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INTRODUCTION

The protein is found in normal urine, and it's divided into:

1-60% plasma proteins: albumin (major) IgG, IgA ,light chains ,heavy chains , transferrin ,haptoglobin ,lysozyme ,amylaze ,kallikrein.

- 2-40% originating from the secretions of the urinary tract including: tamm-horstall, urokinase and secertory IgA.
- The reasonable upper limit of normal protein excretion in healthy-children = 150 mg/24 hrs.

DEFINITION:

The excretion of an excessive amount of protein

(>150mg/day) in the urine as defined by qualitative, semi-quantitative, and quantitative methods.

PATHOGENESIS

There are three basic types of Proteinuria:

1-glomerular

2-tubular

3- Overload

1. Glomerular

- mechanisms:
 - > . increased GFR
 - >. increased glomerular permeability of:
 - basement membrane
 - · endothelial, epithelial cell injury
 - · electrostatic charge barrier

2. Tubulointerstitial

impaired tubular reabsorption of filtered proteins

o proteins: low M.W.(molecular weight) proteins, lysozyme, B2-microglobulin

Tamm-Horstall protein

3. Overload Proteinuria

increased load overloads tubular reabsorptive capacity

o proteins: low MW plasma proteins, Ig light chains, myoglobulin, lysozyme, albumin



Abnormalities of Profeinuria:

1. Benign Proteinuria:

a. Benign Transient Proteinuria:

benign proteinuria associated with precipitating factors - fever, exercise, stress, cold weather, dehydration, high blood pressure, seizures, etc

b. Orthostatic Proteinuria:

- diagnosis based upon the Postural Test
- has a benign clinical course

- **C.** Persistent Asymptomatic Proteinuria
- **d.** Congenital Nephrotic Syndrome
 - **f** Poststreptococcal GN
- **<u>Q. Hemolytic Uremic Syndrome</u>**
- h. Henoch-Schoenlein Purpura
- Primary Glomerulonephritis
- **Secondary Glomerulonephritis**
- k. Hereditary Tubulointerstitial Proteinurias

2. Pathologic Proteinuria

A- Glomerular:

- 1. Hereditary
- 2. Non-Hereditary

B-Tubulointerstitial:

- 1. Hereditary
- 2. Non-Hereditary

Glomerular:

1. Hereditary:

Congenital Nephrotic Syndrome

Alport Syndrome

2. Non-hereditary:

a) Acute (GN)

b) Chronic (primary and secondary)

3. Overload Proteinuria

1. Neoplastic

Amyloidosis

Leukemia (monocytic, monomyelocytic) - lysozymuria

Multiple Myeloma

Waldenstrom's Macroglobinemia

Family History

helps to differentiate <u>hereditary</u> from <u>non-hereditary</u> forms of both glomerular and tubulointerstitial forms of proteinuria:

1. Proteinuria

family members must have had previous urinalysis to ascertain this

2. Renal Disease

Polycystic Kidney Disease
Nephrotic Syndrome, Fanconi Disease
renal dialysis
kidney transplantation

3. Others

hearing/ocular impairment (Alport Syndrome)





INVESTIGATIONS: Proteinuria (Diagnosis)

1. Qualitative - Dipstick

- > measures a range of protein concentrations
- > depth of colour increases in a semiquantitative manner with increasing urinary protein concentration
- > to rule out false positives must have:
 - >3 samples with proteinuria
 - First voided early morning samples
 - >pH < 6.0 with known specific gravity

- >1+ (72-240 mg/24 hrs) or greater is considered abnormal
- >dependent on specific gravity of urine sample
- >FP: gross hematuria, highly alkaline urine, UTI
- 2. Semi-quantitative Protein/Creatinine Ratio in Urine
- o random early morning single voided specimen
 - > children < 2 years : < 0.5
 - > children > 2 years : < 0.2
 - > nephrotic : > 3.5
- o correlates with the 24 hour protein excretion data

3. Quantitative - 24 hr. urine collection

- gold standard
- if dipstick is 1+ or more than obliged to do a 24 hour urine collection
- 24 hr. urine collection
 - > Protein (mg)/m2/hr
 - < 4 mg/m2/hourr (normal)</p>
 - 4-40mg/m2/hour (proteinuria)
 - 40 mg/m2/hour (nephrotic)
 - > Protein (mg)/24 hr
 - 2 to 12 months : > 155 mg/24 hr
 - 3 to 4 years : > 140 mg/24 hr
 - 4 to 10 years : > 190 mg/24 hr
 - 10 to 16 years : > 250 mg/24 hr
- FP: radiographic contrast media, cephalosporins, pencillin analogues, sulfonamide metabolites

MANAGEMENT:

- 1)Treat underlying disorder
- 2)Treat complications