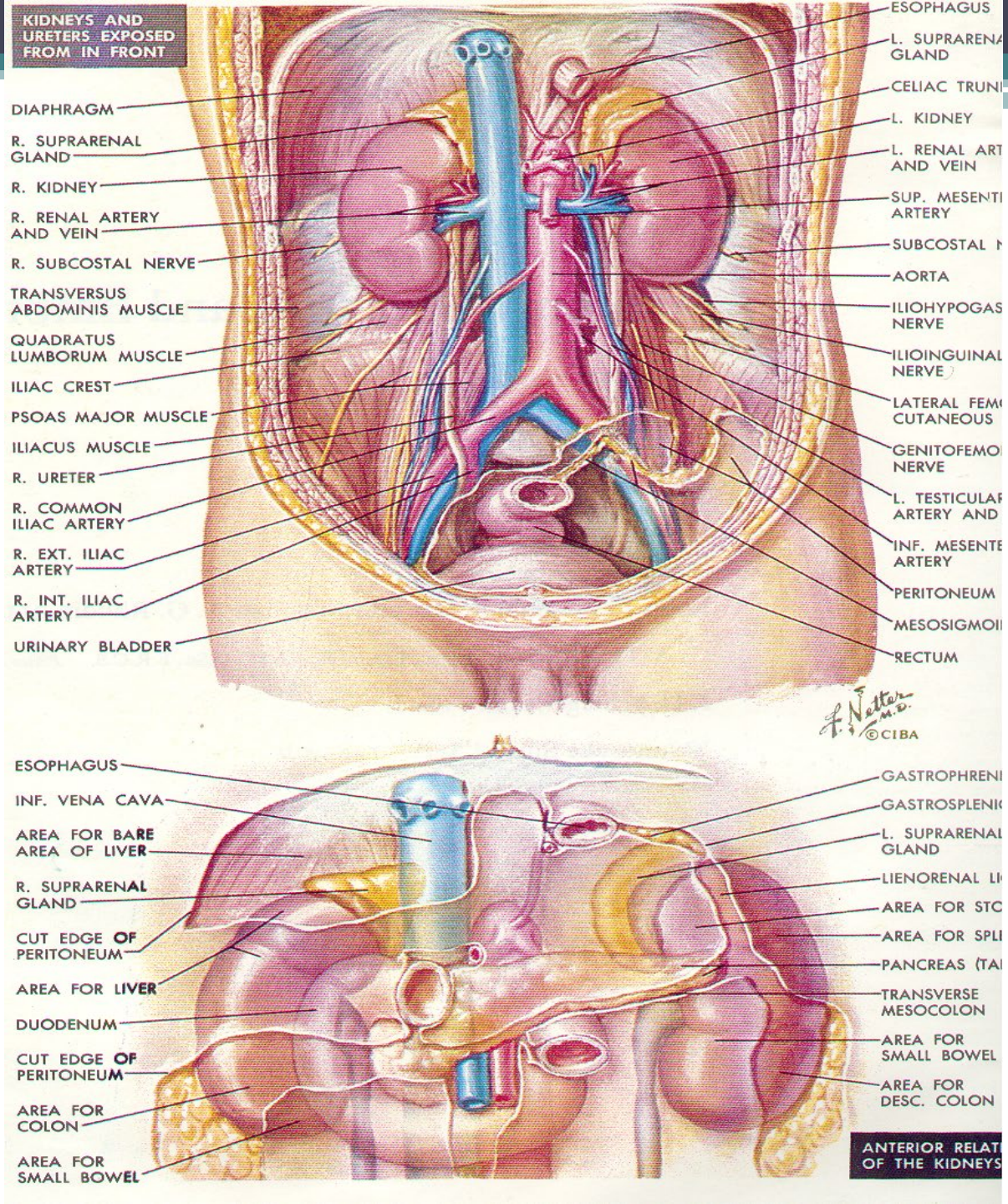
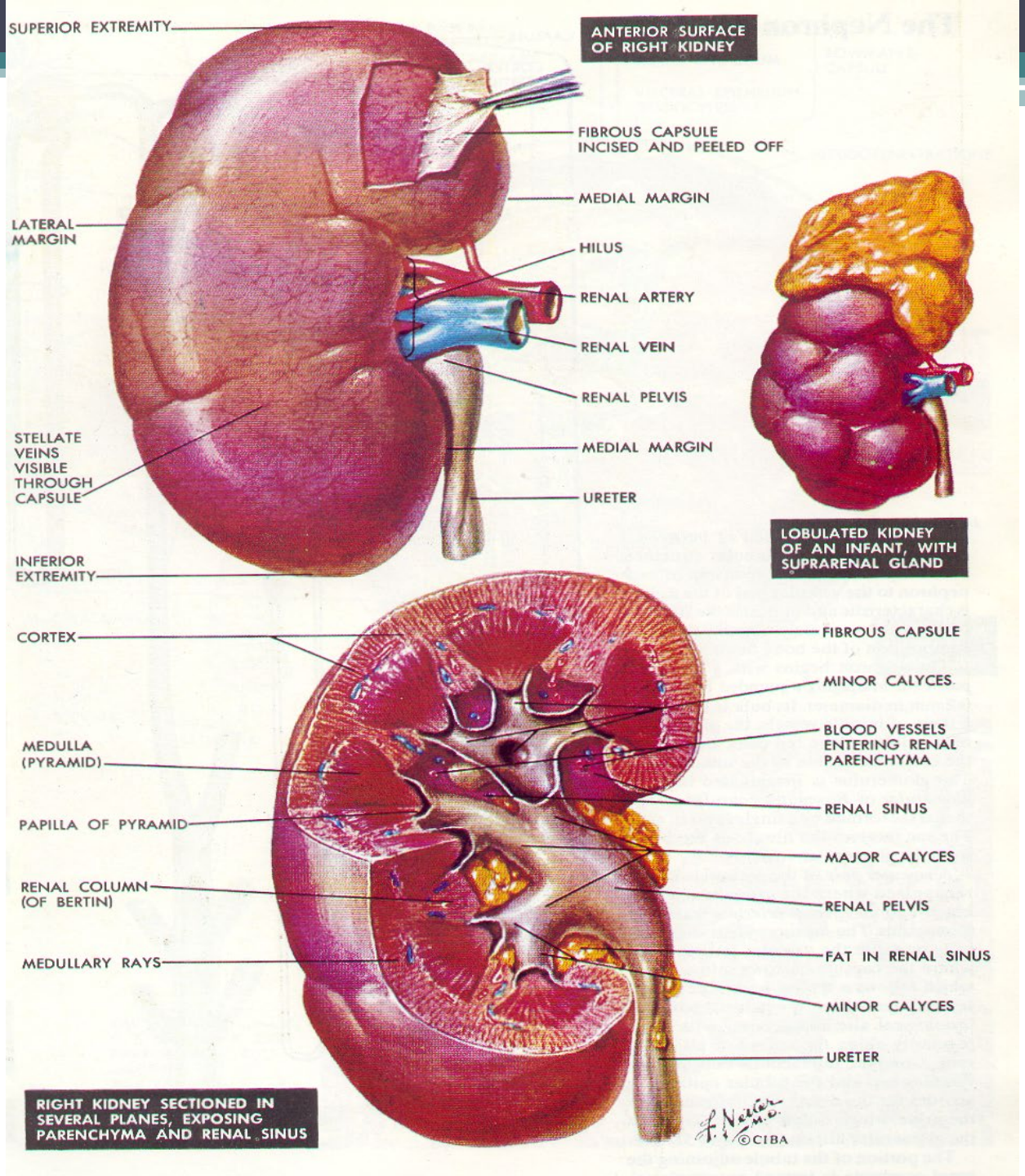
NEFROLOGY INTRODUCTION



The kidney controls the hydro - electrolytic and acid - base balance, also its responsible for the elimination of general metabolism's final products.


SUPERIOR EXTREMITY
ANTERIOR SURFACE OF RIGHT KIDNEY
FIBROUS CAPSULE INCISED AND PEELED OFF
MEDIAL MARGIN
LATERAL MARGIN
HILUS
RENAL ARTERY
RENAL VEIN
RENAL PELVIS
STELLATE VEINS VISIBLE THROUGH CAPSULE
MEDIAL MARGIN
URETER
INFERIOR EXTREMITY
LOBULATED KIDNEY OF AN INFANT, WITH SUPRARENAL GLAND
CORTEX
FIBROUS CAPSULE
MINOR CALYCES
MEDULLA (PYRAMID)
BLOOD VESSELS ENTERING RENAL PARENCHYMA
PAPILLA OF PYRAMID
RENAL SINUS
MAJOR CALYCES
RENAL COLUMN (OF BERTIN)
RENAL PELVIS
MEDULLARY RAYS
FAT IN RENAL SINUS
MINOR CALYCES
URETER
RIGHT KIDNEY SECTIONED IN SEVERAL PLANES, EXPOSING PARENCHYMA AND RENAL SINUS
F. Netter M.D.
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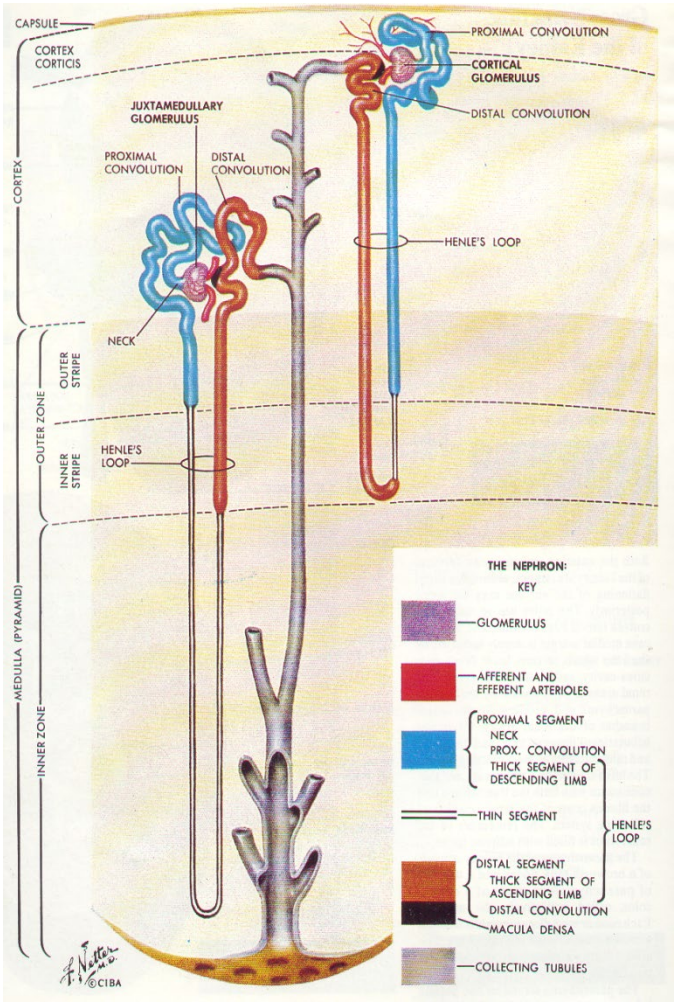
Kidney structure: glomeruli , tubes, interstice, vessels.

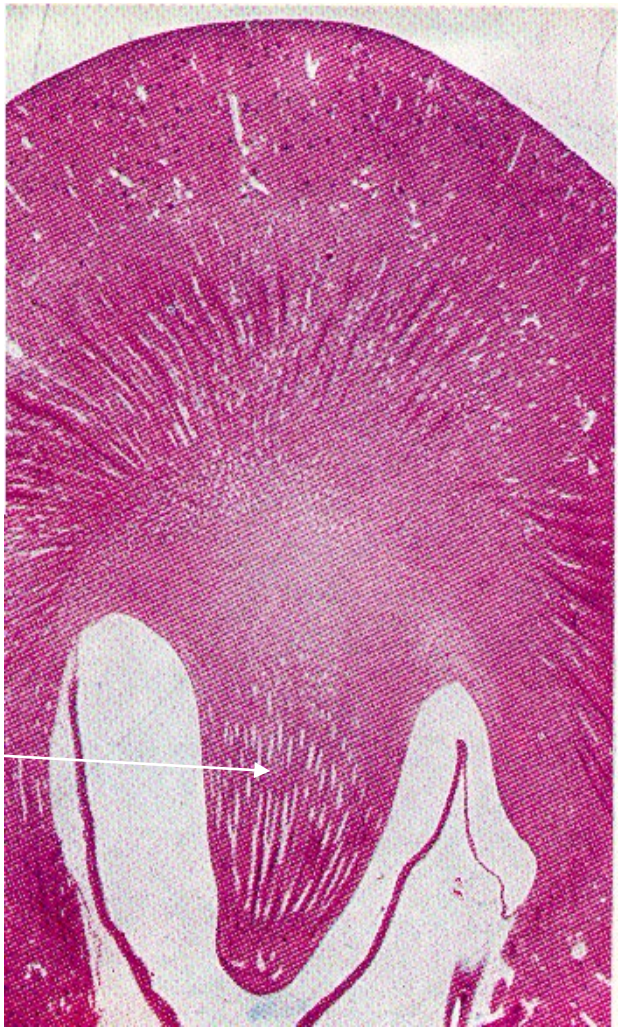


Cortical

Glomeruli ,tubes, interstice.

Medullary

tubes, interstice
glomeruli .



Papilla - tubes, interstice.

Renal deseases clasification

Proceeding from the predominantly affected renal structure (and initially)-

1. **Glomerulopathies**
2. **Interstitial nephropathies - tubulointerstitial nephropathies (ex. pyelonephritis)**
3. **Tubulopathies**
4. **Vessel nephropathies**
    1. **Small vessels**
    2. **Large vessels.**
5. **Obstructive nephropathies**
6. **Nephropathies that involve all the renal structures : ex. Polycystic renal disease with dominant transmission; tumors etc.**

Main syndromes in renal diseases

1. Asymptomatic urinary abnormalities (hematuria, proteinuria)
2. Renal insufficiency
    1. acute
    2. fast progressive
    3. chronic
3. Acute nephritic syndrome
4. Nephrotic syndrome
5. Arterial hypertension
6. Urinary infection
7. Urinary obstruction
8. Renal tubular disorders
9. Reno - urinary lithiasis .

Main syndromes in renal diseases

Urinary isolated anomalies

(proteinuria, hematuria, leucocituria)

Hematuria: lithiasis, tumors, nephritis etc.

Proteinuria: all types of nephropathies; massive proteinuria appears especially in glomerulopathies.

Leucocituria: in contagious or notcontagious glomerular inflammations.

Urinanalysis

- **The urine reflects the functional condition of the kidney also its anatomical integrity and the integrity of the urinary ducts, that's the reason why a "intra vitam " biopsy was indicated**
- **Urinanalysis and anamnesis can establish the nephrological diagnosis**

The urine analysis-

1. **A general summary of urine**
2. **24 hours urinanalysis with the quantitative determination of certain substances,**
3. **fractional urine test exam with the collection of several parameters , (glycosuria, samples of concentration and dilution, caused crystaluria).**

Urinalysis

Collection conditions of a summary urinanalysis:

- **The first morning urine (the most concentrated), is collected.**

!!!24 hours before the collection, the drugs administration (aspirin, sulphamides) is interrupted because they can increase the number of crystals and erythrocytes in the urine.

!!!In the evening a reduced hydro regime is administrated to avoid the determination of a hypotonic urine that can damage the erythrocytes.

!!!A strict genital hygiene , for women the urine summary is not effectuated during their menstrual period.

- **The collection is made from the middle part of the jet in a clean receptacle with out any trace of carbohydrates, lipids or detergent.**

- **The exam should be effectuated in the first 3 hours after the emission or in the first 12 hours if the urine was maintained at 40 ° C. After 3 hours of improper conditions a microbial flora is developing which transforms the urea in ammoniac and modifies the pH.**

- ***The exam is done at least after one day from the use of radioiodated substances. (determines the false positive reaction at the urinary proteins)***

Urinanalysis

- **Macroscopic exam**
- **Physical and Chemical exam**
- **Microscopic exam**

Urinanalysis

Macroscopic exam

- **Normal urine is clear at the emission or muddy if it contains salts in high quantity (urate, carbonates, oxalates, phosphates) or an abundant microbial flora or fat (chylomicrons)**
- **color: golden yellow to yellow**
  - **hypochromic: up to colorless in polyuria**
  - **hipercrome: red: its provided by food colorants, aminofenazona, pirazolon, hemoglobin (Hb) increased urobilinogen, erythrocytes, porphyrins, and red beets.**
  - **yellow-brown: At the increase of urobilinogen and bile pigment, a yellowish foam is formed (urine liver)**
  - **brown: its provided by tannin, quinine, thymol, the prsence of homogentizic acid (intermediate product in the metabolism of phenylalanine and tyrosine) which gives brown color in alkaline medium (alcaptonuria = the change of urine color in time), in melanurie (Melanie pigments increase in melanosarcom)**
  - **green-blue: amitriptyline, methylene blue, copper, biliverdina**

- **Odor:** **bland, aromatic determined by volatile acids and urinoide substances; diseases give a emphasized odor in concentrated urine, unpleasant if consuming asparagus, garlic,horseradish, ammonia smell (in infection, renal tumors), putrid (anaerobic), sour apple (in DM due to ketone bodies).**

Urinanalysis

Physical and Chemical Exam

- **urinary density** (normally : 1015-1022, limits reach 1003 - 1030)
- **urinary osmolality** determination is made by the osmometre, is normal 800-1200mosmoli / l
- **The determination of pH** is done on fresh urine (normally 5.8-7.4, range between 4.5-8) and is made with pepper pH indicators (classic) - decreases during a high protein diet (5.25.3) and increases during the vegetarian diet (7 -7.5),postprandial becomes alkaline .
  - acid pH in neoplasm, fever, metabolic acidosis uremia, renal failure by decreasing the synthesis of ammonia, which neutralizes the acid radicals
  - alkaline pH in the urinary tract infections, after vomiting and during treatment with antormacid
- **Normal Proteinuria is 50-100mg/24**, quantity that doesn't make the usual chemical reactions positive .

Urinanalysis

Microscopic Exam

a. **Organized sediment**

- **Epithelial cells:** flat epithelial cells (superficial) higher within inflammations;
- **Normally rare leucocytes** (3-4 lens/ field), are granulocytes , PMN; proceed at the ducts or kidney level.
- **Erythrocytes** (1-2/2-3 în lens/field) young (more colorful) and old (les colorful)\ ; in glomerular hematuria, predominant are young and shapeless erythrocytes (after the passing throwh the filter membrane they become fragmented).

- **The casts** are waxwork (copy) of the renal tubes
- Urinary casts are formed only in the distal convoluted tubule (DCT) or the collecting duct (distal nephron). Hyaline casts are composed primarily of a mucoprotein (Tamm-Horsfall protein) secreted by tubule cells.
  - The hyaline casts, have rounded extremities, are transparent.
  - The granular casts are hyaline casts on which's surface have adhered the granulation obtained from the cells disintegration (leucocytes, erythrocytes) or plasmatic proteins.
  - Waxy cylinders (of extended stasis) in Kidney insufficiency at the final stage.

Urinanalysis

Microscopic Exam

b. The unorganized sediment is formed from the salts:

- Acid urine: sodium urate, uric acid, calcium oxalate
- Alkaline urine : amonio – magnezium phosphates, bimagnesium phosphates, tyrosine crystals, lucine, bilirubin crystals

Urinanalisys

Bacteriological urine exam

Interpretation :

- Under 10.000 germs/ml represents an insignificant bacteriuria;
- between 10.000-100.000 suspicion of infection ;
- Over 100.000 – urinary infection ;

HEMATURIA

- Hematuria represents the excretion of an abnormal number of erythrocytes came from above the ribbed (striated) sphincter of urethra through urine.

Ethology of haematuria:

- **Other pathologies** with secondary nephropathies (met in systemic diseases like Henoch-Schonlein purpura, polyarteritis nodosa, SLE,-sd.Goodpasture; Wegener's granulomatosis, endocarditis, diabetes mellitus, lymphomas, bleeding, use of NSAIDs, anticoagulants.
- **Renal causes:** glomerulonephritis, amyloidosis, hereditary nephropathies (sd.Alport, sd.Fabry) renal infections, papillary necrosis, renal tuberculosis, gallstones, gout, nephrocalcinosis, malformations, renal infarction; tumors (malignant, benign), injury.
- **Renal tract disease:** hydronephrosis, congenital abnormalities, infections (tuberculosis, prostatitis), parasites, intravesical foreign bodies, radiation or after cyclophosphamide cystitis, stones, tumors, trauma, vascular anomalies, bladder diseases.
- **Essential hematurias**

Guyon sample (test) (the three receptacles):

- If only the urine from the beginning of the jet is red (in the first receptacle) hematuria has cervico – prostatic origin;
- If the last jet is hematuric, that means that the blood has bladder origin;
- If the blood is hematuric in all the receptacles – the haemorrhage is at the renal or bladder level (its relative assessment).

HEMATURIA (1)

- **A. Isolated hematuria:** cystoscopy is done to find the origin:
- **bladder source**: tumor, inflammatory lesions specific / nonspecific, stones, foreign bodies, parasites, ulcers, diverticulitis;
- **from a single ureteral meatus:** kidney cancer (hematuria give abundant, whimsical, resistant) tuberculosis, stones, polyps, developmental anomalies
- **from both ureteric meatus**: amyloidosis, GN, polycystic disease, "horseshoe" kidney, medullary sponge kidney, vascular anomalies, bilateral stones, other disease with kidney involvement

Hematuria (2)

- **B. Hematuria associated with:**
- **unilateral nefromegaly:** tumor, cyst, pyonefroze
- **bilateral nefromegaly:** polycystic disease, tumor, bilateral hydronephrosis or by obstacle
- **pain:** stones, tuberculosis, tumors, renal infarction
- **with signs of bladder** (polachiuria, dysuria, changes in jet) :
  adenoma / carcinoma of the prostate, bladder stones or bladder tumor

Fever and pyuria indicate infection but 15% of malignant tumors evaluate with fever, proteinuria and cylindruria.

- **Proteinuria and cylindruria**
  - **proteinemia dominated diseases: diabetes, amyloidosis, SLE, benign nefroangiosclerosis**
  - **Hematuria dominated diseases: renal polycyistozsis , coagulopathy, TBC, lytiasis, papillary necrosis**

HEMATURIA

- About glomerular etiology of hematuria optical microscopic examination informs us : glomerular red blood cells in urine are young, deformed (dismorfe) and accompanied by proteinuria and cylindruria
- Hematuria with clots is from urinary tract.
- Profuse hematuria is sudden, abundant (over 30% of urine volume) lasting and difficult to treat (appears in urinary malformations, trauma, inflammation specific / nonspecific tumors, prostate adenoma, stones).

Hematuria

Differential diagnosis of hematuria:

- Microscopic hematuria from normal hematuria :

in macroscopic hematuria urine looks bright red

- **brown urine**: hemoglobinuria, myoglobinuria, porphyirinuria, elevated urates level.
- **red-orange:** after pain relievers, laxatives, anticonvulsants, sedatives, antibiotics, tranquilizers, antihypertensives, antiparkinsonian drugs, myorelaxants.
- **uretroragia** – case in which blood can be removed and outside urination - with bleeding from the genital tract
- glomerular hematuria differentiation of the urinary tract hematuria is made by examining red blood cells: the first case they are younger, smaller and distorted (by passing through the glomerular membrane filter)

PIGMENTURIA

- **Hemoglobinuria** – is a presence of hemoglobin (Hb) free in the urine due to hemoglobinemias (after intravascular hemolysis)
- Hemoglobinaemia etiology :
- **Hereditary haemolysis:** erythrocyte membrane defects (spherocytosis), hemoglobin defects (qualitative - siclemia, quantitative - thalassemia), enzyme defects
- **Acquired haemolysis :**
  - paroxysmal nocturnal hemoglobinuria
  - immunological with to hot / cold autoantibodies, iso-Ac
  - nonimmunological: toxic, drugs (phenacetin), physical agents (hypotonic solution, burns), bacterial, parasitic (malaria), mechanical (microangiopathic hemolytic anemia)

PIGMENTURIA

- **Myoglobinuria (Mg)** means the presence of myoglobin in urine: fresh urine is pink, then it becomes brown
- **Etiology:** rhabdomyolysis (skeletal striated muscle lysis) - Red cell membrane abnormalities
- **classification**
  - sporadic myoglobinuria: the trauma, exercise, ischemic, toxic, drug (heroin, barbiruriates, codeine, etc.), infectious, metabolic diseases (decrease or increase in temperature), idiopathic polymyositis.
  - hereditary myoglobinuria: myophosphorilasic deficiency, deficiency of other enzymes of muscle metabolism

CHILURIA

- Chiluria - elimination of urine is mixed with lymph (milkiness aspect, fatty, oily urine) shows communication between the lymphatic and the renal systems.
- Etiology:

➢ parasites: filariosis, echinococcosis, cysticercosis, ascaridiosis

➢ nonparasitic: lymphatic aneurysms, malformations, compressions of the thoracic duct.

PROTEINURIA

- Proteinuria is a sign commonly seen in kidney disease, but the classification and analysis of the cause is something more complicated.

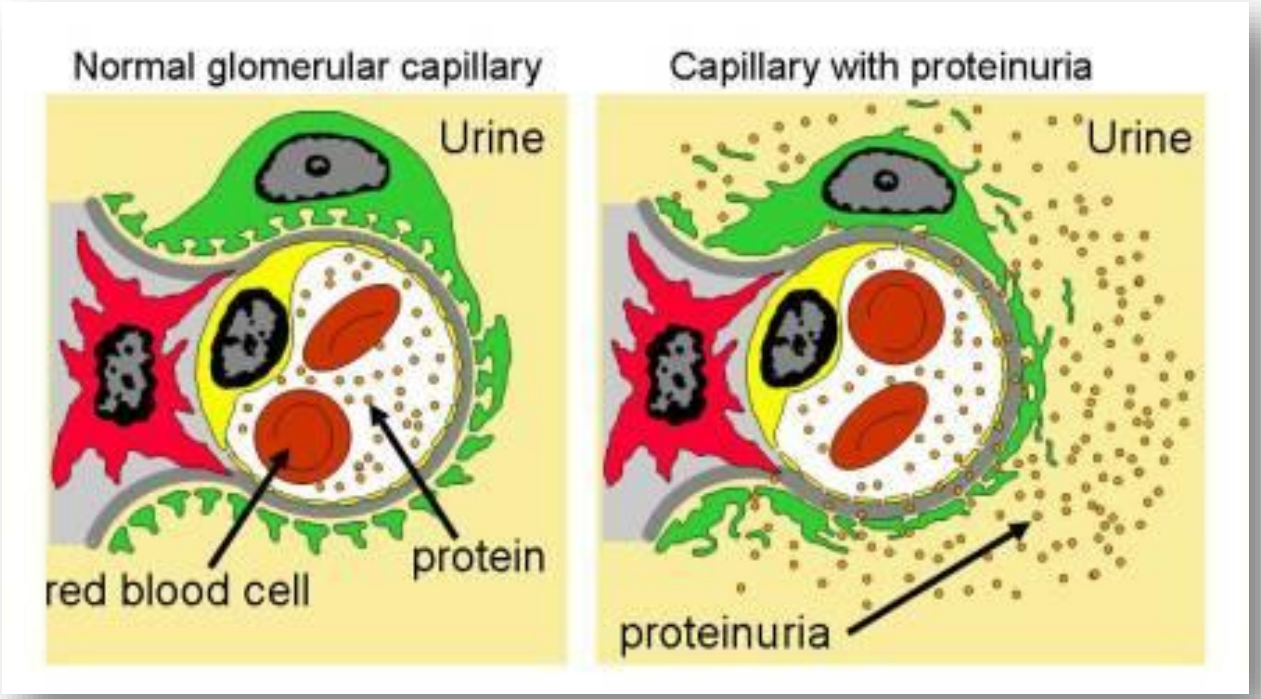
- Methods of assessment:
  detection (turbidity)
  qualitative (electrophoretic)
  quantitative (24 hours urine)

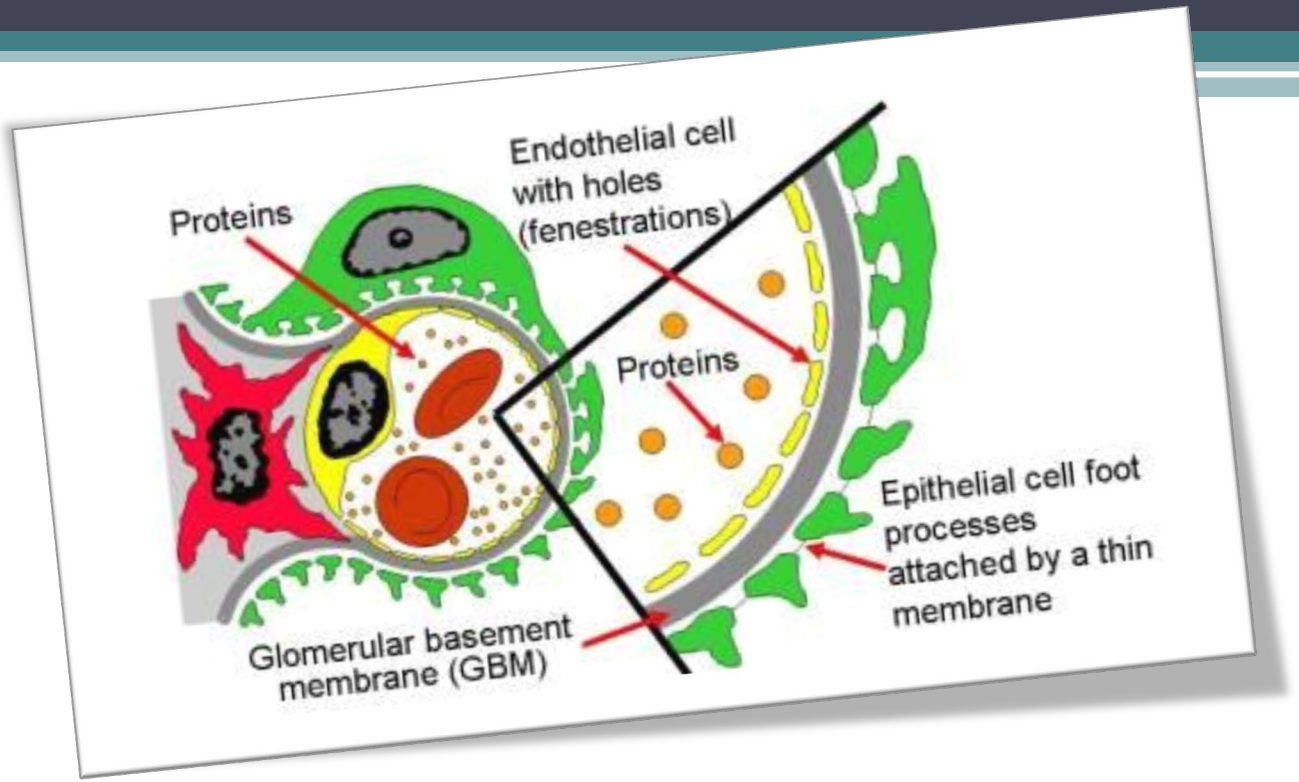
Classification of proteinuria

Immunochemical point of view:

- selective proteinuria (albumin> 85%, globulune <15%)
- non-selective (global)
- tubular (electrophoretic globulin trace)
- paraproteins

- The normal filter of the vessel wall keeps blood cells and most proteins in the blood. In patients with proteinuria, protein leaks across the wall into the urine.





Drawn below are the barriers that keep protein and blood cells out of the urine. These are the endothelial cell, basement membrane and epithelial cell (podocyte). The epithelial cell (podocyte) seems to be most important. Injury to these barriers causes protein and blood to leak into the urine.

Classification of proteinuria

- **Depending on the etiopathogenesis**
- **Prerenal (overload)**
  - normal protein (Hb, Mb, amylose)
  - with abnormal prot. (Bens-Jons, chain H) etiopatogenic
- **Renal**
  - glomerular
  - permanent (in all urine samples)
  - intermittent (functional) occur in some samples, after some maneuvering, are benign, transient and are given by changes in renal hemodynamics:
    - effort proteinuria
    - orthostatic proteinuria
    - proteinuria in febrile diseases
    - proteinuria of stasis in constrictive pericarditis, cirrhosis
    - cyclic proteinuria teenager, proteinuria postprandial
    - neurological proteinuria, proteinuria by physical agents

- **Postrenal** (nefrourologic) very low proteinuria (0.5-1.5g/24) and non-selective due to descuamation and inflammation of connective epithelium (in cystitis, pyelitis, urothelial tumors, renal tract tuberculosis, stones)

PROTEINURIA

- Microalbuminuria of 150-300mg/24h to distribute to patients with diabetes and hypertension is an early sign of kidney damage Proteinuria over 3 g/24 h is likely glomerular

- Microalbuminuria - 150-300mg/24h in patients with diabetes and hypertension early sign of kidney damage
- Proteinuria over 3 g/24h is likely glomerular

The main syndromes in kidney disease
Nephritic syndrome
I. Acute nephritic syndrome

Acute inflammation of renal parenchyma

Acute glomerulonephritis - hematuria, proteinuria, edema, hypertension, ±renal failure .
Ex. poststreptoccocal acute glomerulonephritis, IgA nephropathy

Acute interstitial nephritis : Leucocyturia, reduced and tubular proteinuria, hematuria, ± renal failure

Nephritic syndrome (glomerulus)

- Represents a “first step diagnosis”
- NS may evolve acute or chronic
- It is characterized by:

  -Proteinuria

  -Hematuria

  -Cilindruria - casts: red blood cell casts, granular casts.

  -Edema

  -Hypertension with / without renal dysfunction

Acute nephritic syndrome

- It is characterized by rapid induction of signs of renal dysfunction, with clinical manifestations often very pronounced in a healthy person until then.
- The clinical picture can outline acute nephritic syndrome, isolated or associated with extra renal signs, that integrates clinical nephropathy in a more general context that includes manifestations from other organs.

Acute nephritic syndrome

typical	atypical
- Oliguria - Edema - HTA - Proteinuria - Haematuria - commonly - Acute kidney injury (IRA)	- Acute kidney injury (IRA) or proteinuria / hematuria (isolated) or Isolated acute hypertensi - on

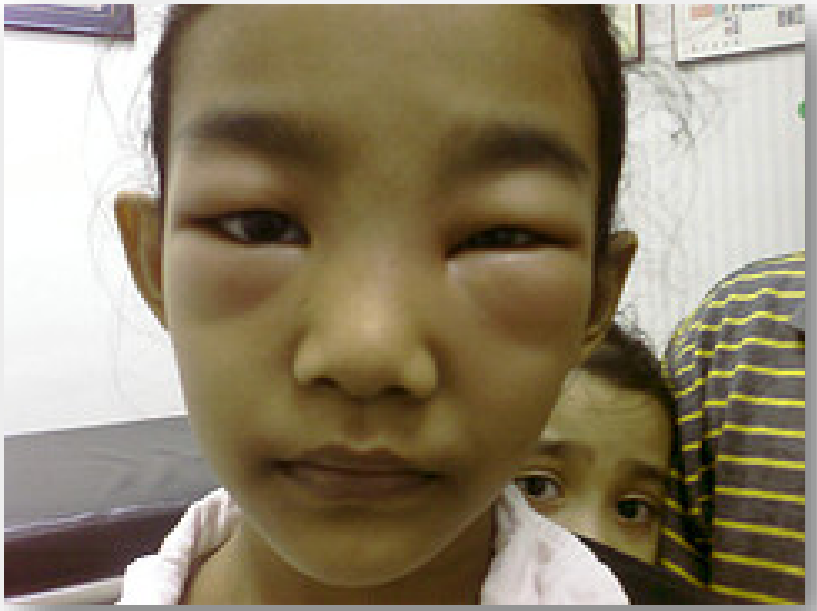
Chronic nephritic syndrome

- Usually, continues the nephritic acute syndrome, rarely is installed from the start with chronic aspect.
- Clincal Characters:
  -evolving long time with polyuria
  -persistent proteinuria, 0.5 - 2 g / day
  -persistent microscopic hematuria
  -granular casts are more frequent
  -edema , present only in exacerbation
  -Hypertension is more frequent and more severe
  -CRI is installed progressively and irreversibly

The nephrotic syndrome

occurs due to increased glomerular capillary wall permeability to protein and is characterized by massive proteinuria (over 3.5 g/24h), with its consequences:

- **hypoalbuminemia,**
- **overall hypoproteinemia ;**
- **hiperlipoproteinemia**
- **(hipercolesterolemia,**
- **hipertrigliceridemia);**
- **Edema etc.**



The nephrotic syndrome

- Associates three fundamental signs
  - **proteinuria over 3.5 g/24 hours (> 2.5 mg / min),**
  - **hypoproteinaemia below 30g / l**
  - **hypercholesterolemia over 3g / l. A persistent proteinuria over 3.5 g/24 h or 2.5 mg / min allows the assertion of nephrotic syndrome**

- If proteinuria is higher, the appearance of clinical manifestations of nephrotic syndrome is earlier
- Resulting hiposerinemia explains most other manifestations, edema is what defines this.

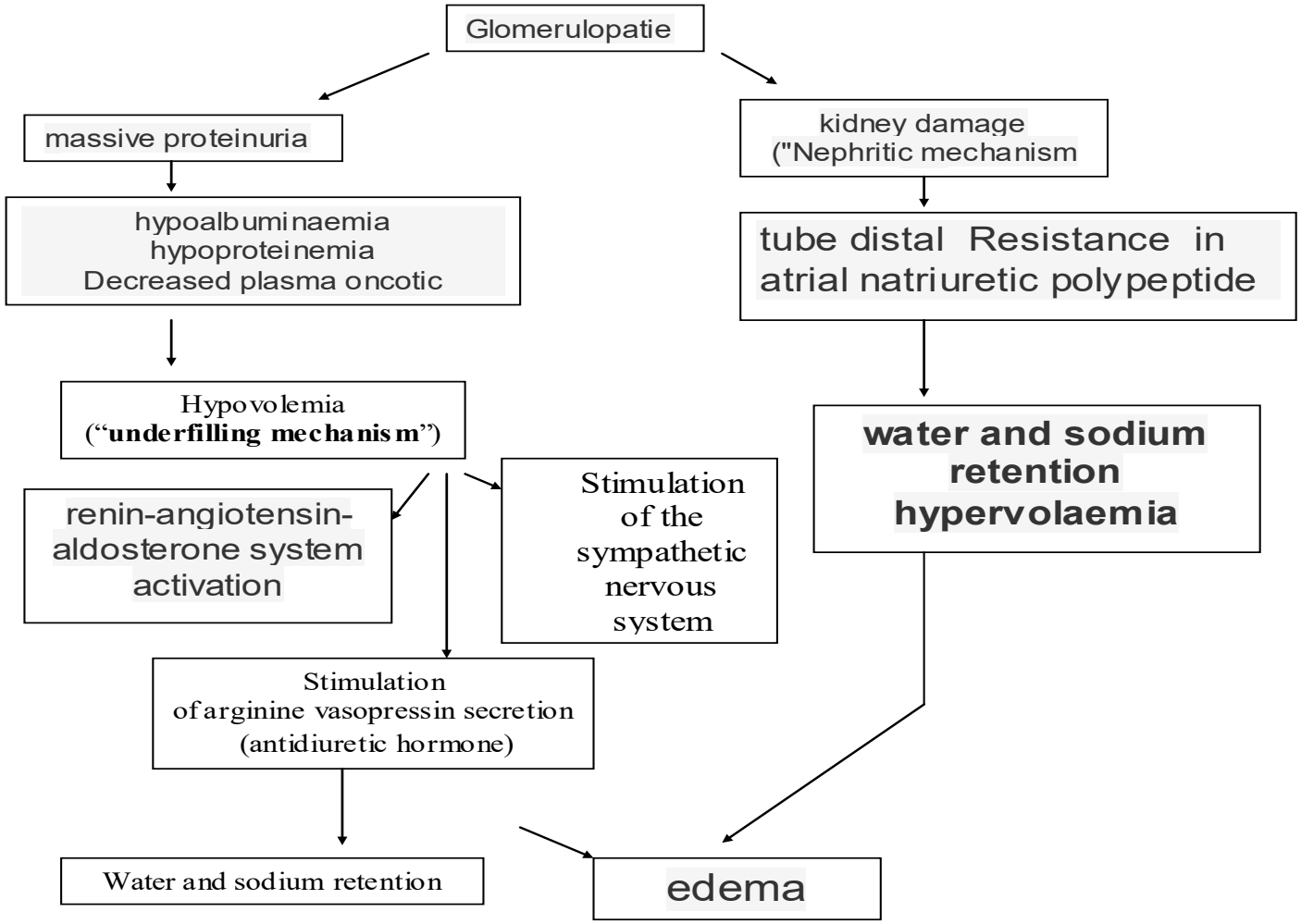
Variants of nephrotic syndrome (NS)

a) **Pure SN** is characterized by:
-absence of macroscopic hematuria
-absence of hypertension
-IRC absence
-high frequency at children

b) **Impure SN** associates to the nephrotic syndrome elements:
-persistent hematuria
-HTA
-BCR
-equal frequency at children and adults

Occurs at patients with systemic vascular disease:
diabetes mellitus, amyloidosis,SLE, Henoch-Schonlein purpura.

Pathogenesis of NS



Nephrotic syndrome (NS)

Etiology / Classification

I. Congenital and hereditary NS

-congenital NS
- family NS
- infantile NS
- NS from hereditary nephropathies (sdr.Alport, Lowe)

II. Primitive or idiopathic NS - appears in the primitive glomerular nephropathies. After histological lesions (Glassock et al.)

1. NS with minimal glomerular minimal (Lipoid nephrosis)
2. Mesangial proliferative GN,
3. NS with focal glomerular sclerosis
4. membranous GN
5. Mezangio-capillary GN: type I, type II
6. Less common lesions
   - -GN with crescent
   - -segmental proliferative and focal GN
   - -unclassified lesions

Nephrotic syndrome (NS)

Etiology / CLASSIFICATION (2)

- III. secondary NS - is a concequence of highlighted etiologic factors
  1. Infectious causes:
  a) bacterial
- (streptococcal β-hemolytic , staphylococcus (GN in endocarditis), and al., the shunt nephritis, parasits, syphilis.
  b) viral (HBV, cytomegalovirus, Epstein-Barr virus, herpes zoster, HIV-1)
  2. Allergy: Insect-venom, reptiles, etc. inhalation of pollen.
  3. Immunization: immunization (DTP), seroterapie.
  4. System diseases: SLE, PAN, rheumatoid arthritis, systemic vasculitis, sd.Goodpasture, etc. sarcoidosis.
  5. Metabolic disease: amyloidosis, diabetes, 1antitripsină alfa- deficit.
  6. Malignancies: Hodgkin d., pheochromocytoma
  7. Drugs and other chemicals: salts of Au, Bi, captopril, mercury, contrast agent, tolbutamide, rifampicin, etc. Interferon.
  8. Other causes: reflux nephropathy, thrombosis vv. renal, reno-vascular HT,sferocitoză, thyroiditis, hyperthyroidism, constrictive pericarditis, IC.

Nephrotic syndrome (NS)

"pure“ Nephrotic syndrome	"impure“ Nephrotic syndrome
Selective type proteinuria ELFO prot. urinary (GM fraction <100 000: albumin,siderofilină, no IgM)	**Non-selective type proteinuria, in extreme cases to the appearance of "serumdiluted" of urine in ELFO**
Microscopic hematuria only at the beginning , mandatory, transitory	**Microscopic hematuria and / or macroscopic than 1 liter after disease debut**
HTA only at the debut, not obligatory, transitory	**Frequent, persistent HTA**
Mandatory Nitrogen retention , only at the debut during oliguria	Persistent nitrogen retention and after resumption of diuresis

Nephrotic syndrome (NS)

clinical-evolutive CLASSIFICATION

"pure“ Nephrotic syndrome	"impure“ Nephrotic syndrome
Good response to corticosteroids (only 5% of children with pure NS are primary corticorezistent)	**Generally responded poorly to corticotherapy Small proportion of patients respond to high doses of cortisone administration and for a long time)**
Favorable prognosis (cure after 1-4 years of evolution in which relapse)	**Poor prognosis (overall trend toward IRC exitus in terminal uremia)**
Histologically: minimal glomerular lesions	**Histologically: glomeruli lesions, focal sclerosis, etc..**
Overlap on the primitive NS	Overlap on the secondary NS

Nephrotic syndrome (NS)

PATHOGENS

I. IMMUNOLOGICAL MECHANISM

a) NS of the CIC disease (mechanism similar to that of RNG) - LES, shunt nephritis, subacute bacterial endocarditis, HVB

b) NS produced by anti-MBG: the rapidly evolving rapidly progressive GN to IRCand terminal uremia exitus in 6 to 12 months (rare in children)

c)NS with allergic reaginic type, with renal relapses seasonal ty pe duringexposure to allergen, with increased serum IgE.

II. TOXIC MECHANISM

the effect of toxic and pathogenic agents local

III. FAILURE MECHANISM SET (SN "idiopathic")

Lipoid nephrosis (NS pure):

Disturbance of cellular immunity resulting in an anomaly of LT-dependent, resulting in release of toxic mediators working for. MBG MBG-factor of the leak.

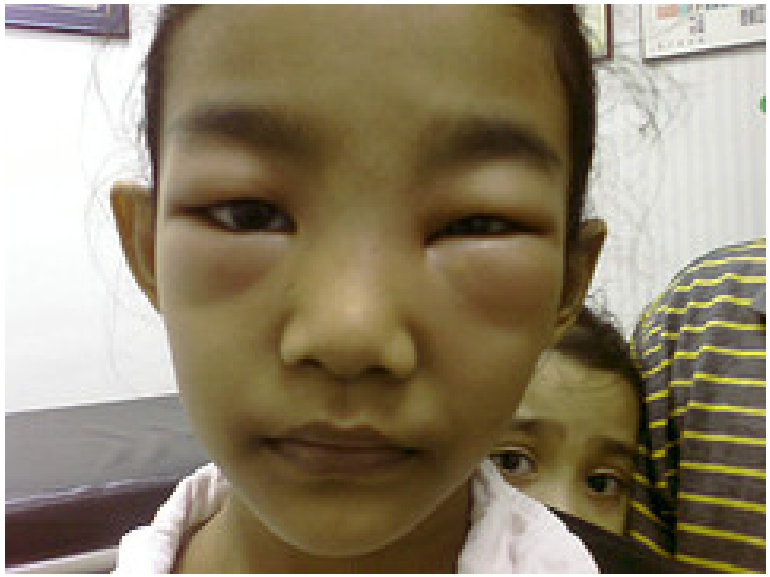
Nephrotic syndrome (NS)

- Pathology
  Macroscopic: Lipoid nephrosis in (NS pure) increased kidney volume, pale appearance (white)
  histopathology:
  a) minimal glomerular lesions - in 77% of cases.
  - In MO: normal optical glomerule
  -In ME: swelling
  of epithelial cells with disruption and merging processespodocitare.
- -
  "Disease with minimal glomerular lesion" Lipoid nephrosis feature a child withreversible changes.
  b) membranous glomerular injury - diffuse thickening of the MBG and the formation of deposits between citoplasmele triangular lamina cells
  and dense-looking "gear wheel"
  -irreversible damage with progression to sclerosis
  c) sclerosing glomerular lesions aspect of systematic sclerosis: glomeruligl obular, with 4-6 lobes in glomerule segmented or diffuse sclerosis.

Nephrotic syndrome (NS)

- **Clinical**
  The maximum frequency between 1 and 1 / 2 years - 4 years, M / F = 2:1
  Circumstances of occurrence: - after an episode of infection (throat, the lowerairways)
  - During a known renal disease
  - After a poisoning
  - No history of pathological
  Onset: insidious, lasting 2-4 weeks.
  - Pallor, loss of appetite, irritability, restlessness, fatigue, low grade fever / fever,diffuse abdominal pain, headache,
  - Installed swelling gradually progressive
  suddenly - in rare cases.

- **Period status:** proteinuria - a cardinal sign
  edema = consequence and expression
  of clinical proteinuria
  1. hidropigen syndrome
  2. urinary syndrome
  3. antibody syndrome
  4. renal function



Nephrotic syndrome (NS)

- 1. Hidropigen syndrome: -
  Renal edema character (facies bufi, maleolare, pretibiale)
  - Fluid in serous effusions (pericarditis, pleural effusion, ascites, hydrocele) form generalized anasarca
  - Skin and white waxy
  - Skin infections in the NIV. eyelids or reg. genital.
  - Loss of appetite / chronic diarrhea (intestinal villous edema)
- 2. Urinary Syndrome:
  - Oliguria:1 - 2 urinary/day, diuresis <250ml/zi retention during HS
  - proteinuria

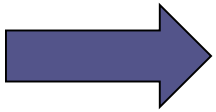
quantitatively important
> 5 -15 g / l

60-90% albumin
α1 and γ globulin
α2 and β globulin

- Normal urine density (Increased amount of protein)
- Urinary sediment: rarely - microscopic hematuria, granular and hyaline cylinders

Nephrotic syndrome (NS)

- 3. Antibody syndrome:
  - Hypoproteinaemia: cardinal sign and binding in NS reach values of 3-5 g% disproteinemie: hypoalbuminaemia hipogammaglobulinemie
    α2 and β hyper globulinemie
  reversal of the albumin / globulin
  - Hyperlipaemia: 10-30g / l
  - Loss of protein
  - Increased hepatic synthesis
  - hypercholesterolemia
  - hipertrigliceridemie
  - Hiper betalipoproteinemie



serum lactescent

4. Renal function: normal or slightly reduced during oliguria
   Other tests: accelerated ESR - reflects hypo / disproteinemia
   ASO and the normal complement

Differential diagnosis of NS

- cardiac edema are cyanotic, latch, cold, sick at the "old" heart with signs of IC.
  liver: clinical hepatomegaly, + / - jaundice,
  stars vascular laboratory signs of liverdistress
  Allergic contact with the allergen /
  post insect bite, clinical signs of allergy.
  hypoproteinaemia: proteinemică malnutrition, urinalysis i s normal, normal or low fat
  mixedemul: appearance characteristic facies, dry skin, infiltrates, macroglossia, TSH↑, T4 and T3↓)
  other renal edema: RNG, acute pyelonephritis, are excluded on the basis
  of: urinesamples and renal exploration;

Nephrotic syndrome (NS)

TREATMENT

1. Hygienic-Dietetic

-bed rest during oedematous and relapse
-desodat diet
-diet:
-Protein: 1 g / kg C / day
-fat - moderate restriction, especially those with high cholesterol and triglyceridecontent
-4-8 g carbohydrate / kg C / day

serotheraipy and vaccinotherapy total contraindication (for 2.5 years after full remission)
avoiding exposure to bad weather (cold, moisture)

Nephrotic syndrome (NS)

TREATMENT

- 2. pathogens:
  Cortisone therapy: Prednisone
  short schedule
  First flare attack therapy: 2 mg / kg C / day, max. 80 mg / day in 4 divided doses,four weeks
  Consolidation therapy: 2 mg / kg C / day, max. 80 mg / day in single dose in the
  morning, another alternative under four weeks and then may stop suddenly
  Relapse therapy (presence of proteinuria + + or higher in a patient withoutproteinuria), the resumption of corticosteroid therapy and the dose rate used in the attack

Nephrotic syndrome (NS)
TREATMENT

- Cortisone therapy

long-term scheme

-Attack therapy: 2 mg / kg C / day - time = 8 weeks (until the disappearance ofproteinuria 14 consecutive days)
-Consolidation therapy: single dose and treatment duration = AC 2 months (minimum) to gradually reduce the dose to 3-4 weeks total duration = 6-12 months
-Relapse therapy (proteinuria = + + in a patient without proteinuria) - Resumption ofcorticosteroid dose therapy pace attack used by protein uria disappears 3 consecutive days, then resume treatment building.

Nephrotic syndrome (NS)
alternative TREATMENT

- **Cytostatic therapy:**

Major indications

- NS corticosensibile frequently relapsed
-NS relative steroid sensitive phenomena corticointolerance

Contraindications

- Secondary forms, steroid-resistant
- Congenital and familial forms
- Cortico-sensitive forms without corticointolerance

Regimens

Cyclophosphamide 2.5-3mg / kg / day IV - 3 weeks (+ / - prednisone)
Levamisole 2.5 mg / kg / dose Alternative 4 - 12 months
Cyclosporine 5-7 mg / kg / day 4 weeks - 8 months
Other: chlorambucil, azathioprine, methylprednisolone iv

Nephrotic syndrome (NS)
TREATMENT

3. symptomatic

- diuretics -
  the need, if only edema massive, debilitating
- serumalbumină - only to cases that develop symptomatic hypovolaemia
- puncture evacuees in need (in case of important collections)
- HTA trtamentul
- Cardiac insufficiency - treat. ECG surveillance.

Nephrotic syndrome (NS)
TREATMENT

Adjuvant for sec. ef. prevention of cortisone therapy

- Fluid restriction (after restoring fluid balance) lichide/24 total hours = 250 ml/m2/zi + diuresis previous day
- -Na + restriction (maximum contribution 1g/24h)
- K supplementation (1-2 g KCl / day) for diuretics that induce hipoK
- Administration of lactate Calcium is 1-2 g / day
- Administration of gastric dressings (Dicarbocalm etc.) High
- protein diet, normocaloric, moderate hipoglucidic, hipol ipidic

Nephrotic syndrome (NS)

EVOLUTION

a) Healing: - Recurrent flare-ups in 2 to 4 years, with complete healing. is the most commonsituation at children

b) Incomplete remission

c) chronicity: slow evolution in recurrent flare-ups over the years, gradual onset IRC (rare situation to child)

e) Installation IRA (very rare at children)

Nephrotic syndrome (NS)

COMPLICATIONS

a) Related to evolution of the disease

Intercurrent infections (pneumococcus, v. measles, varicella)

- Massive fluid retention (ascites, hydrothorax, compression)
- Abdominal pain crises, crises of tetanus
- Trombembolii (by blood hypercoagulability)

b) related to cortisone therapy and immunosuppressants

! Corticosteroid therapy: growth retardation / puberty, obesity / diabetes, striae, hirsutism, plethoric facies, hypertension, hypocalcemia, osteoporosis, decreasedresistance to infection, suppression of CSR

! Chemotherapy: marrow depression (! Leukopenia), alopecia, digestive disorders, infertility, etc.


Thank you for attention