

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. _____
 - 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) Oxybutynin 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

IV. Acute glomerulonephritis

- A. 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.
- B. 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 h₂O, then edema and circulatory congestion. Hypertension occurs also.
- C. Clinical Manifestations- usually _____ 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
- D. Diagnosis-
 1. UA- _____ , _____ & _____
 2. Serum- BUN and Creatinine elevated often
 - a) + ASO – antistreptolysin titer which means a recent strep infection occurred
- E. 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= _____
- F. Complications
1. _____
 2. _____
 3. _____
- G. Nursing Considerations/ Care
1. _____
 2. _____
 3. _____
- H. Prognosis- complete recovery with follow ups

V. 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
1. _____
 2. _____
 3. _____