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FACULTY OF NURSING

Nephrotic Syndrome

BY:-

Arpit Kamal

Nursing Tutor

MSN Department

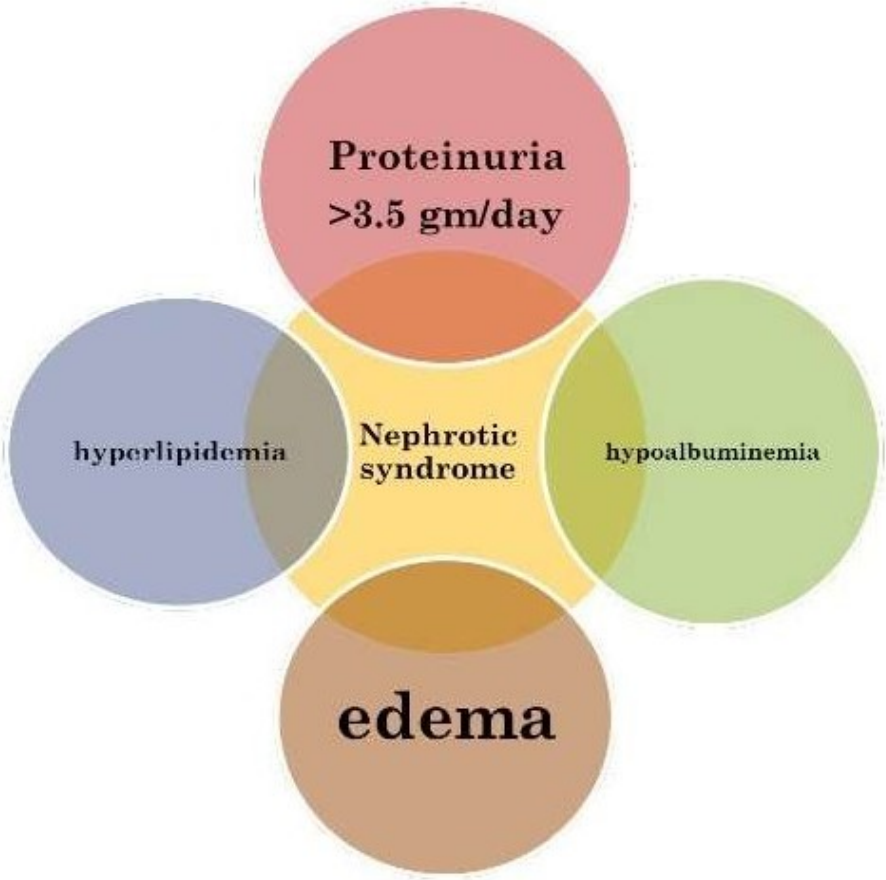
Rama College Of Nursing

Definition

Nephrotic syndrome is a clinical complex characterized by a number of renal and extrarenal features, most prominent of which are

- Proteinuria (in practice > 3.0 to 3.5gm/24hrs),
- Hypoalbuminemia,
- Edema,
- Hypertension
- Hyperlipidemia,
- Lipiduria and
- Hypercoagulabilty.

NEPHROTIC SYNDROME IS NOT A DISEASE



Classification

Nephrotic syndrome can be

- **Primary,** being a disease specific to the kidneys,
- **Secondary**, being a renal manifestation of a systemic general illness

Primary causes

Primary causes include-

- Minimal-change nephropathy(70-90% children and 10-15%inadult)
- Focal glomerulosclerosis (15%inadult)
- Membranous nephropathy (30%inadult)
- Mesangial proliferative glomerulonephritis .
- Rapidly progressive glomerulonephritis

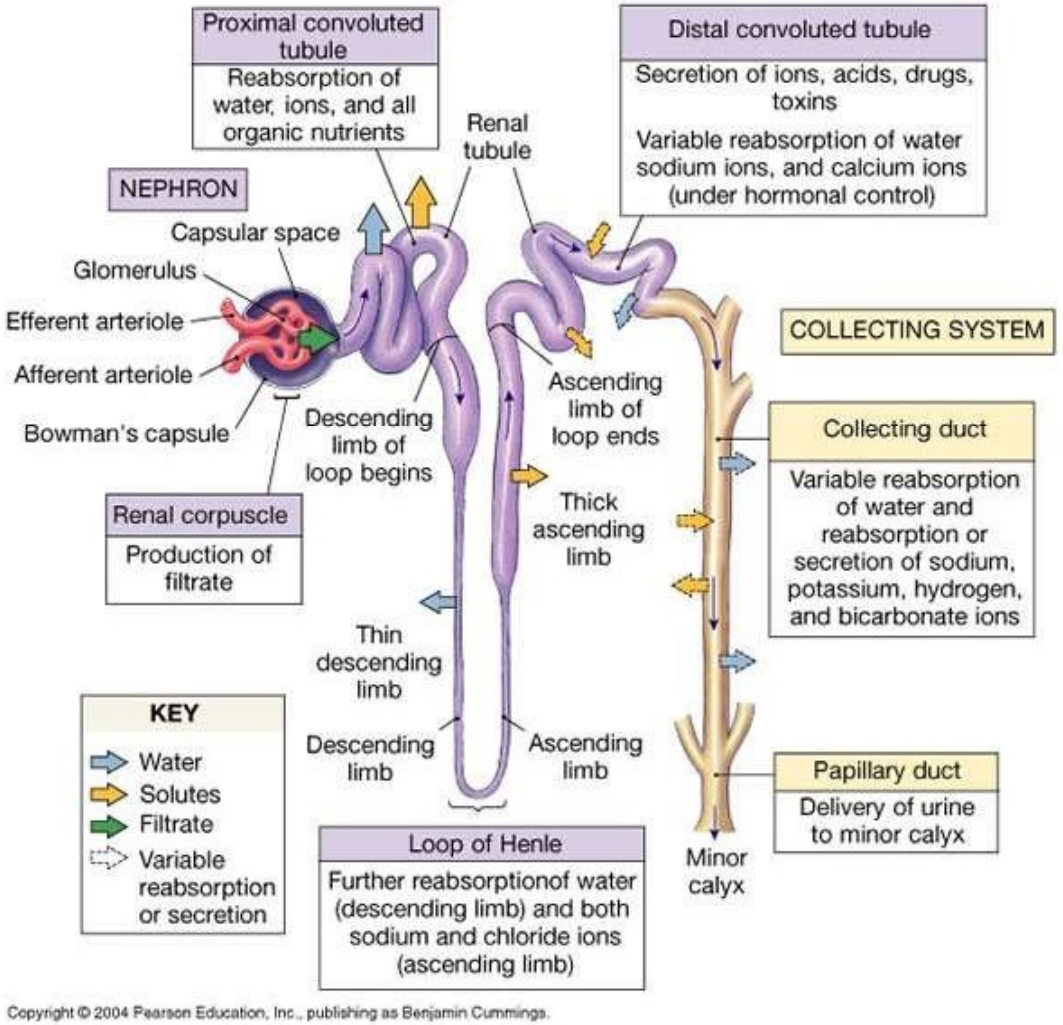
Secondary causes

Secondary causes include-

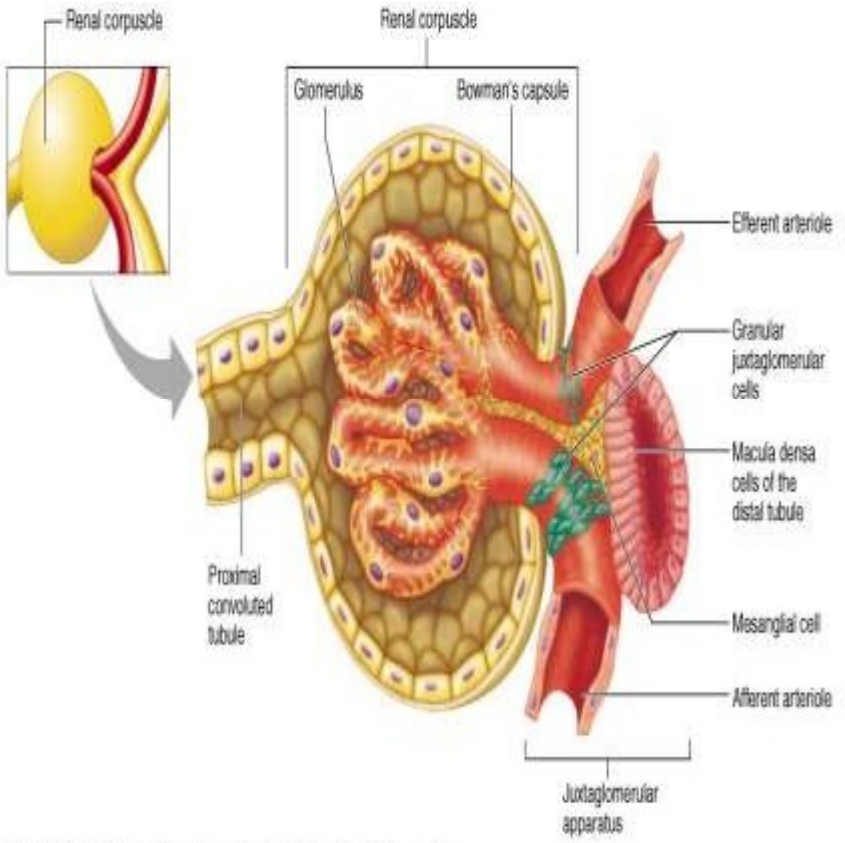
- Diabetes mellitus
- Lupus erythematosus
- Amyloidosis and paraproteinemias
- Viral infections (eg, hepatitis B, hepatitis C, HIV)
- Preeclampsia

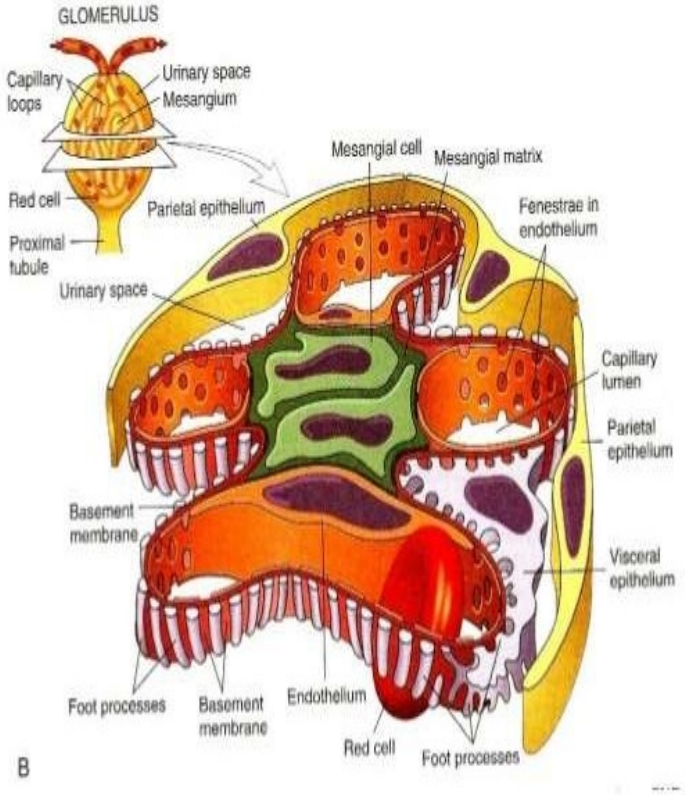
- Nephrotic syndrome is 15 times more common in children
- Most cases in children are due to minimal-change disease.
- In adults, the most common form is membranous glomerulonephritis, followed by FSGS.
- Diabetic nephropathy is emerging as a major cause of nephrotic syndrome

Nephron



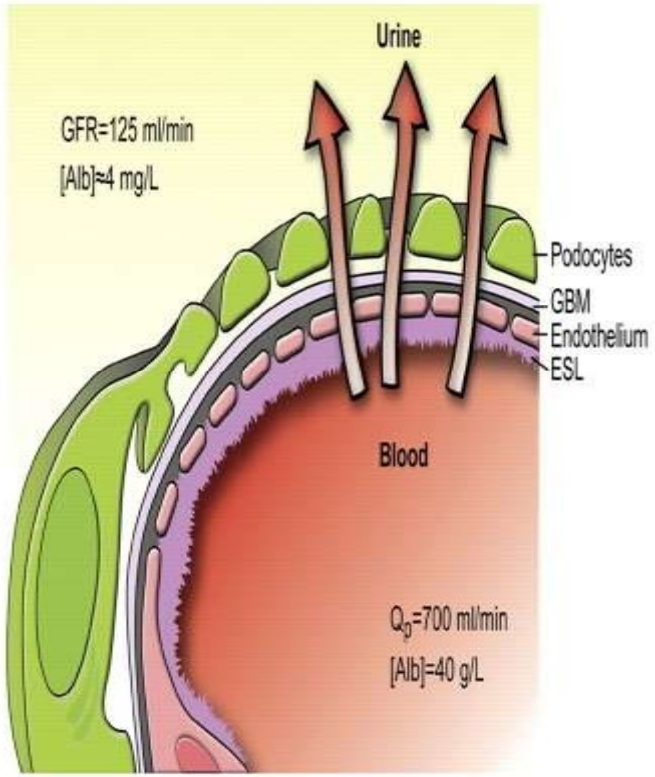
Normal glomerulus





Pathophysiology of proteinuria

- In a healthy individual, less than 0.1% of pl. albumin may traverse the glomerular filtration barrier.
- glomerular capillaries are lined by a fenestrated endothelium that sits on the glomerular basement membrane
- Which in turn is covered by glomerular epithelium, or podocytes, which envelops the capillaries with cellular extensions called foot processes. In between the foot processes are the filtration slits.
- These 3 structures are the glomerular filtration barrier



Pathophysiology of proteinuria

- The glomerular structural changes that may cause proteinuria are damage to the endothelial surface, the glomerular basement membrane, or the podocytes.

- Glomerular haemodynamics (Intraglomerular hypertension and hyperfiltration) can alter Glomerular permeabiality.

- **Selectivity of proteinuria-** Excretion of relatively low M.W. protein (Albumin or transferrin) is known as selective proteinuria while if excretion is predominately high M.W. protein (IgG, IgM or α2 macroglobulin) it is nonselective proteinuria.

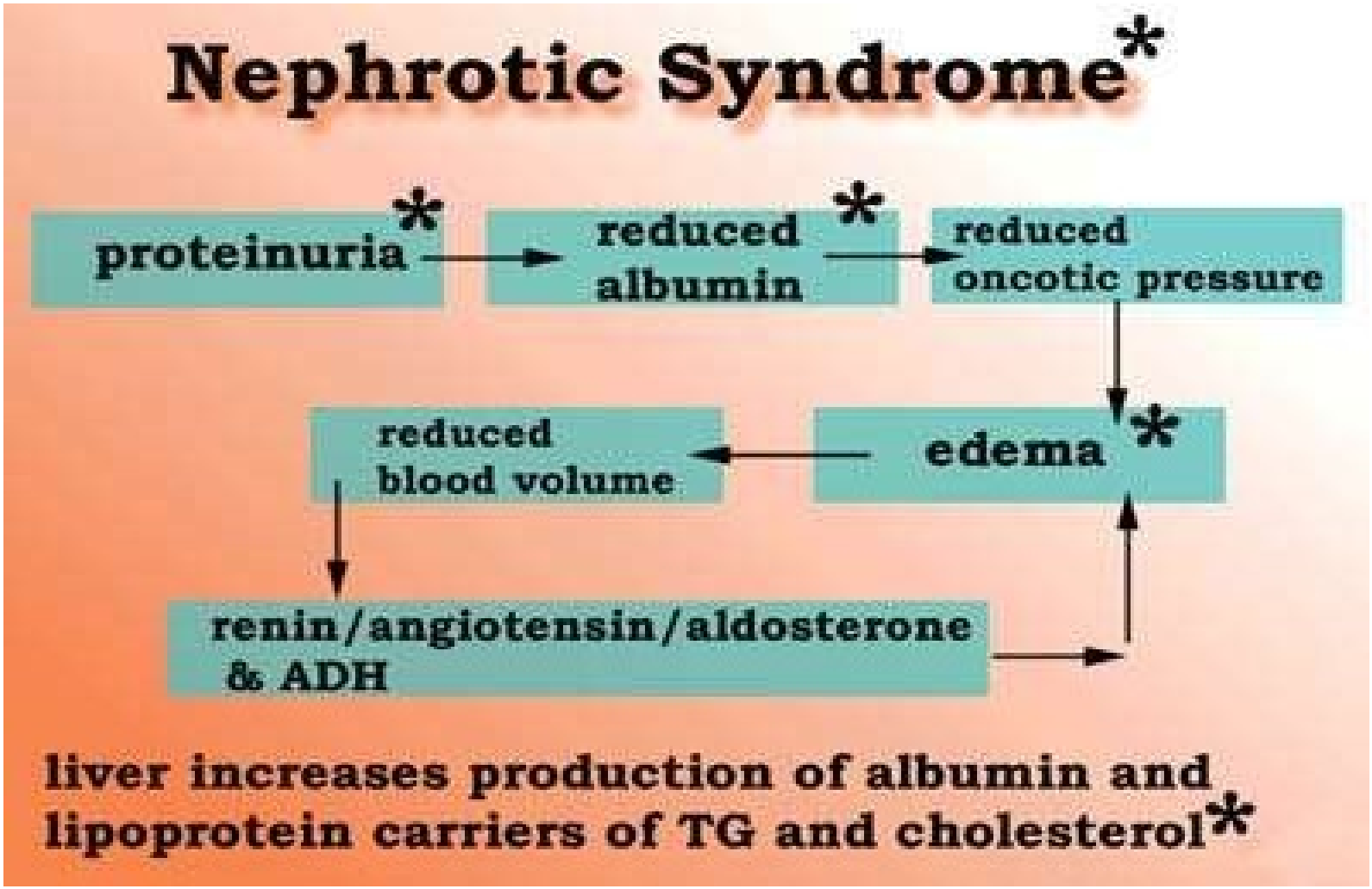
- It is also related to relative damage of Glomerular filter.

Hypoalbunemia

- It is due to both the proteinuria and due to the increase renal catabolism (in tubules).

- In fact hepatic albumin synthesis is increased from 145±9mg/kg/day to 213±17mg/kg/day in nephrotic patients.

Pathogenesis of edema



Metabolic consequences of proteinuria

Metabolic consequences of the nephrotic syndrome include the following:

- Infection
- Hyperlipidemia and atherosclerosis
- Hypocalcemia and bone abnormalities
- Hypercoagulability
- Hypovolemia

Proposed explanations of Infection in NS

Proposed explanations include the following:

- Urinary immunoglobulin losses
- Edema fluid acting as a culture medium
- Protein deficiency
- Decreased bactericidal activity of the leukocytes
- Immunosuppressive therapy
- Urinary loss of a complement factor (properdin factor B) that opsonizes certain bacteria

Hypperlipedemia

- Due to increase hepatic lipoprotein synthesis that is triggered by reduced oncotic .
- Defective lipid catabolism has also important role.
- LDL and cholesterol are increased in majority of patients whereas VLDL and triglyceride tends to rise in patients with severe disease.
- It increases the relative risk for MI 5.5 fold and coronary death 2.8 fold.
- It also increases progression of renal disease

Hypercoagulability

- Multifactorial in origin
- Increase urinary loss of antithrombin III.
- Altered levels and/or activity of protein C & S.
- Hyperfibronogenemia due to increase hepatic synthesis.
- Impaired fibrinolysis due to decrease plasminogen.
- Increase platelet aggregability – relative immobility - haemoconcentragtion from hypovolemia. – hyperlipidemia
- Alteration in endothelial function

Hypocalcemia

- Hypocalcemia is common in the nephrotic syndrome, but rather than being a true hypocalcemia, it is usually caused by a low serum albumin level.

- Nonetheless, low bone density and abnormal bone histology are reported in association with nephrotic syndrome.

- This could be caused by urinary losses of vitamin D–binding proteins, with consequent hypovitaminosis D and, as a result, reduced intestinal calcium absorption.

Hypovolemia

- Hypovolemia occurs when hypoalbuminemia decreases the plasma oncotic pressure,
- Resulting in a loss of plasma water into the interstitium and causing a decrease in circulating blood volume.
- Hypovolemia is generally observed only when the patient's serum albumin level is less than 1.5 g/dL.
- Hypotension is a late feature

FUNCTIONAL CONSEQUENCE OF URINARY LOSS OF PLASMA PROTEIN

- Thyroid binding globulins and thyroxin – may lead to hypothyroidism.
- Vit D binding protein – oesteomalacia, but rare
- Total calcium is also low due to low albumin level.
- Transferrin and erythropoietin and – microcytic hypochromic anemia.
- ARF – is rare in nephrotic syndrome. In whom it occur patient are elderly of minimal changes disease / FGSS

Symptoms and signs

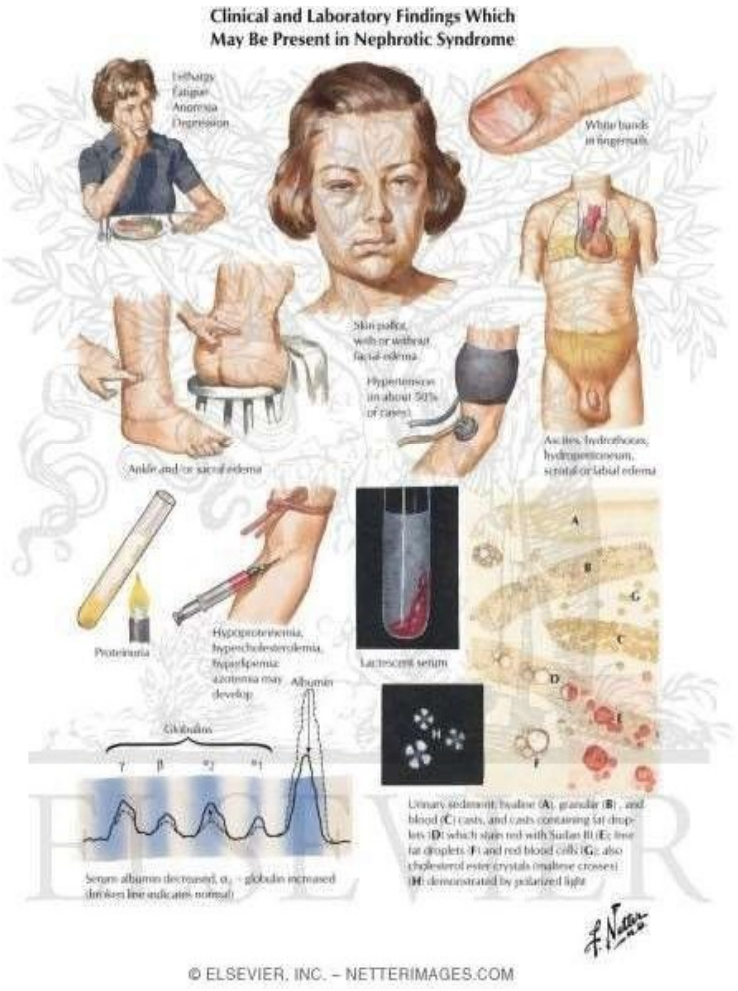
- Include anorexia, malaise, puffy eyelids, retinal sheen, abdominal pain, wasting of muscles, and edema.

- Most often, the **edema** is mobile - detected in the eyelids in the morning and in the ankles after ambulation

- **Focal edema** may be the reason for seeking help for such complaints as:
  - difficulty breathing (pleural effusion or laryngeal edema),
  - substernal chest pain (pericardial effusion),
  - scrotal swelling,
  - swollen knees (hydroarthrosis),
  - swollen abdomen (ascites), and
  - abdominal pain from edema of the mesentery.

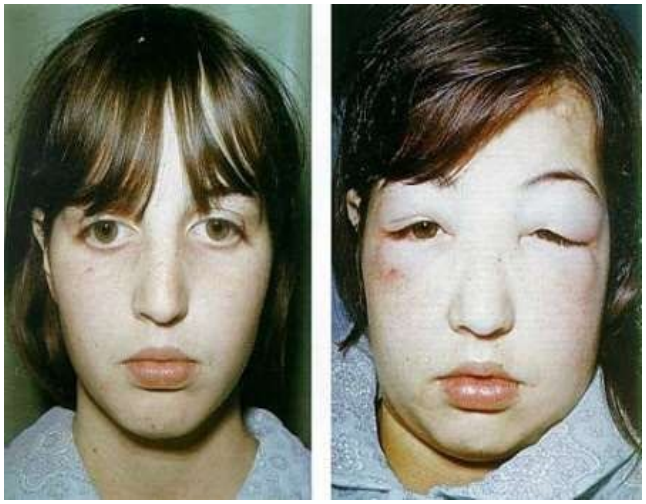
Symptoms and signs

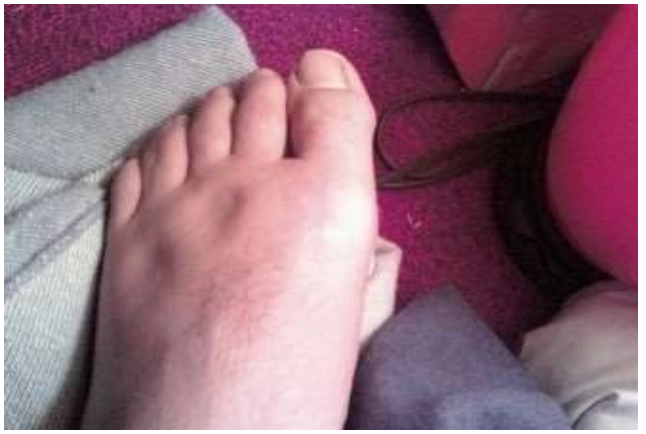
- An early sign of NS is frothy urine.
- At presentation,proteinuria is usually > 2 gm/m2/day, or a urine protein/creatinine ratio is > 2
- Orthostatic hypotension and even shock may develop in children.
- Adults may be hypo-, normo-, or hypertensive.
- Oliguria and even
- Acute renal failure may develop because of hypovolemia and diminished perfusion.



Symptoms and signs

- Prolonged NS may result in nutritional deficiencies, including protein malnutrition
- ,myopathy,
- Decreased total Ca++, tetany
- Spontaneous peritonitis and opportunistic infections
- Coagulation disorders, with decreased fibrinolytic activity
- Episodic hypovolemia, are a serious thrombotic risk (renal vein thrombosis).
- Hypertension with cardiac and cerebral complications.





Differential Diagnosis

- Heart failure
- Cirrhosis
- Glomerulonephritis

Workup

Diagnostic studies for nephrotic syndrome may include the following:

- Urinalysis
- Urine sediment examination
- Urinary protein measurement (24-hr)
- Serum albumin
- Serologic studies for infection and immune abnormalities
- Renal ultrasonography
- Renal biopsy

IMPORTANT DEFINITIONS

- **RESPONSE**; protein free urine on 3 consecutive days within 7 days.
- **RELAPSE;** protein +ve urine on 3 consecutive days within one week with edema.
- **FREQUENT RELAPSING NS;** steroid sensitive nephrotic syndrome with 2 or more relapses in 6 months or more than 3 in one year.
- **STEROID DEPENDANT**; responder who relapses while steroid is being tapered or within 14 days of stopping steroid treatment.
- **INITIAL NON RESONDER;** no response during initial 8 weeks of therapy.
- **LATE NON RESPONDER;** an initial steroid responder who fails to respond to 4 week treatment in relapse.

Management

Specific treatment

- In minimal-change nephropathy, glucocorticosteroids, such as prednisone, are used. Children who relapse may be treated with rituximab]
- In some lupus nephritis, prednisone and cyclophosphamide are useful
- Secondary amyloidosis with nephrotic syndrome may respond to anti-inflammatory treatment of the primary disease.
- In membranous nephropathy, expectant management without immunosuppression can be used for the first 6 months, in patients at low risk for progression (ie, those with serum creatinine level < 1.5 mg/dL). Patients with renal insufficiency (serum creatinine level > 1.5 mg/dL) are at greatest risk for the development of end-stage renal disease and should receive immunosuppressive therapy.[37]

Management

Diet and activity

- The diet in patients with nephrotic syndrome should provide adequate energy (caloric) intake and adequate protein (1-2 g/kg/d).
- A diet with no added salt will help to limit fluid overload.
- Management of hyperlipidemia could be of some importance if the nephrotic state is prolonged.
- Fluid restriction *per se* is not required.
- Ongoing activity, rather than bed rest, will reduce the risk of blood clots.

Management

Acute Nephrotic Syndrome in Adults

- **Diuretics** will be needed; furosemide, spironolactone, and even metolazone may be used. Volume depletion may occur with diuretic use, which should be monitored.
- **Anticoagulation** has been advocated by some for use in preventing thromboembolic complications,
- **Hypolipidemic agents** may be used, but if the nephrotic syndrome cannot be controlled, the patient will have persistent hyperlipidemia.
- **ACE inhibitors and/or ARB** are widely used. These may reduce proteinuria by reducing the systemic blood pressure, by reducing intraglomerular pressure, and also by direct action on podocytes.

Management

- **Long-Term Monitoring-** Follow-up care in patients with nephrotic syndrome includes
- Immunization,
- Treatment of relapses of steroid-responsive nephrotic syndromes,
- Monitoring for steroid toxicity, and
- Monitoring of diuretic and angiotensin antagonist regimens.

Medication Summary

- Corticosteroids (prednisone),
- Cyclophosphamide,
- Cyclosporine
- Rituximab
- Mycophenolate
- Diuretics
- ACE inhibitors and
- ARB

THANK YOU