Chronic Kidney Disease

Diagnostic Studies	• Chem 10 • UA w/ urine protein:Cr ratio • CBC/diff/retic + iron studies • 25-OH Vitamin D, PTH • Fasting lipid panel • If etiology uncertain: see sections on proteinuria/hematuria, consider renal U/S and bx
Management	Stage G1/G2 → • Monitor kidney function closely • Educate about nephrotoxin avoidance (NSAIDs, contrast, smoking, obesity, dehydration) • BP control w/ ACEI/ARB ▪ ESCAPE trial - N Engl J Med. 2009;361(17):1639. Using ramipril (starting at 6 mg/m2/d and inc dose / adding other agents as needed), targeting 50th %ile BP for age, sex, and weight vs 90th %ile slowed rate of progression to ESRD Stages G3 and above, add the following → • Prepare for possibility of transplant, ideally prior to dialysis (HD vs peritoneal) • Na-restricted diet (2-3g/d) +/- diuretics (furosemide 0.5-2 mg/kg/d, HCTZ 1-3 mg/kg/d) • Management of hyperkalemia (low K diet, diuretics), acidosis (Na bicarb), hypocalcemia/ hyperphosphatemia (Vitamin D, calcimimetics, phos binders) • Rx anemia to goal Hgb 10-12 g/dL w/ EPO-stimulating agents (erythropoietin alfa, darbepoetin alfa) • In pts with significant uremia, consider preoperative DDAVP to prevent bleeding

Hemolytic-Uremic Syndrome

Definition	• **Hemolytic Uremic Syndrome**: microangiopathic hemolytic anemia + AKI + thrombocytopenia • **Thrombotic Thrombocytopenic Purpura**: triad of HUS + fever + neurologic changes
Etiology	• Principally affects children under the age of five years. • 90% due to shiga toxin; of those 70% due to *enterohemorrhagic* E. Coli • Occurs in 6-9% of EHEC infections; usually begins 5-10 days after diarrhea onset • Non-diarrheal (atypical) HUS associated can be due to *S. pneumo* infection or due to defects in the complement system (e.g., mutations in complement regulatory proteins)
Pathophysiology	• **HUS**: Shiga toxin binds to receptors in glomerular, colonic, and cerebral cells → promotes adhesion and aggregation of platelets onto endothelial cells → thrombocytopenia and RBC shearing (microangiopathic anemia); in kidney, glomerular damage • **TTP**: due to deficiency or immune-mediated inhibition of ADAMTS13, a metalloproteinase responsible for breakdown of vWF. No vWF cleavage → coagulation occurs at a higher rate, particularly in microvasculature → platelet consumption → thrombocytopenia and microthrombi → microangiopathic hemolytic anemia.
Clinical Manifestations	• **Microangiopathic hemolytic anemia**: jaundice, pallor, dark urine • **Thrombocytopenia**: petechiae, bleeding • **Acute renal failure**: HTN, edema • **Central nervous system**: seizures, coma, stroke • **Cardiac**: dysfunction due to ischemia, uremia, fluid overload. • **Pancreas**: transient DM • **Liver**: Hepatomegaly, increased serum transaminases • **Heme**: In addition to anemia and thrombocytopenia, leukocytosis is common in diarrhea-induced HUS; the prognosis is worse with increased white blood cell counts

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