Renal Outline - Child

I. Wilm's Tumor (Nephroblastoma)

A. Overview
   1. Renal malignancy that may involve one or both kidneys
   2. Most common malignant neoplasm of the kidney in children

B. Symptoms & Diagnosis
   1. Often discovered by a parent who notices an increased abdominal size or by MD during routine physical exam (abdominal mass)
   2. "mass appears overnight" usually favors left kidney
   3. S&S- abdominal pain, vomiting, fever, hematuria, some have HTN

C. Treatment
   1. Depends on staging of the tumor process
   2. DO NOT palpate the abdomen any more than necessary
   3. Surgery – chemo – radiation

D. Prognosis- Usually excellent, however if relapse – prognosis is poor

II. Enuresis

A. Definition – involuntary voiding of urine beyond the expected age at which voluntary control should be achieved after successful toilet training.

B. Classification
   1. Primary – never achieved dryness for at least 3 mths
   2. Secondary- child dry for a period of time, then starts wetting again
   3. Diurnal- daytime wetting only (more common in girls)
   4. Nocturnal- nighttime wetting only (more common in boys)

C. Diagnosis
   1. Organic factors: structural disorders, UTI, constipation, diabetes, kidney disorders
   2. Non-organic factors: sleep disturbances/disorders, stress
   3. Physical exam and detailed history
   4. Voiding diary by parents
   5. Renal ultrasound

D. Treatment
   1. If known cause- treat the specific cause
   2. Medications – used with other therapies. Not a cure, part of treatment
      a) Oxybutyin chloride (Ditropan)- reduces overactive bladder symptoms
      b) Desmopressin (DDAVP)- reduces flow of urine (nighttime usage)
      c) Imipramine (Tofranil)- inhibits urination, can lighten sleep
   3. Bed wetting alarms – sensor on pad under child or on the undergarments, alarms when wet
   4. Motivational therapies – positive reinforcement/rewards for staying dry
   5. Elimination diets- Avoid certain foods and eating/drinking too late in the evening before bed

III. Nephrotic Syndrome

A. Definition-clinical state that includes massive proteinuria, hypoalbuminemia, hyperlipidemia, and edema. Affects male more, preschool to school age more commonly. Cause unknown.

B. Patho- not fully understood-however we think:
   1. Glomeruli stop working properly and become permeable to proteins and albumin due to some type of issue
   2. Results in massive proteinuria, hypoproteinemia, hypoalbuminemia, and hyperlipidemia
   3. Changes in osmotic pressure occur within intravascular spaces and extracellular edema occurs especially in the abdomen.

C. Main Type

1. Minimal Change Nephrotic Syndrome (MCNS)-most common form 80% of cases

D. S&S- Characteristic symptoms of Minimal Change Nephrotic Syndrome
- **a)** Proteinuria
- **b)** Edema/Ascites
- **c)** Low serum albumin
- **d)** Increased blood lipid level- not fully understood why this happens

E. Clinical Manifestations
1. Weight gain
2. Puffiness, Edema to face, extremities, abdomen
3. Edema of intestinal mucosa- can cause GI disturbances
4. Volume of urine decreases, and urine is frothy
5. Behavior- irritable, lethargic, fatigued

F. Diagnostics
1. History- do the characteristic symptoms exist?
2. UA- looking for increased protein
3. Serum- decreased protein and albumin levels, plasma lipids elevated
4. Renal biopsy- to differentiate between other types of nephrotic syndrome

G. Therapeutic Management
1. Goal: reduce excretion of protein, reduce fluid retention, prevent infection, minimize complications
2. Diet- low salt diet, fluid restriction
3. Corticosteroid therapy- Prednisone – safest, long course though of ~8 weeks
4. May have tendency to relapse 1-3 times a year due to allergies/immunizations/illness

H. Nursing Considerations
1. Strict I&O
2. Daily weights
3. Abdominal girths
4. Loss of appetite support
5. Parent teaching to help avoid relapses, med regimen with steroids, good prognosis usually

I. <u>Acute glomerulonephritis</u>

H. Definition- Inflammation of glomeruli. A reaction that occurs often as a by-product of a recent strep infection or some other type of viral infection.

I. Pathophysiology- what we think happens:
1. Immune complexes or the Strep infection – causes a release of membrane like material from an organism into the circulation – antibodies are formed – immune complex reaction occurs – leukocytes get trapped in glomeruli and occlude the capillary lumen
2. Decreased glomerular filtration rate which causes sodium retention, accumulation of h20, then edema and circulatory congestion. Hypertension occurs also.

J. Clinical Manifestations- usually 1-3 weeks after an illness such as strep
- **a)** Puffiness of face/eyes worse in the AM, edema generalized
- **b)** anorexia
- **c)** Dark colored urine and decreased volume
- **b)** Pale
- **c)** Lethargy, irritable
- **d)** Other: Headache, abdominal discomfort, vomiting,
- **e)** HTN- mild to moderate

K. Diagnosis-
1. UA- hematuria, proteinuria
2. Serum- BUN and Creatinine elevated often
   - **a)** + ASO – antistreptolysin titer which means a recent strep infection occurred

L. Therapeutic Management

1. Antibiotics- to rid the offending organism if strep suspected to be persistent
2. Diuretics-more so given to help htn along with antihypertensives
3. Supportive: Rest periods with activity, good nutrition
4. Improvement= increased urination amount, decreased edema

M. Complications
1. Hypertensive encephalopathy- neurologic complications
2. Acute cardiac decompensation- CHF s/s
3. Acute renal failure

N. Nursing Considerations/ Care
1. Assess Fluid balance, VS, Daily weights

O. Prognosis- complete recovery with follow ups

IV. Hemolytic Uremic Syndrome (HUS)

A. Definition- acute renal disease primarily in infants and small children
1. Clinical Features associated with syndrome:
   - **a)** Hemolytic anemia
   - **b)** Thrombocytopenia
   - **c)** Acute renal failure
   - **d)** Can also have CNS symptoms

B. Etiology- exact cause unknown, could be linked to undercooked meat, contaminated h20, chemicals, viruses, bacteria

C. Pathophysiology
1. Site of injury: endothelial lining of small glomerular arterioles of the kidney
   - **a)** Deposit of platelets and fibrin – partial or complete occlusion of arterioles and capillaries of the kidneys
   - **b)** Erythrocytes and platelets try to travel these occluded vessels – they are fragmented- RBC's are damaged and removed by the spleen = anemia & thrombocytopenia from damage to platelets
   - **c)** Renal failure due to clogged filtering system

D. Clinical Manifestations
1. Hemorrhagic manifestations-bruising, petechiae, bloody diarrhea
2. Vomiting, irritable, lethargic, pale skin
3. Possible CHF symptoms- swelling hands/feet
4. Decreased renal function-oliguria, anuria
5. CNS involvement- possible seizures, coma

E. Diagnosis
1. Labs
   - **a)** Serum- BUN/Creatinine elevated, low Hgb and Hct indicating anemia
   - **b)** Urine- protein and blood

F. Management- treat the AKI and anemia
1. Treat anemia prn- blood transfusions
2. Dialysis prn if severe case
3. Prevent circulatory overload

G. Prognosis- good recovery with prompt treatment

H. Prevention