Urinary System
Chronic Kidney Disease (CKD)

Involves progressive, irreversible loss
Defined as one of the following:

1. Kidney damage (markers of damage)

2. Low glomerular filtration rate
   < 60 mL/min for 3 months or longer

Disease staging based on decrease in GFR:

Normal GFR: 125 mL/min (creatinine clearance)

Last stage of kidney failure: GFR < 15 mL/min
End-stage kidney disease (ESKD)

Stages of Chronic Kidney Disease

Table 1. Stages of CKD[a]

Stage	Description	GFR (mL/min/1.73 m²)
1	Kidney damage with normal or GFR	≥ 90
2	Kidney damage with mild GFR	89-60
3A	Mild to moderate GFR	59-45
3B	Moderate GFR	45-30
4	Severe GFR	30-15
5	Kidney failure	< 15 or dialysis

CKD, chronic kidney disease; GFR, glomerular filtration rate.

[a]Adapted from the Renal Association. http://www.renal.org/whatwedo/InformationResources/CKDeGUIDE/CKDstages.aspx. Accessed November 16, 2013.

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- Recognized later
- Remaining nephrons compensate
- End result- every organ involved
- Prognosis and course – variable
- Loss gradual or severe

Incidence

- 1/9 Americans
- Mortality- 19-24% stage 5 on dialysis

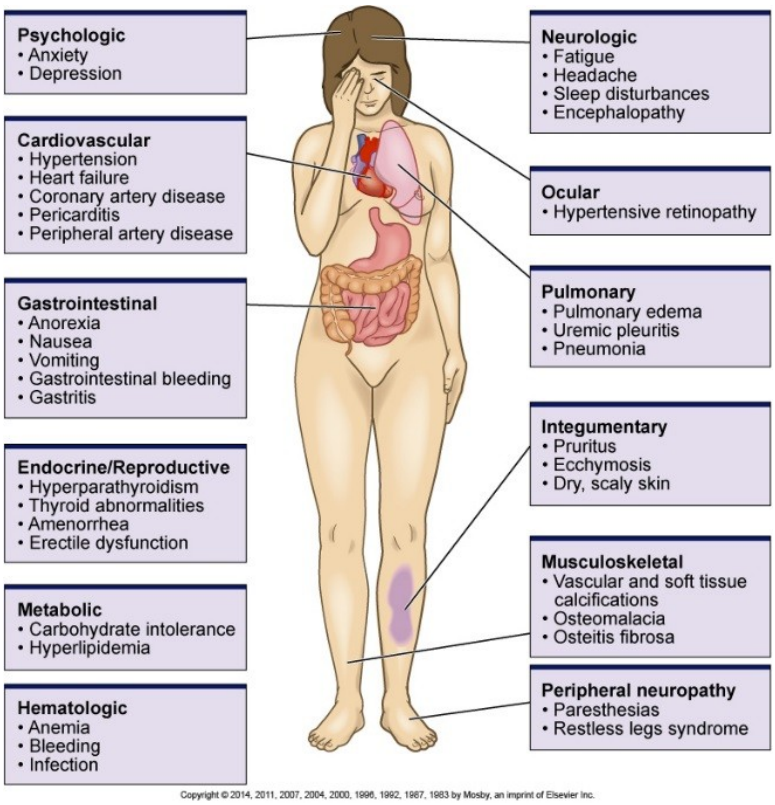
Leading causes of ESKD

- Diabetes
- Hypertension
- Other: polycystic disease, chronic glomerulonephritis, repeat infections, nephrotoxins, SLE, sickle cell, scleroderma, TB, medications - NSAIDs

Clinical Manifestations

Result of retained substances

- Urea
- Creatinine
- Phenols = C6H5OH
- Hormones
- Electrolytes
- Water
- Other substances



Clinical Manifestations

Uremia

- Syndrome - incorporates all s/sx seen in various systems
- Often occurs when the GFR is ≤ 10 mL/min

Urinary System

Polyuria – most noticeable symptom

- Results from inability to concentrate urine
- Occurs at night
- Specific gravity fixed at 1.010

Oliguria

- As CKD worsens

Anuria

- Urine output < 40 mL/24 hrs

As CKD worsens, fluid retention → need for dialysis; anuria

Metabolic Disturbances

Waste product accumulation

-As GFR ↓, BUN and serum creatinine levels ↑
-BUN level ↑ : Kidney failure, protein intake, fever, steroids, catabolism
-s/sx: N/V, lethargy, fatigue, impaired thought processes, and HA
Serum creatinine clearance = GFR; most accurate indicator of function

Altered carbohydrate metabolism

-Caused by impaired glucose metabolism
-Insulin does not affect the cells of glucose like normal; **less sensitive**
-Mild to moderate hyperglycemia and hyperinsulinemia
-*insulin and glucose metabolism may improve with dialysis- may require less insulin*
-Patients with diabetes who develop uremia may require less insulin after onset of CKD
-Excretion of insulin dependent on kidneys – stays in circulation longer, ***change dose for diabetics**

Elevated triglyceride levels (cause atherosclerosis)

-Hyperinsulinemia stimulates hepatic production of triglycerides
-↑ VLDLs, ↑ LDLs, ↓ HDLs
-Altered lipid metabolism:
↓ Levels of enzyme lipoprotein lipase
Important in breakdown of lipoproteins
-**most CKD patients die from CVD!!**

Electrolyte/Acid-Base Imbalances

Potassium

Hyperkalemia

Most serious electrolyte disorder
Fatal dysrhythmias (7 – 8 mEq/L)
Results from: decreased excretion, protein breakdown, metabolic acidosis, food, supplements, IV infusions

Sodium

May be elevated, normal, or low
Sodium is retained ➔Water is retained

- edema
- Hypertension
- HF

If large quantities of water is retained – dilutional hyponatremia
Low sodium diet: 2 g/day

Calcium and phosphate alterations

Hypocalcemia (r/t low vitamin D)
Hyperphosphatemia (r/t bone demineralization & less excretion)

Magnesium alterations

Hypermagnesemia: not a problem unless ingesting more (medications with magnesium)
s/sx: absent reflexes, decreased mental status, cardiac dysrhythmias, hypotension, respiratory

failure

Metabolic acidosis

Results from inability to excrete excess acid
Defective reabsorption/regeneration of bicarbonate
Acid is buffered by bicarb – wnl 80 – 90 mEq of acid/day; bicarb falls to 16-20 mEq/L (indirect measure of acidosis)

Hematologic system

Anemia

-r/t low erythropoietin production
-other: nutritional, decrease RBC lifespan, increased hemolysis of RBC's, frequent blood sampling, bleeding from GI tract
-s/sx: weakness, fatigue, HA, paleness, dizziness, SOB, chest pain, problems concentrating

Bleeding tendencies

Defect in platelets
Caused by impaired aggregation and impaired release of platelet factor III

Infection

- Change in WBC function
- Altered immune response: decreased inflammatory response = r/f infection
- Other factors: hyperglycemia, trauma (catheters, needles, vascular access)
- Impaired phagocytic cell ability

Cardiovascular system:

CVD- most common cause of death in CKD patients
(MI, ischemic heart disease, PAD, HF, cardiomyopathy, CVA)
Risk factors: HTN, elevated lipids, vascular calcification, and arterial stiffness)

Hypertension- most common CV problem- hypervolemia, RAAS

Heart failure- HTN and fluid overload

Left ventricular hypertrophy

Peripheral edema

Dysrhythmias- High K Mag, low Ca, acidosis, low perfusion

Uremic pericarditis- → pericardial effusion and cardiac tamponade (s/sx: friction rub, chest pain, low grade fever)

Respiratory system

Kussmaul respirations – acidosis (remove excess CO_2 by exhalation)

Dyspnea may occur with:

Fluid overload

Pulmonary edema

Uremic pleurisy (predisposing factor for respiratory infections)

Pleural effusions

Respiratory infections → PNA

Gastrointestinal system

Every part is affected r/t excessive urea

Mucosal ulcerations

Stomatitis- exudates, ulcerations, metallic taste

Uremic fetor- urinous odor of breath

GI bleeding- mucosal irritation and platelet defect

Anorexia, nausea, vomiting - if CKD not treated with dialysis

Constipation: ingestion of iron salts and/or calcium-containing phosphate binders (decreased activity and fluid restrictions)

Neurologic system

Expected as renal failure progresses; CNS becomes depressed

Attributed to:

Increased nitrogenous waste products

Electrolyte imbalances

Metabolic acidosis

Atrophy

Demyelination of nerve fibers

-Lethargy, apathy, fatigue
-Restless legs syndrome: s/sx- burning feet, inability to find a comfortable position, gait changes, foot drop, paraplegia)
-Muscle twitching
-Irritability
-Decreased ability to concentrate
-Peripheral neuropathy
-Altered mental ability

-Seizures- ^ BUN
-Coma- ^ BUN
-Dialysis encephalopathy

*Treatment for neurologic problems = dialysis or transplant
*AMS is a **late manifestation** of CKD Stage 5

Musculoskeletal system (renal osteodystrophy)

CKD-MBD (mineral and bone disorder)
Systemic disorder of mineral and bone metabolism

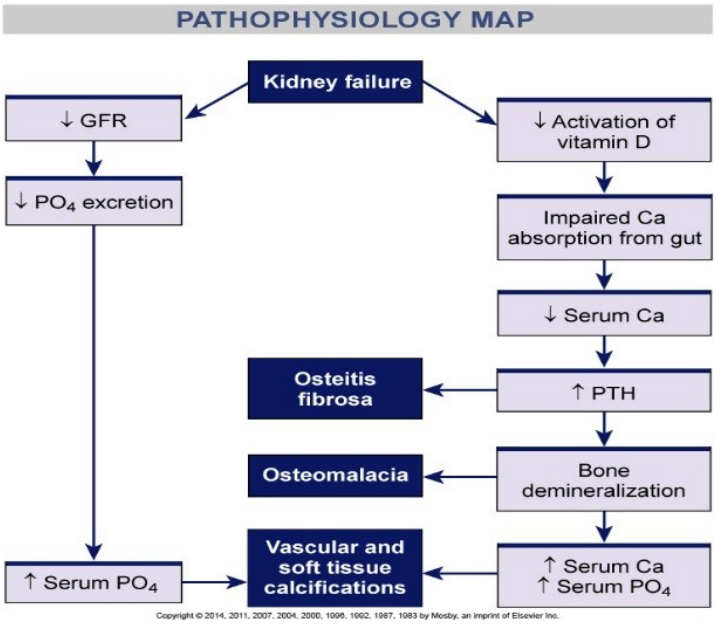
Results in:

Skeletal complications: (osteomalacia, ostetis fibrosa: calcium reabsorption from bone and replacement with fibrous tissue)

Extraskeletal: vascular calcifications

*as renal function deteriorates, less vitamin D = decreased calcium absorption; low Ca levels = release of PTH which demineralizes bone to release Ca; phosphate released too = hyperphosphatemia – also from reduced excretion by kidneys. Hyperphosphatemia results in further hypocalcemia & vitamin D activation

Mechanisms of CKD-MBD



* Blood vessels, joints, lungs, heart, and eyes **(uremic red eye- calcium deposits in eye)**

Integumentary system

Absorption and retention of urinary chromagens
Pruritus: multifactorial (dry skin, calcium-phosphate in skin, sensory neuropathy)

Uremic frost: rare; urea crystallizes on skin and seen with BUN levels are extremely elevated > 200 mg/dL

Dry brittle hair, thin nails, petechiae, ecchymosis

Reproductive system

Infertility

Decreased libido

Low sperm counts

Sexual dysfunction: ? anemia, may improve with dialysis and normalize with transplant

- female: low estrogen, progesterone and LH ➔ anovulation and usually amenorrhea
- male: loss of testicular consistency, low testosterone, low sperm counts, ED

Psychologic changes

Personality and behavioral changes – can be dramatic depending on underlying personality

Emotional lability

Withdrawal & depression

Change in body image can contribute to depression, etc.

Life threatening disease = extreme stress

Diagnostic Studies

History and physical

Dipstick evaluation (persistent proteinuria: 1+ protein two or more times over a 3 month period should be worked up further; usually first indicator of damage)

UA – RBC, WBC, casts, protein, and glucose

Albumin-to-creatinine ratio – first morning void

GFR - use MDRD study to calculate

Renal ultrasonography – detect obstructions and size of kidneys

Renal scan – establish diagnosis and cause

CT scan – establish diagnosis and cause

Renal biopsy – may need for definitive diagnosis

Conservative therapy:
detect and tx cause, preserve function, reduce CV risk, prevent complications, comfort

-Correction of extracellular fluid volume
-Nutritional therapy
-Erythropoietin therapy - epogen
-Calcium supplementation, phosphate binders
-Antihypertensive ’s
-Lower potassium

Drug therapy

Hyperkalemia

IV insulin – pushes K into cells; given with glucose; eventually diffuses back into blood

IV glucose – prevents hypoglycemia

IV 10% calcium gluconate – raises threshold

Sodium bicarbonate – corrects acidosis

Sodium polystyrene sulfonate (Kayexalate)

Cation-exchange resin

Resin in bowel exchanges potassium for sodium

S/fx: diarrhea; never give to paralytic ileus = bowel necrosis

Hypertension – target 130/80 CKD, and 125/75 for pts with significant proteinuria

Weight loss
Lifestyle changes – alcohol, smoking cessation, exercise
Diet recommendations – DASH
Sodium and fluid restriction
Antihypertensive drugs: dependent on whether diabetic or non-diabetic

(ACE & ARBs given to diabetic and those with non-diabetic proteinuria because they decrease proteinuria and delay progression)

Digoxin – cardiac output

Diuretics – furosemide

CCB – nifedipine, amlodipine, diltiazem

ACE inhibitors – captopril, lisinopril

ARB agents – irbesartan, losartan, valsartan

*** ACE & ARBs ↓ GFR and ↑ K+ (CAUTION)

CKD-MBD – limit dietary phosphorus, admin phosphate binders, supplement vitamin D, control hyperparathyroidism

Phosphate not restricted until requires renal replacement therapy

Phosphate intake restricted to < 1000 mg/day

Phosphate binders: **admin with meals**

Calcium acetate (PhosLo)

Calcium carbonate (Caltrate) – bind phosphate in bowel and in feces

Sevelamer hydrochloride (Renagel) – lowers cholesterol and LDL levels

Side effect: constipation

Supplementing vitamin D

Calcitriol (Rocaltrol) doxercalciferol/Hectorol, paricalcitol/Zemplar

Serum phosphate must be lowered before calcium or vitamin D administered

Controlling secondary hyperparathyroidism

Calcimimetic agents: increase sensitivity to calcium receptors in parathyroid glands

Cinacalcet (Sensipar)

Subtotal parathyroidectomy: only if it becomes severe; parathyroid tissue auto-transplanted into forearm and if PTH becomes excessive, remove cells from forearm

Anemia

Erythropoietin

Epoetin alfa (Epogen, Procrit)
Darbepoeitin alfa (Aranesp) – weekly or every 2 weeks
IV or subQ
Increased hemoglobin and hematocrit in 2 - 3 weeks
Side effect: HTN, iron deficiency
Use lowest dose first to reduce r/f CV events

Iron supplements

If plasma ferritin level is less than 100ng/mL
Side effects: GI irritation, constipation, dark stool
Do not administer PO iron with phosphate binders

Folic acid supplements (RBC formation)

Goal- avoid blood transfusions (unless acute loss or symptomatic)
Transfusions suppress erythropoiesis
Transfusions increase amount of antibodies developed ➔ difficult to cross-match for transplant

Dyslipidemia

Goal

Lowering LDL level below 100 mg/dL
Lowering triglyceride level below 200 mg/dL

Statins

HMG-CoA reductase inhibitors – most effective for lowering LDL

Simvastatin ^ r/f myopathy, if developed switch to atorvastatin

Fibrates – gemfibrozil/Lopid lower triglycerides and can increase HDL levels

Complications

Drug toxicity: delayed and decreased elimination = accumulation; adjust dosages and frequencies
- Digoxin – excreted by kidneys
- Diabetic agents – metformin
- Antibiotics – many nephrotoxic
- Pain medication (meperidine/Demerol- seizures, NSAIDs)

Nutritional therapy

Calorie-protein malnutrition

Dietary guidelines for HD & PD differ (PD – excessive protein loss)

Protein restriction – not routinely restricted for dialysis
- Benefits are being studied
- Monitor labs: serum albumin, prealbumin, ferritin, and BMI = nutritional status

Water restriction
- Intake depends on daily UO
- Fluid = anything liquid at room temperature
- Insensible loss + previous day's output = daily allowance- hemodialysis

Sodium restriction
- Diets vary from 2 – 4 g, depending on edema & HTN
- Sodium and salt should not be equated (1g NaCL = 400 mg Na)
- Salt substitutes – do not use (potassium chloride)
- Avoid high sodium foods!

Potassium restriction
- Limit: 2 – 3 g
- Avoid: oranges, melons, bananas, tomatoes, beans, legumes, green/yellow veggies

Phosphate restriction
- Limit: 1000 mg/day
- Most foods high in phosphate are high in protein – phosphate binders, high protein diet
- Dairy products

Nursing Management: Nursing Assessment
- Complete history – any existing renal disease, family history
- Long-term health problems
- Diet habits
- Current & past prescription medications, OTC, herbal remedies (antacids, NSAIDs, decongestants, antihistamines)

Nursing Management: Nursing Diagnoses
- Excess fluid volume
- Risk for electrolyte imbalance
- Risk for injury
- Imbalanced nutrition: less than body requirements

Nursing Management: Nursing Implementation

Health promotion

Identify individuals at risk for CKD

- History of renal disease
- Hypertension
- Diabetes mellitus
- Repeated urinary tract infection

Urinary appearance, frequency, and volume

DM – urine checked for microalbuminuria if negative for protein

Reduce risk for CV disease

Acute intervention

Daily weight (1 lb wt gain = 500 mL retained fluid)

Daily BP

Identify signs and symptoms of fluid overload

Identify signs and symptoms of hyperkalemia

Strict diet adherence

Ambulatory and home care

HD, PD, and transplantation

Patient and family need clear explanation of dialysis and transplantation

Acute intervention

Medication

Motivate management