RENAL VASCULAR PROBLEMS (P. 1142)

NEPHROSCLEROSIS

- Consists of sclerosis of the small arteries and arterioles of the kidneys
  - Decreased blood flow →patchy necrosis of renal parenchyma
  - Ischemic necrosis, glomeruli destruction and fibrosis may occur
- Benign nephrosclerosis
  - From HTN and atherosclerosis vascular changes
  - Normal renal function in early stages, may only detect HTN
  - Treat like normal HTN
- Accelerated (malignant) nephrosclerosis
  - Associated with malignant HTN (sharp increase in BP with diastolic >130)
  - Renal insufficiently progresses rapidly
  - Aggressive anti-HTN therapy
- Complications = renal failure and dysfunction due to HTN
- Prognosis is poor if untreated

RENAL ARTERY STENOSIS

- Partial occlusion of one or both renal arteries and their major branches
- Due to atherosclerotic narrowing or fibromuscular hyperplasia
- Should be considered is HTN suddenly occurs, especially in patients 30-50
- Renal arteriogram to identify renal artery stenosis
- Goal is to treat HTN and restore kidney perfusion
- Renal ischemia or renovascular hypertension = surgical intervention to restore renal blood flow

RENAL VEIN THROMBOSIS

- Unilateral or bilateral
- Due to trauma, extrinsic compression (tumor or aortic aneurysm), renal cell carcinoma, pregnancy, contraceptive use and nephrotic syndrome
- Flank pain, hematuria, fever or nephrotic syndrome
- Treatment
  - Anticoagulation (heparin or warfarin) → avoid pulmonary emboli
  - Corticosteroids for nephrotic syndrome
  - Surgical thrombectomy

HEREDITARY RENAL DISEASES (P. 1142 - 1143)

POLYCYSTIC KIDNEY DISEASE (PKD)

- Most common, life threatening genetic disease
- Involves both kidneys, the cortec and medulla are filled with large, thin-walled cysts that range from mm to cm
  - The cysts enlarge and destroy surrounding tissue, may be filled with fluid, blood or pus
- Childhood manifestation – rare, due to autosomal recessive disorder – rapidly progressive
  - Has been found in newborns
- Adult manifestation – autosomal dominant disorder – latent then appears around 30-40
- Clinical manifestation

    - Early – no S/S → symptoms begin with cyst enlargement(some never have S/S and are never diagnosed)
    - First clinical manifestation can be UTI or renal calculi
    - Later – HTN, hematuria (cyst rupture), feeling of heaviness in the back, side or abdomen
    - Chronic pain can be constant and severe
    - Bilateral kidneys are often palpable
- Diagnosis: clinical manifestations, family history, ultrasound (best screening measure) or CT
- Progressive loss of kidney function → ESRD (50% by age 60)
- Can affect the liver (liver cyst), heart (valve abnormalities), blood vessels (aneurysm) and intestines (diverticulosis) **Most serious complication is cerebral aneurysm**
- Nursing and collaborative interventions
    - Aim to prevent infection or prompt infection treatment
    - Possible nephrectomy is pain, bleeding or infection becomes choric
    - Dialysis or transplant may be necessary
    - Similar nursing management as for ESRD
        - Diet modification and fluid retriction
        - Antihypertensives
        - Assist patient to accept chronic disease process
        - Assist patient and family to cope with financial and heredity concerns

MEDULLARY CYSTIC DISEASE

- Autosomal recessive
    - Renal failure before 20
- Autosomal dominant
    - Renal failure after 20
- Most cysts are in the medulla, asymmetric shaped kidneys with signifant scarring
- Polyuria, progressive renal failure, severe anemia, metabolic acidosis, and poor sodium conservation (defects in concentration abilities)
- ESRD interventions , HTN is often a terminal event

ALPORT SYNDROME (AKA chronic hereditary nephritis)

- Classic Alport syndrome
    - Inherited as a sex-linked disorder with hematuria, sensorineural deafness and anterior surface lens deformities
- Nonclassic Alport syndrome
    - Autosomal trait causing hematuria but no deafness or lens defects
- Men are effect earlier and more severely
    - Usually diagnosed before age 10
- Gene mutation altering synthesis of GBM
- Hematuria and progressive uremia is most common
- Treatment is supportive because corticosteroids and cytotoxic drugs do not work
    - Does not reoccur after transplantation