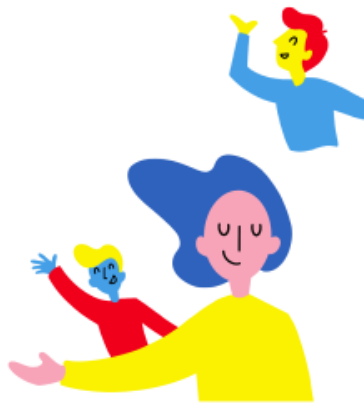
Indications for kidney replaceme therapy

- Pericarditis or pleuritis (urgent indication).
- Progressive uremic encephalopathy or neuropathy, with signs such as confusion, asterixis, myoclonus, wrist or foot drop, or, in severe, cases, seizures (urgent indication).
- A clinically significant bleeding diathesis attributable to uremia (urgent indication).
- Fluid overload refractory to diuretics.



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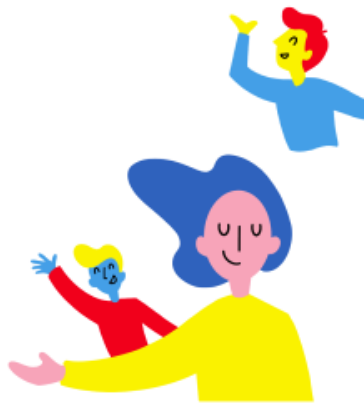
Indications for kidney replacement therapy …

- Hypertension poorly responsive to antihypertensive medications.
- Persistent metabolic disturbances that are refractory to medical therapy. These include hyperkalemia, hyponatremia, metabolic acidosis, hypercalcemia, hypocalcemia, and hyperphosphatemia.
- Persistent nausea and vomiting.
- Evidence of malnutrition.



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