| **Disease** | **Clinical findings** | | **Urinalysis** | **Investigations** | **Treatment** |
| --- | --- | --- | --- | --- | --- |
| **GROSS HEMATURIA** | | | | | |
| **Non-blood differential** | Heme positive – hemoglobin or myoglobin  -myoglobinuria secondary to rhabdo  -acute or chronic hemolysis | | | Heme negative  -drugs (ibuprofen, flagyl, rifampin, Macrobid, salicylates, sulfasalazine, deferoxamine)  -food (blackberries, beets)  -dyes  -urine metabolites (homogentisic acid, melanin, methemoglobin, porphyrin, tyrosinosis, urates) | |
| **Postinfectious glomerulonephritis** | Antecedent infection – usually 1-3weeks post  Hypertension  Edema | | Hematuria  Proteinuria  Coke/tea colour  RBC casts | ASOT  C3 LOW  Cutaneous strep – antideoxyribonuclease B level  Positive streptozyme  Strep throat swab  Renal biopsy only in acute renal failure/nephrotic | Usually self-limited  Complement normal in 6-8weeks, microscopic hematuria for 6-12m  Can tx with systemic abx  Complications: HTN, PRES |
| **Membranoproliferative glomerulonephritis** | Found in older children/adults  F>M | | Hematuria  Proteinuria | C3 LOW  Renal biopsy |  |
| **Hemolytic uremic syndrome**  Microangiopathic hemolytic anemia, thrombocytopenia and renal insufficiency  Toxins directly cause endothelial cell damage, activate platelets, localized thrombosis, consumptive thrombocytopenia, mechanical damage to RBCs passing through damaged/thrombotic vasculature | E.coli (STEC) O157:H7 (undercooked meat, unpasteurized milk and apple cider)  Shigella dysenteriae  Strep pneumoniae – starts with pneumoniae with empyema  Atypical – genetic (ADAMST13)  Gastroenteritis (often bloody diarrhea), abdominal pain, fever, pallor, weakness, oliguria  If strep – pneumoniae, empyema, bacteremia | | Microscopic hematuria  Low-grade proteinuria | Hemolytic anemia with schistocytes  Thrombocytopenia  Leukocytosis  Creatinine elevation  INR/PTT normal  Coombs negative (except in Pneumococcal)  Renal failure + hemolysis – life-threatening hyperkalemia  Rarely need biopsy | Complications:  CNS – irritability, lethargy, encephalopathy, seizures, ischemic  CVS – arrhythmias, HTN  GI – inflammatory colitis, perforation, intussusception, pancreatitis  GU – oliguric or anuric renal failure, volume overload  Heme – anemia, petechiae, severe bleeding rare  Treatment:  50% require dialysis, 30% left with chronic renal insufficiency  Worse prognosis with non-diarrheal  Fluid management – correct volume deficit, control hypertension, dialysis for oliguria  pRBC transfusion – washed if pneumococcal  NO platelets – consumed  NO abx – increased toxin release (unless pneumococcal)  Annual follow up with primary care |
| **Henoch-Schlonlein Purpura (HSP)**  Small vessel vasculitis  **Diagnosis**: palpable purpura with at least one of: abdominal pain (75%), IgA deposition on biopsy specimen, arthritis/arthralgia (80%), renal involvement (30-50%) (hematuria/proteinuria) | Usually follows URTI, can be related to GAS  Palpable purpura in pressure-dependent areas  Edema  Abdominal pain – colicky, bloody stools, bowel edema  Intussusception  Arthritis/arthralgia – large joints of lower extremities, migratory  Renal findings 1-6m after initial presentation – microscopic hematuria to crescentic GN to ESRD  Rare: CNS encephalopathy or seizures, scrotal involvement | | Hematuria  Proteinuria | No specific findings to HSP  CBC – may have leukocytosis  Serum IgA elevated in half  Normal: ANA, dsDNA, ANCA  NORMAL complement  IgA deposition in glomerulus, skin and blood vessels of GI tract | Usually self-limited in 4-6weeks, 1/3 relapse within 1y  Treatment supportive  NSAIDs for joint pain  Corticosteroids for abdominal complications  Immunosuppression for renal involvement (cyclophosphamide, calcineurin inhibitors – cyclosporin, tacrolimus, cell cycle inhibitors – MMF)  IVIG, PLEX or transplant  Monitor for GI complications (intussusception, ischemia, necrosis, perforation)  Monitor for GU complications – major morbidity, especially if proteinuria present initially |
| **IgA nephropathy (Berger disease)**  **GN with illness** | Recurrent gross hematuria with illness/exercise  Adolescence  Recent URTI (2-3d post – contrast from post-strep GN)  Diagnosis requires renal biopsy | | Hematuria  Proteinuria | NORMAL complement  No need to do IgA level | Uncommon for ESRD in childhood (differs from adults) but need long-term followup BP and proteinuria control - ACEi |
| **Alport syndrome**  **GN with illness PLUS systemic features (SNHL and anterior lenticonus)**  Mutation in type IV collagen of glomerular basement membrane  X-linked in 85% | Sensorineural hearing loss  Anterior lenticonus – pathognomonic  Intermittent episodes of hematuria  Diagnosis: clinical features, skin biopsy, genetics testing | | Hematuria  Progressively worse proteinuria | Progressive sclerosis | Chance of ESRD most common in X-linked or AR cases |
| **Renal vein thrombosis**  Starts in intrarenal venous circulation, extends to renal vein and to IVC  Endothelial cell injury from hypoxia, endotoxin or contrast media  Newborns/infants – asphyxia, dehydration, shock, sepsis, congenital hypercoagulable states, maternal diabetes  Older children – nephrotic syndrome, cyanotic heart disease, inherited hypercoagulable states, sepsis, post-renal transplant, post angiographic contrast agent exposure | Sudden onset gross hematuria  Unilateral or bilateral flank masses  Any combo of: microscopic hematuria, flank pain, HTN, microangiopathic hemolytic anemia with thrombocytopenia or oliguria  DDX – other causes of hematuria that have rapid development of microangiopathic hemolytic anemia or enlargement of kidney  -HUS  -hydronephrosis  -PCKD  -Wilms tumour  -intrarenal abscess or hematoma | | Hematuria | RUS + Doppler – to confirm  Radionuclide studies – little to no function in affected kidney  AVOID contrast  Evaluate for coagulability | Correction of fluids and electrolytes  TPA and unfractionated heparin followed by continued anticoagulation with unfractionated or LMWH  Antihypertensives – but if refractory, may need nephrectomy  Prognosis: risk of renal insufficiency, renal tubular dysfunction and HTN |
| **Sickle cell disease/trait**  Occlusion of vasa recta capillaries causing renal papillary infarcts |  | |  |  |  |
| **SLE nephritis** |  | | Hematuria  Proteinuria | LOW complement (C3 AND C4) |  |
| **Painless gross hematuria with trauma** |  | |  | Ultrasound – ureteropelvic junction obstruction |  |
| **SYMPTOMATIC MICROSCOPIC HEMATURIA** | | | | | |
| Nonspecific – fever, malaise, weight change  Extrarenal – malar rash, purpura, arthralgia/arthritis, headaches  Localized with urinary tract symptoms – dysuria, suprapubic pain, flank pain, edema, oliguria | | | e.g. malar rash, arthritis, pericardial rub, edema and HTN – likely SLE  e.g. fever, flank pain, N/V – upper urinary tract involvement  e.g. dysuria, frequency, urgency, incontinence – crystalluria or UTI | | |
| **ASYMPTOMATIC (ISOLATED) HEMATURIA -** Rarely have significant renal disease (25% normalized within 5y) | | | | | |
| **Benign familial hematuria (thin basement membrane disorder)**  Positive family history – AD  Can be sporadic | No long term complications as in Alports (renal, ocular, hearing) | | YES  No proteinuria | Biopsy – diffuse thinning of glomerular basement membrane | Monitor for development of HTN or proteinuria |
| **Hypercalciuria** | Associated with: immobilization, diuretics, vitamin D intoxication, hyperparathyroidism, sarcoidosis | |  | Urinary calcium-creatinine ratio of >0.2  24h urinary calcium >4mg/kg/d | Risk of urolithiasis |
| **ASYMPTOMATIC HEMATURIA AND PROTEINURIA** – combo concerning for serious renal disease. First confirm if proteinuria is orthostatic with first morning urine protein (normal protein to creatinine ratio <0.2) | | | | | |
| Renal biopsy – recurrent episodes of gross hematuria, coexisting nephrotic syndrome, coexisting hypertension with nephritic component, renal insufficiency, family history suggesting hereditary nephritis, coexisting systemic symptoms | | | | | |
| **NEPHROTIC SYNDROME** – proteinuria, hypoalbuminemia, edema and hyperlipidemia  Glomerular basement membrane found between fenestrated endothelium and epithelial podocyte/foot process layer  Nephrotic syndrome – effacement of podocyte foot processes leading to proteinuria  Primary (idiopathic) vs. secondary (genetic), congenital nephrotic syndrome, infantile nephrotic syndrome  Idiopathic nephrotic syndromes – minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy | | | | | |
| **PROTEINURIA** | | | | | |
| **Transient proteinuria** | Contributing factors:  -temp >38.3, exercise, dehydration, cold exposure, heart failure, seizures or stress | | Not greater than 2+ |  |  |
| **Orthostatic proteinuria**  Most common cause of persistent proteinuria in school-age children and adolescents | When upright, urinary protein excretion increased 10x (up to 1g/24h) with NO other findings | | No hematuria | First morning urinalysis and protein/creatinine ratio <0.2 on 3 consecutive days  If >0.2 = fixed proteinuria = needs evaluation | Monitor for nonorthostatic proteinuria |
| **Fixed proteinuria** | Glomerular proteinuria – urine protein:creatinine ratio >1 with HTN, hematuria, edema or renal dysfunction  If urine protein:creatinine ratio 0.2-1, reevaluate q4-6m unless symptomatic | | | | |
| **IDIOPATHIC NEPHROTIC SYNDROME** | | | | | |
| **General**  Hypoalbuminemia  Edema  Hyperlipidemia | Sudden onset gravity dependent edema – either from decreased oncotic pressure or primary sodium retention  Complications:  -thrombosis (venous, combo of hereditary risk factor, intravascular depletion, urinary loss of coagulation cascade regulators, increase in hepatic procoagulants)  -infections (loss of immunoglobulins, increased risk encapsulated, e.g. peritonitis from Strep pneumoniae)  -dyslipidemia  -renal dysfunction  -loss of vitamin D and thyroid binding proteins (risk of vitamin D deficiency and metabolic bone disease, hypothyroidism) | | Proteinuria >50mg/kg/d (3.5g/24h) or spot urine protein:creatinine ratio > 2  Hematuria | Hypoalbuminemia  Hyperlipidemia (decreased oncotic pressure and increased activity of other enzymes)  Electrolytes usually normal, Ca low from hypoalbuminemia  Can have hyponatremia (low effective circulating volume and SIADH)  Consider autoimmune or infectious workup  Biopsy  NORMAL complement | Corticosteroids after biopsy  -prednisone 2mg/kg/d x 4-6w  -1.5mg/kg/d qotherday x 2-5m with tapering  Natural course – relapse and remitting  Diuretics if edema  Monitor for dyslipidemia  Monitor for infections  23-valent pneumococcal vaccine after 2y  Corticosteroid resistant (usually not minimal change) – high chance progression to ESRD (dialysis or transplant)  -can still have recurrence in transplanted kidney in FSGS  -ACEi or ARBs |
| **Minimal change disease** | Most common in school-aged children | | Proteinuria  Hematuria | Light microscopy – normal glomeruli  Electron microscopy – fusion of foot processes | Good prognosis, uncommon to have renal failure |
| **Focal segmental glomerulosclerosis**  **Diagnosis:**  Biopsy, may require a second to ensure haven’t dx minimal change by accident |  | | Proteinuria  Less hematuria compared to others | Histology – some glomeruli normal, others segmental sclerosis/scarring |  |
| **Membranous nephropathy**  ?autoimmune |  | | Proteinuria  Hematuria | Histology – diffuse thickening of capillary walls |  |
| **SECONDARY NEPHROTIC SYNDROME** | | | | | |
| **Infectious** | Hepatitis B or C; HIV; Toxoplasmosis; Syphilis; Malaria | | | | |
| **Disease** | Amyloidosis; Lupus; HSP; Lymphoma; IgA nephropathy; MPGN, hereditary | | | | |
| **Medications/Drugs** | Lithium; NSAIDs; Penicillamine; Gold; Interferon gamma; Pamidronate; Heroin | | | | |
| **PULMONARY RENAL SYNDROMES** | | | | | |
| **Granulomatosis with polyangiitis (Wegeners)**  Granulomatous necrotizing inflammation of small and medium vessels | Glomerulonephritis  General – fever, loss of energy, vague joint complaints  Nasal – ulceration, septal perforation, pain, sinusitis, epistaxis  Pulm – cough, hemoptysis, dyspnea, chest pain, infiltrates on CXR, pulmonary hemorrhage | |  | ANCA positive – PR3  Biopsy lung – granulomas with vasculitis  Renal biopsy – rarely demonstrates granulomas or vasculitis (pauci-immune)  HRCT for lung imaging  Elevated ESR/CRP | Steroids  Cyclophosphamide  During remission – methotrexate or azathioprine  PLEX during acute to remove ANCAs  Prophylaxis with Septra for PJP |
| **Microscopic polyangiitis**  Small vessel necrotizing vasculitis | Glomerulonephritis with little immune complex deposition  NO granulomatous inflammation  Similar presentation to GPA but no sinus involvement \, predominant systemic features | |  | ANCA positive – MPO  Elevated ESR/CRP | Same as GPA |
| **Eosinophilic granulomatosis (Churg-Strauss syndrome)**  Small vessel necrotizing allergic granulomatous vasculitis | Refractory asthma and peripheral eosinophilia  Granulomatous inflammation  Rare to have cartilage destruction  Uncommon renal involvement | |  | ANCA positive  Biopsies with eosinophilic infiltrate  Elevated ESR/CRP |  |
| **Goodpasture syndrome (anti-glomerular/alveolar basement membrane antibody)** | Pulmonary hemorrhage and crescentic glomerulonephritis  Hypertension  Renal failure in days-weeks | | Hematuria  Proteinuria | NORMAL complement  Serum anti-GBM present  ANCA high | Poor prognosis untreated  Treat with high-dose IV methylpred, cyclophosphamide and plasmapheresis  Often progress to ESRD despite therapy |
| **ACUTE KIDNEY INJURY**  Term neonates – all nephrons but only 25% of adult function, not able to concentrate their urine; mature GFR by 2y  Renal blood flow controlled by afferent and efferent arterioles, NaCl sensing by juxtaglomerular apparatus  Decreased renal perfusion – afferent vasodilation secondary to prostaglandins, nitric oxide and bradykinins; efferent vasoconstriction by SNS, endothelin and activation of RAS and production of angiotensin II 🡪 aldosterone 🡪 increase Na (distal tubule) and H2O absorption to increase extracellular volume; ADH 🡪 reabsorption of urea and water | | | | | |
| **AKI**  Acute decrease in GFR resulting in increased Cr | Elevated creatinine & urea (creatinine can be delayed by 48h)  Urine sodium, urea, creatinine, urinalysis  RBUS – larger kidneys = acute process with inflammation; small = chronic scarring; hydronephrosis suggesting obstruction | Prevention: hydration, minimizing nephrotoxic drugs  Management:  FLUIDS:  -NS boluses or pressors  -trial of diuretics if oliguric  -restriction of fluid to insensibles (300-500mL/m^2/d)  ELECTROLYTES:  -manage Na  -hold K and PO4 in regular fluids but monitor  -hyperkalemia – fatigue, weakness, tingling, nausea, paralysis, cardiac conduction abnormalities (peaked T, wide QTS, flat P waves, prolonged PR)  -if stable, trial potassium binder or Lasix dose  -if unstable or >7 – calcium gluconate, sodium bicarb, beta-2 agonists, insulin and glucose  -acidosis – elevated AG (kidneys can’t excrete H or reabsorb HCO3) – no bicarb as will lower calcium = tetany  MEDICATIONS: avoid nephrotoxic or dose adjust  NUTRITION: catabolic state, need to ensure adequate calories and protein intake (don’t restrict protein to avoid increasing urea)  RRT indications: volume overload of 10-20%, severe acidosis, hyperkalemia, uremia, symptomatic, or difficulty providing nutrition  LONG TERM: at risk of CKD  -yearly HTN and urinalysis | | | |
| KDIGO used for staging | | | | | |
| **Prerenal AKI**  Decrease in renal blood flow leading to hypoperfusion (decreased effective circulating volume, loss of vascular tone, decreased cardiac output, redistribution of fluid from decreased oncotic pressure or capillary leak) | NSAIDs worsen AKI as they decrease prostaglandins and prevent afferent vasodilation  ACEi prevent angiotensin from vasoconstricting efferent arterioles  RAS and ADH – increased sodium and urea reabsorption  At risk patients – neonates, sickle cell | |  | Normal U/A  Concentrated urine osm >500  FENa <1  FEUrea <35%  Urine sodium <20 |  |
| **Intrinsic AKI**  Direct renal parenchymal damage or dysfunction  Most common in hospitals from conversion of prerenal AKI to ATN | **Tubular** – acute tubular necrosis. Damage from hypoperfusion leads to cellular necrosis and debris build-up and blockage of tubular flow. Manifestation during recovery  **Interstitial** – after exposure to offending agent (antibiotics, PPIs, NSAIDs, diuretics) or nephrotoxic exposure (chemotherapy agents, calcineurin inhibitors, radiocontrast)  **Glomerular** – glomerulonephritis, systemic disease  **Vascular** – microangiopathic processes (HUS, TTP), systemic vasculitides | |  | Loss of ability to concentrate urine  Muddy granular casts = ATN  Red cell casts = GN |  |
| **Postrenal AKI**  Obstructive processes that block urine flow | Bilateral ureteral obstruction by tumor, renal calculi, clots in bladder | |  |  |  |
| **CHRONIC KIDNEY DISEASE** | | | | | |
| **CKD**  Younger patients: structural anomalies  Older patients: glomerular diseases  33% GN  25% VUR/obstruction/  infections  16% hereditary nephropathies  11% hypoplasia/  dysplasia  5% vascular  Lifespan shortened by 50y in those with ESRD  With transplant still shortened by 25y | **Diagnosis:**  1. Kidney damage for 3m or longer by structural or functional abnormalities – either pathologic or markers of kidney damage (blood, urine or imaging changes)  2. GFR <60 for 3m or longer  **Classification:**  1. Kidney damage, normal GFR  2. Mild reduction, GFR 60-89  3. Moderate, GFR 30-59  4. Severe, GFR 15-29  5. Failure, GFR <15 | | Comorbidities:  CVS: HTN, dyslipidemia, obesity, LVH  Metabolic: electrolyte disturbances, metabolic bone disease, anemia  Nutrition: anorexia, malnutrition  Growth: decreased linear growth  Neurocognitive: lower IQ, impaired memory, sleep problems  Disease burden: QOL, depression  Immunosuppression  Reproduction: impaired fertility | Management:  ACEi – blood pressure control and early decreases in proteinuria slowed progression of CKD  Immunizations – including 23-pneumococcal, avoid live vaccines in those on immunosuppressants  CVS: hypertension, dyslipidemia and glucose metabolism – control HTN, lipids, anemia  MBD: retention of PO4 and inability to make active 1,25-OH2 D 🡪 stimulates parathyroids 🡪 secondary hyperparathyroidism; supplement Vit D, restrict PO4  Anemia: epo and iron supps  Nutrition/Growth: involve dietitians, may require feeding tubes, will help optimize growth  Mental health: screen for depression, anxiety, ADHD  Renal: dialysis, transplant | |
| **RENAL TUBULAR ACIDOSIS** | | | | | |
| **RTA**  Normal anion gap (hyperchloremic) metabolic acidosis with normal GFR | Normal function:  Excretion of H+ (proximal tubule and collecting tubule) in exchange for HCO3- (90% proximal tubule) | |  | Confirm normal anion gap metabolic acidosis, electrolyte abnormalities, rule out other reasons for acidosis (diarrhea)  RBUS – structural  Type IV – hyperkalemic metabolic acidosis  Urine pH - <5.5 = proximal, >6.0 = distal  Glycosuria, proteinuria, hematuria = global dysfunction Ca – hypercalciuria | Bicarb replacement (much higher requirements in proximal vs distal)  Phosphate replacement (Fanconi’s)  Monitor for nephrolithiasis in distal – may require thiazide diuretics to decrease Ca excretion  Hyperkalemia – Kayexalate |
| **Proximal (Type II) Renal Tubular Acidosis**  Inability to resorb bicarb  **Fanconi syndrome** | Present with growth failure in 1st year  Polyuria, dehydration, anorexia, vomiting, constipation, hypotonia, rickets | | Others: Cystinosis, galactosemia, tyrosinemia, Wilson disease, hereditary fructose intolerance, Lowe syndrome | Non anion gap metabolic acidosis  Urine pH <5.5 (distal mechanisms intact)  Low molecular weight proteinuria, glycosuria, phosphaturia, aminoaciduria |  |
| **Cystinosis – type 2 RTA** | Polyuria, polydipsia, growth failure, rickets, ocular (photophobia, retinopathy, poor visual acuity), hypothyroidism, hepatosplenomegaly, delayed sexual maturation, fair features (decreased pigmentation) | |  | Cystine crystals in cornea  Leukocyte cystine content | Correct metabolic abnormalities  Cysteamine PO and eye drops  Kidney transplant for renal failure |
| **Lowe syndrome – type 2 RTA**  X linked | Congenital cataracts, mental retardation, Fanconi syndrome  Renal – nonspecific tubulointerstitial changes, thickening of glomerular basement membrane | | Proteinuria |  |  |
| **Distal (Type 1) RTA**  Impaired functioning of transports/proteins in acidification process  e.g. medullary sponge kidney, Sjogren’s syndrome, Wilson disease, primary biliary cirrhosis, lymphocytic thyroiditis | Loss of bicarb, K, Ca, citrate  Nephrolithiasis from hypercalciuria (differentiates from pRTA)  Bone disease from mobilization of bone stores to compensate for acidosis  Growth failure | |  | Non anion gap metabolic acidosis |  |
| **Hyperkalemic (Type IV) RTA**  Impaired aldosterone production or impaired renal responsiveness (pseudohypoaldosteronism)  Can also happen from obstructive uropathies | Aldo affects H/ATPase responsible for H secretion  Aldo stimulates K secretion therefore get hyperkalemia, worsens H secretion  Growth failure, polyuria, polydipsia, hyperkalemia | |  | Elevated urinary sodium  Decreased urinary potassium |  |
| **HYPERTENSION** | | | | | |
| Prevalence 5-20%  Lifestyle: physical inactivity, increased caloric intake, high salt intake, obesity  Screening: any child >3 should have BP measured with appropriate cuff by manual method (if automatic cuff used and concern for HTN need to repeat with manual)  -children <3 – premature or VLBW, CHD, renal/urologic malformations, solid-organ transplant, malignancy/BMT, meds that raise BP, systemic illness with known HTN | Definition: BP over 90th%  Prehypertension – between 90-95th%, or >120/80 in adolescent  Hypertension - >95th%  Stage 1 – 95-99th% + 5  Stage 2 – >99th%  **Diagnosis:**  3 or more separate office visits  Nonspecific – sleep disturbance, daytime fatigue, inattention, headache, SOB  Renal – hematuria, edema, polyuria, nocturia  Endo – weight loss, tremors, excessive sweating  Consider pmhx prematurity, CHD, recurrent UTIs, FHx | | Most common etiologies:  -renal parenchymal disease  -renal vascular disease (neurofibromatosis, Williams, Wilms tumour, thrombosis or stenosis)  -endo – rare but treatable  -pheos – rare  -iatrogenic – OCP, steroids  -coarct  A screenshot of a cell phone  Description automatically generated | Before diagnosis:  -ambulatory BP measurement  With diagnosis:  -CBC, U/A, creatinine, urea, lytes, fasting lipids, fasting glucose  -RBUS +/- Doppler  -renin (if high, consider renovascular disease)  If endo: -thyroid, aldosterone, steroid levels, urine metanephrines and plasma catecholamines  End-organ dysfunction:  -echo – LVH, q6m if +  -ophtho referral  -albumin:creatinine ratio  Prehypertension – q6m  Stage 1 – q3-4m  Stage 2 – q2weeks initially then q3-4m | NONPHARM:  Diet: dietitian, reduce sodium to 2-3g/d, reduce cholesterol, reduce sweetened drinks, limit portion sizes, avoid skipping meals  Physical activity: 60min daily, reduce sedentary to 2h/d  PHARM:  -start immediately with confirmed HTN and end-organ changes, diabetes, CKD or stage 2 HTN  -goal to lower <90th%  -see below for meds  -antihypertensives  -diuretics (first line in adults, not in peds)  -central alpha-agonists (clonidine – limited by adverse effects – dry mouth, sedation, fatigue, severe rebound HTN with discontinuation  -vasodilators (hydralazine) for acute setting |
|  | | | | | |
| ACEi:  Captopril (multiple doses in a day)  Enalapril  Lisinopril  Ramipril | -block angiotensin 1 to 2 and degradation of vasoD bradykinin, cardio and renal protective  -s/e dry cough  -monitor CBC, lytes, Cr, urea q3-6m  -contraindicated – bilateral RAS, hyperK, pregnancy | | | | |
| ARBs:  Losartan  Irbesartan | -blocks angII binding, no kinin activity (no cough) | | | | |
| CCBs:  Dihydropyridines – nifedipine, isradipine, amlodipine  NonDHP – verapamil, diltiazem (not used as much in peds) | -block influx of calcium into smooth muscles = dilation and decreased resistance  -amlodipine s/e – edema, flushing, headache, gingival hyperplasia, orthostatic hypotension | | | | |
| Beta-blockers  Cardioselective (B1)  Nonselective (B1,2)  Propranolol  Atenolol  Metoprolol  Labetalol (also alpha activity) | Inhibition of renin secretion  Reduction in peripheral resistance  Lowering cardiac output  Decreasing plasma volume  Contraindication – athletes (decrease cardiac output), asthma (potential bronchospasm), DM (mask symptoms of hypoglycemia)  Adverse effects – orthostatic hypotension, fatigue, depression, altered lipid profiles, impotence, hyperkalemia | | | | |
| **Hypertensive Emergency**  CNS: retinopathy, encephalopathy, seizures, hemiplegia, facial palsy  CVS: tachypnea, pulmonary edema, murmur  Renal: peripheral edema, gross hematuria, change in u/o, abdo bruit  Endo: exophthalmos, tremors, hair loss  Abdo mass: Wilms, neuroblastoma, hydronephrosis, PCKD  Skin: NF-1, TS | **Diagnosis:**  BP > stage 2 cutoff with life-threatening symptoms or end-organ dysfunction  Normally autoregulation keeps BP within range, but once outside limits, results in endothelial dysfunction, vessel wall edema, and CNS/cardiac/renal complications | |  | CBC, lytes, creatinine, urea, U/A, beta-HCG  CXR – heart failure  Echo – heart failure  Tox screen  RBUS  CT head if CNS symptoms | ICU for monitoring  Lower by 25% in first 8h, then normalizing over 24-48h  Infusions: nicardipine, labetalol, nitroprusside  PRNs: clonidine, hydralazine |
|  |  | |  |  |  |

