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	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,	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

Disease	Clinical findings	Urinalysis	Investigations	Treatment
passing through damaged/thrombotic vasculature	fever, pallor, weakness, oliguria If strep – pneumoniae, empyema, bacteremia			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	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,

Disease	Clinical findings	Urinalysis	Investigations	Treatment
	Renal findings 1-6m after initial presentation – microscopic hematuria to crescentic GN to ESRD Rare: CNS encephalopathy or seizures, scrotal involvement			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	Sudden onset gross hematuria Unilateral or bilateral flank masses Any combo of: microscopic hematuria, flank pain, HTN, microangiopathic hemolytic	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

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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	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			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				

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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)				

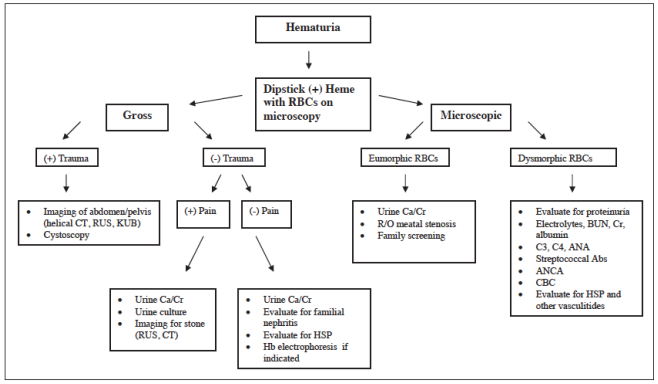


Figure 3. Algorithm for evaluation of hematuria. ANA=antinuclear antibody, ANCA=antineutrophil cytoplasmic antibody, Abs=antibodies, BUN=blood urea nitrogen, C=complement, Ca=calcium, CBC=complete blood count, Cr=creatinine, CT=computed tomography scan, HSP=Henoch-Schönlein purpura, Hb=hemoglobin, KUB=kidney-ureter-bladder radiograph, RBC=red blood cell, R/O=rule out, RUS=renal ultrasonography

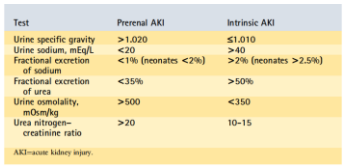
Renal biopsy – recurrent episodes of gross hematuria, coexisting nephrotic syndrome, coexisting hypertension with nephritic component, renal insufficiency, family history suggesting hereditary nephritis, coexisting systemic symptoms

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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)	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)	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

Disease	Clinical findings	Urinalysis	Investigations	Treatment
	-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)		Consider autoimmune or infectious workup Biopsy NORMAL complement	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			

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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

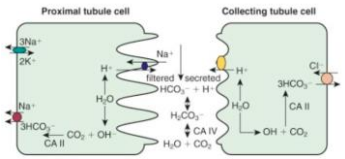
Disease	Clinical findings	Urinalysis	Investigations	Treatment
				Often progress to ESRD despite therapy
ACUTE KIDNEY INJURY Term neonates – all nephrons but only 25% of adult function, not able to concentrate their urine; mature GFR by 2y Renal blood flow controlled by afferent and efferent arterioles, 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	

Disease	Clinical findings	Urinalysis	Investigations	Treatment
Pediatric RIFLE (1) Stage / Change in estimated Creatinine Clearance[b] / Urine Output At Risk of kidney injury: Decrease by 25% / <0.5 mL/kg/h for 8 hours Injury to the kidney: Decrease by 50% / <0.5 mL/kg/h for 12 hours Failure of the kidney: Decrease by 75% or <35 mL/min/1.73 m^2 / <0.5 mL/kg/h for 24 hours or anuria for 12 hours Loss of kidney function: Failure for >4 weeks End stage kidney disease: Failure for >3 months				
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)		Loss of ability to concentrate urine Muddy granular casts = ATN Red cell casts = GN 	

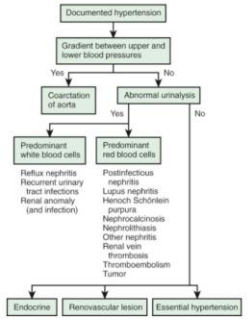
Test	Prerenal AKI	Intrinsic AKI
Urine specific gravity	>1.020	≤1.010
Urine sodium, mEq/L	<20	>40
Fractional excretion of sodium	<1% (neonates <2%)	>2% (neonates >2.5%)
Fractional excretion of urea	<35%	>50%
Urine osmolality, mOsm/kg	>500	<350
Urea nitrogen–creatinine ratio	>20	10–15

AKI=acute kidney injury.

Disease	Clinical findings	Urinalysis	Investigations	Treatment
	Glomerular – glomerulonephritis, systemic disease **Vascular** – microangiopathic processes (HUS, TTP), systemic vasculitides			
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	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	

Disease	Clinical findings	Urinalysis	Investigations	Treatment
		Reproduction: impaired fertility		
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	

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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)	Aldo affects H/ATPase responsible for H secretion Aldo stimulates K secretion therefore get hyperkalemia, worsens H secretion		Elevated urinary sodium Decreased urinary potassium	

Disease	Clinical findings	Urinalysis	Investigations	Treatment
Can also happen from obstructive uropathies	Growth failure, polyuria, polydipsia, hyperkalemia			
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 	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

Disease	Clinical findings	Urinalysis	Investigations	Treatment

CLASSIFICATION	SBP OR DBP PERCENTILE	FOLLOW-UP
Prehypertension	90th to <95th percentile or, if BP exceeds 120/80 mm Hg, to <95th percentile	Recheck in 6 mo and consider school or home BP monitoring
Stage 1 hypertension	95th to 99th percentile plus 5 mm Hg	Evaluate within 1 mo
Stage 2 hypertension	>99th percentile plus 5 mm Hg	Evaluate within 1 wk or immediately if symptomatic
White-coat hypertension	BP >95th percentile in the medical setting but normal outside the medical setting	Consider ABPM, as well as school or home BP monitoring
Masked hypertension	BP <95th percentile in the medical setting but >95th outside the medical setting	Consider ABPM in high-risk populations (ie, chronic kidney disease and diabetes)

ABPM=ambulatory blood pressure monitoring, BP=blood pressure, DBP=diastolic blood pressure, SBP=systolic blood pressure.
Adapted with permission from National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. *2004;114:555–576.*

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

Disease	Clinical findings	Urinalysis	Investigations	Treatment
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

Question

- Q16. List four diagnoses that would be consistent with a low C3:
  - Lupus
  - PSGN
  - Membranoproliferative glomerulonephritis
  - C3 glomerulopathy
  - Shunt nephritis
  - Subacute bacterial endocarditis