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Учреждение образования  
«Гомельский государственный медицинский университет»

Кафедра патологической физиологии  
Обсуждено на заседании кафедры  
Протокол №7 от 30.08.2017

**МЕТОДИЧЕСКАЯ РАЗРАБОТКА**

Для проведения занятия со студентами  
3 курса ФПСЗС, обучающихся на английском языке  
по патологической физиологии

Тема: **Патофизиология почек**

Theme: **Pathophysiology of kidneys**

Время 3 ак. часа

**1.Actuality of the theme.** The renal failure depend to severe pathological states. The disorder of constance of internal environment of organism, which one thus arise, often demand emergency treatment. To the most often causes, which one cause disturbance of functions of kidney, the disorder of their blood supply, infections deseases, intoxication, autoallergy damages, violation of outflow of urine concern. Knowledge of etiology and pathogenesis of kidney diseases, mechanisms of disturbance, which one arise in renal failure, are necessary for selection pathogenetic based methods of preventive maintenance and treatment.

**Learning goals of the lesson:** to study main typical pathological and compensatory-adaptive processes occurring in urinary system.

**Educational goals of the lesson:** formation of scientific outlook and theoretical basis of future specialists on the basis of fundamental knowledge and the latest achievements of pathological physiology.

**Objectives of the lesson:**

1. To know causes and mechanisms of disturbance of filtration, reabsorption and secretory functions of kidneys, as well as pathogenesis of main syndromes arising from their defeat.
2. To be able to use data on the quantitative and qualitative composition of urine and blood to explain a pathogenesis of main pathological processes in kidneys and resulting homeostatic disorders.

**To repeat the following questions from related disciplines to ensure absolute mastery of the material:**

1. Structure of nephron (histology, cytology, embryology disciplines).
2. Main factors of neurohumoral regulation of filtration, reabsorption and secretion (normal physiology discipline).

**Control questions of the lesson:**

1. Violations of glomerular filtration: types, causes, mechanisms and manifestations.
2. Disturbances of tubular functions: types, causes, mechanisms and manifestations.
3. Renal and adrenal syndromes.
4. Glomerulopathies: etiology, pathogenesis, morpho-functional manifestations, complications, outcomes.
5. Nephrotic syndrome: types, mechanisms of development.
6. Pyelonephritis: etiology, pathogenesis.
7. Acute and chronic renal failure: etiology, pathogenesis, stages, complications and outcomes.
8. Nephrolithiasis: etiology, pathogenesis, complications.

**Calculation of study time**

Total study time 3 ac.hours

№ п/п	Contents	Calculation of study time
1.	Introduction. Motivational characteristic of the theme	3 minutes
2.	Written control of students on the topic of the lesson	15 minutes
3.	Interviews with students about the topic of the lesson	60 minutes
4.	Self-managed student work	15 minutes
5.	Summing up the results of the lesson	5 minutes
6.	Decision of situational tasks	20 minutes
7.	Task for the next lesson	2 minutes

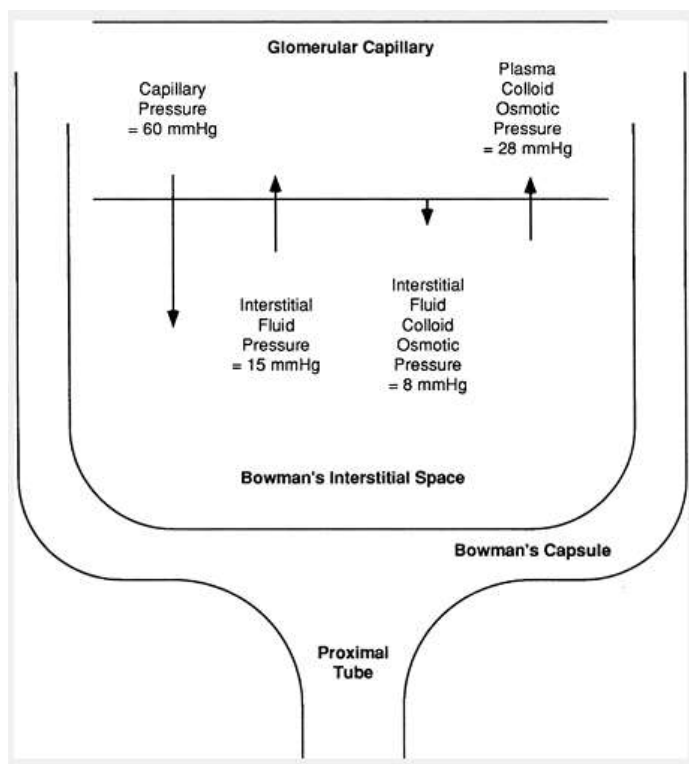
## Additional materials

### Anatomy of kidneys

The anatomic unit of kidney function is the **nephron**, a structure consisting of a tuft of capillaries termed the **glomerulus**, the site at which blood is filtered, and a **renal tubule** from which water and salts in the filtrate are reclaimed. Each human kidney has approximately 1 million nephrons.

A glomerulus consists of an **afferent** and an **efferent arteriole** and an intervening tuft of capillaries lined by endothelial cells and covered by epithelial cells that form a continuous layer with those of **Bowman's capsule** and the renal tubule. The space between capillaries in the glomerulus is called the **mesangium**. Material comprising a basement membrane is located between the capillary and the epithelial cells.

The renal tubule itself has a number of different structural regions: the **proximal convoluted tubule**, from which approximately 80% of the electrolytes and water are reclaimed; the **loop of Henle**; and a **distal convoluted tubule** and **collecting duct**, where the urine is concentrated and additional electrolyte and water changes are made in response to hormonal control.



**Forces favoring filtration and reabsorption across the glomerular capillary**

### Functions of kidneys

1. excretory
2. homeostatic
3. control of arterial blood pressure and blood volume
4. erythropoiesis stimulation
5. antigen presentation
6. control of blood coagulation
7. hormone degradation: insulin, glucagon, gastrointestinal hormones and others

### Creatinine filtration rate (CFR)

Creatinine filtration rate characterizes excretory and filtration renal functions.

Creatinine is usually present in blood of man. It is excreted only by filtration in man over 11 years old (it is not reabsorbed or secreted in tubules). It means that 100–120 ml of blood is filtered from creatinine by kidneys in 1 minute, and at the same time, 100–120ml of primary urine is formed.

Impairment of filtration and excretory renal function is characterized by low parameters of CFR.

Compounds that are also used in clearance test: exogenous inulin, hyposulphite — 100–120ml/min, urea (55–70 ml/min), but it is partially reabsorbed in renal tubules.

Clearance index (amount of blood (ml) which is completely cleared from dif. substances per minute)

$$C = U \times V / P$$

C – clearance of substance (ml/min)

U – concentration of substance in urine (mg/ml)

V – diuresis (ml/min)

P – concentration of substance in plasma (mg/ml)

### Diuresis impairments

N – 1000–2000 ml per day (children =  $600 + (n-1) \times 100$ , n – years) (4–6 urine output)

**Polyuria** — more than 2500–3000ml per day

**Oliguria** — less than 500ml per day (<24 ml/kg/day)

**Anuria** — less than 100–50ml per day

**Nicturia** – night diuresis > 50% (norm in day 65-80% from total volume of urine)

**Polakuria** is increasing of frequent of urination.

Relatively density of daily urine (1,002-1,035, often – 1,012-1,026 g/ml)

**Hyperstenuria** – density of the urine more than 1,030-1,035

**Hypostenuria** – density of urine less than 1,002-1,012

**Isostenuria** – small changing density of urine (< 1,010) during day.

**Proteinuria** is presence of proteins in urine.

**Hematuria** is appearance of erythrocytes in urine (>3 Er for female).

**Hemoglobinuria** is a presence of Hb in urine.

**Cylindruria** is appearance in urine of cylinders. Cylinders show by itself molds of kidney tubules. They will appear at the damage of epithelium of tubules and consist of coagulative albumen and lost cells (leucocytes and epytheliocytes). In dependence on a structure distinguish hyaline, grainy (granular), cereous (wax-like), lipoid (fatty), hemoglobin, leukocytic, erythrocytic and epitheliums cylinders.

**Leucocyteuria** is appearance in urine of leucocytes (for female > 5, for man >1 in eyeshot). Pyuria is the state, when the amount of lekocytes in eyeshot is more than 100 cells. Principal reason of leukocyteuria is inflammatory processes in kidneys and urinary tracts.

**Crystalluria** in a surplus amount: crystals of urinary acid as yellow, oxalate, urates, phosphates, crystals of oxalic calcium, crystals of cystine crystals of tyrosine, crystals of cholesterol.

### **Common mechanisms of occurrence and development of renal disease**

- Violations of glomerular filtration rate (increasing or decreasing volume of glomerular filtrate)
- Violations of tubular reabsorption
- Violations of secretion

#### **Decreasing volume of glomerular filtrate**

##### **Renal factors:**

- ↓EFP:
  - ✓ ↓functioning glomeruli (necrosis, CGN, fibrosis) ↓permeability of filtration barrier: thickening or reorganization of BM (CGN, DM, amyloidosis).
  - ✓ sclerotic changes in afferent arterioles and interlobular vessels
- increase in pressure in the Bowman's capsule due to higher intrarenal pressure (interstitial edema or obturation of tubules and urinary tract)

##### **Extrarenal factors:**

- ↓ systemic blood pressure (HF, vascular insufficiency, blood loss, dehydration → ↓EFP)
- ↑plasma oncotic pressure (↑concentration of proteins (MM), the injection of protein drugs, hemoconcentration)

#### **Increasing volume of glomerular filtrate**

- ↑ EFP:
  - ✓ ↑ tone of SMC of efferent arterioles (ADH, catecholamines, angiotensin)
  - ✓ ↓ tone SMC of afferent arterioles (under the influence of kinins, Pg, etc.)
  - ✓ hyponkemia of blood (hepatic failure, starvation , prolonged proteinuria).
- ↑ permeability of the filtration barrier: loosening of BM by BAS (histamine, kinins, hydrolytic enzymes).

#### **Violations of tubular reabsorption**

- enzymopathies
- defect of transepithelial transport of substances (AA, albumin, glucose, lactate)
- membranopathies of epithelium and BM of renal tubules

#### **Violations of secretion**

- gene defects lead to cystinuria, aminoaciduria, phosphaturia, renal diabetes, renal acidosis

### **Classification of renal pathology**

#### **Etiological:**

- acquired
- hereditary
- infectious
- noninfectious
- ✓ physical (traumatic, vascular (ischemic), IR)
- ✓ chemical (nephrotoxic poisons, metabolic)
- ✓ biological (immunological, allergen)

#### **Topographic:**

- prerenal
- renal
  - ✓ glomerular
  - ✓ tubular
  - ✓ tubulointerstitial
- postrenal

#### **Pathogenetic:**

- primary (enzymo-, membranopathy, cystic disease, dysplasia)
- secondary

#### **By volume of damage:**

- total
- partial
  - ✓ filtrative
  - ✓ reabsorbtive
  - ✓ secretive
  - ✓ incretive

#### **Clinical:**

- acute
- chronic

#### **Etiology**

- Exogenous factors:
  - ✓ traumatic (trauma, crushing injury, IR, cold, burns)
  - ✓ toxic (salts of heavy metals, sulfanilamids, antibiotics, corticosteroids, mushroom, snake venom, pesticides)
  - ✓ infectious
  - ✓ immune
- Endogenous factors:
  - ✓ Vascular: systemic (shock, AH) or local (ischemia, thrombosis, embolization) disorders
  - ✓ Hormonal and neural disturbance of renal functions
  - ✓ Genetic
  - ✓ Traumatic (mechanical injury by stones)
  - ✓ Toxic (products of dismetabolism in diabetes mellitus, amyloidosis, hepatic insufficiency, peritonitis, pathological pregnancy, gout)
  - ✓ Immune

#### **Prerenal factors**

- systemic disorders of blood circulation ( $\downarrow$ BP or  $\downarrow$ BCV)
- dehydration, hemoconcentration
- AH
- acute systemic intoxication (crush syndrome, burns)
- massive hemolysis of erythrocytes

#### **Renal factors**

- toxic
- infectious
- immune

- vascular (local disorders of blood supply)

### **Postrenal factors**

- obstruction of the urine ways (calculi, tumors)
- retention of urine (neural disorders)

### **Clinical Syndromes**

- nephritic syndrome
- nephrotic syndrome
- urinary syndrome

### **Nephrotic syndrome**

Massive proteinuria (>3g/day)

Hypo- , dysproteinemia (albumin < 30g/l)

Hyperlipidemia

Hypercholesterolemia

Edema, hydropsy of serosal cavity

### **Types of nephrotic syndrome**

- Primary (renal disease)
  - ✓ lipoid nephrosis
  - ✓ membranous GN
  - ✓ Focal-segmented glomerulosclerosis
  - ✓ membranous-proliferative GN
- Secondary
  - ✓ systemic disease (SLE, DM, vasculitis)
  - ✓ infectious
  - ✓ drugs (NSAIDs)

### **Mechanisms of nephrotic syndrome**

#### ***Proteinuria***

- a decrease in work of filtration barriers of glomeruli (an increase in basement membrane permeability results in glomerulus injury)
- changes in capillaries of glomerular filter
- a decrease in tubular epithelium ability to reabsorb protein (it results from the secondary injury)
  - loss of albumins, Ig, some factors of blood coagulation, erythropoietin, protein-hormone transmitters, inhibitors of blood coagulation (antithrombin III, protein C, S)
  - loss of proteins with urine results in normochromic normocytic anemia
  - endocrine pathology

#### ***Hypoalbuminemia***

- loss of albumins with urine prevail over protein synthesis
- an increase in albumin synthesis in liver
- compensatory decrease in albumin catabolism

#### ***Hyperlipidemia***

- stimulation of very low-density lipoprotein synthesis
- an increase in mevalonic acid synthesis (precursor of cholesterol synthesis)
- a decrease in activity of lipoproteinlipase in tissues results from an increase in plasma free fat acid level
  - a decrease in activity of LCAT results from a decrease in albumin level and an increase in lysolipid level (lysolipid is an inhibitor of LCAT)
  - an increase in cholesterol and triglycerides in plasma
  - an increase in low-density lipoproteins and very low-density lipoproteins

#### ***Edema***

Proteinuria results in hypoalbuminemia (a decrease in colloid osmotic pressure) → hypovolemia (a decrease in in-vascular blood volume) → a decrease in renal blood flow. The decrease in renal blood flow

results in activation of rennin synthesis → angiotensin II synthesis → increased aldosterone secretion → an increase in ADH level → an increase in water reabsorption, edema. At the same time, a decrease in renal blood flow results in a decrease in glomerular filtration, activation of aldosterone secretion causes sodium reabsorption → an increase in water reabsorption, edema.

### Complications of nephrotic syndrome

- increased sensitivity to infections, firstly to in-capsulated microorganisms (pneumococci), results from the loss of Ig and complement
- an increased risk of thromboembolism (thrombosis of profound veins of low extremities results in pulmonary embolism)
- a decrease in blood coagulation is caused by the loss of antithrombin III and other inhibitors and blood coagulation controls.

### Nephritic syndrome

- oliguria
- hematuria
- azotemia
- hypertension
- mild edema

### Urinary syndrome

- proteinuria
- hematuria
- leucocyteuria

### Acute diffuse glomerulonephritis

It is autoimmune mediated inflammation of glomerular apparatus and subsequently fibrotic events

#### Etiology:

- Infectious agents: **mostly  $\beta$ -Hemolytic Str.group A type 12**, pneumo-, meningococcus, salmonella, Treponema pallidum, viruses (hepatitis, EBV), malarial plasmodium, toxoplasma
- Noninfectious agents:
  - ✓ autoaggressive and / or cross-Ab
  - ✓ CIC in blood
  - ✓ foreign proteins (vaccines, serum, proteins of tumor cells or damaged tissues)

#### Pathogenesis

- Formation of Ab to Str. Ag
- Ag of BM is similar to H.Str. Ag
- AntiStr.Ab denature a proteins of Str. and BM
- Direct damage of nephron structures by Str. toxins → ↑AutoAb
- AutoAb potentiates kidney damage

### Chronic diffuse glomerulonephritis

#### Etiology:

- Infectious agents (bacteria, viruses, plasmodium)
- Non-infectious factors (Ag of tumors (lung, stomach, kidney), Ag resulting from massive tissue damage (burn disease, crushing tissues))

It is an outcome of the acute glomerulonephritis in 10–20% of cases, and it is primary chronic in 80–90% of cases.

#### Pathogenesis

Chronic, autoimmune damage of glomerular BM. Intracapillary damage is accompanied by the lesion of tubular epithelium and interstitial tissue.

## **Pyelonephritis**

It is a group of syndromes (diseases) caused by microbes and characterized by the development of inflammatory process in the renal pelvis and renal interstitium.

### **Etiology:**

In more than 80 percent of cases of acute pyelonephritis, the etiologic agent is *Escherichia coli*. Other etiologic causes include aerobic gram-negative bacteria, *Staphylococcus saprophyticus*, and enterococci. Patients who have diabetes mellitus tend to have infections caused by *Klebsiella*, *Enterobacter*, *Clostridium*, or *Candida*.

Most occur secondary to bacterial ascent through the urethra and urinary bladder. In men, prostatitis and prostatic hypertrophy causing urethral obstruction predispose to bacteriuria. Hematogenous acute pyelonephritis occurs most often in debilitated, chronically ill patients and those receiving immunosuppressive therapy. Metastatic staphylococcal or fungal infections may spread to the kidney from distant foci in the bone or skin.

### **Pathogenesis:**

- The microorganisms enter in the kidney → cause inflammation of the mucous membranes of the calyx, pelvis, and / or in the interstitium
- Generalization of infection is associated with the penetration of microbes in the tubules and glomeruli (developing glomerulonephritis)
- Infections often result in necrotic areas formed mucosa and renal abscess. Tubule epithelium can undergo degradation. Tearing away the dead skin cells causing obstruction of the lumen of tubular cell detritus.
- These changes are accompanied by abnormal processes of filtration, reabsorption and secretion.

## **Acute renal failure (ARF)**

Acute renal failure is a clinical syndrome resulting in the abruptly decrease in glomerular filtration and kidney failure.

### **Etiology:**

- prerenal (functional):
  - ✓ hypotension
  - ✓ decrease in intravascular volume depletion
  - ✓ ionic-water imbalance: cardiac failure, cirrhosis
- intrarenal (structural):
  - ✓ post-ischemic tubular necrosis
  - ✓ toxic ischemic tubular necrosis (heavy metals, ethylene glycol, insecticides, poison mushrooms, carbon tetrachloride)
- postrenal (obstruction):
  - ✓ intra-renal factors: salt dropout in renal tubules
  - ✓ extra-renal factors: large stones or blood clots in ureters, bladder obstruction, urethra obstruction

### **Pathogenesis**

The leading mechanisms: intubular obstruction by necrotic cells, backward glomerular filtrate flow through the tubular epithelium ruptures.

### **ARF stages:**

1. Initial (1-2 days): intoxication, pain, nausea, decrease in arterial blood pressure, tachycardia, pallor. Absence of azotemia.
2. Oligo-anuria (7 days): diuresis 200-250 ml / day, increase of nitrogenous wastes in the blood, uremic intoxication, metabolic acidosis, hyperhydration, hyperpotassemia, hypocalcemia, anemia, hypertension, azotemia
3. Poliuria (up to 2 weeks): lack of urinary concentrating ability, dehydration, hypocalcemia, decrease in azotemia level
4. Clinical recovery (several months, year). Azotemia missing.

## **Chronic renal failure (CRF) (Chronic kidney disease, CKD)**

It is a clinical syndrome resulting from irreversible progressive nephron destruction and a decrease in number of functioning nephrons.



## Etiology:

- Primary glomerular disease: Acute glomerular disease including rapidly progressive glomerulonephritis, chronic glomerulonephritis
- Primary tubular disease: Chronic hypercalcaemia, chronic hypokalemia, heavy metal poisoning like lead and cadmium.
- Vascular disease: ischemia of the kidneys due to congenital or acquired renal artery stenosis; accelerated or malignant hypertension, hypertensive nephrosclerosis
- Infection: chronic atrophic pyelonephritis (reflux nephropathy), tuberculosis.
- Obstruction: renal stones, retroperitoneal fibrosis, prostatomegaly, urethral strictures and tumors.
- Vasculitis: systemic lupus erythematosus, polyarteritis nodosa, Henoch-Schonlein nephritis, scleroderma, Wegener's granulomatosis
- Metabolic renal disease: Diabetes mellitus, amyloidosis, analgesic nephropathy, gout, primary hyperparathyroidism and milk alkali syndrome.
- Congenital abnormality: hypoplasia of kidneys, reflux nephropathy, medullary cystic disease, polycystic kidneys.
- Unknown cause

## Pathogenesis

The result of diffuse renal disease, the death of a large number of nephrons → slowly progressing → reduced filtration and concentration functions of renal → hypoisostenuria → toxic products are distinguished by: GIT, the exhalant air, salivary gland

## Stages

Stage	GFR*	Description
1	90+	Normal kidney function but urine findings or structural abnormalities or genetic trait point to kidney disease
2	60-89	Mildly reduced kidney function, and other findings (as for stage 1) point to kidney disease
3A 3B	45-59 30-44	Moderately reduced kidney function
4	15-29	Severely reduced kidney function
5	<15 or on dialysis	Very severe, or <b>endstage</b> kidney failure (sometimes call <b>established renal failure</b> )

GFR (glomerular filtration rate) values are normalized to an average surface area (size) of  $1.73\text{m}^2$

## Manifestations:

### Early chronic renal insufficiency (Stages 1 to 2)

- Physical symptoms: usually few or absent.
- Blood: mainly a slightly elevated serum creatinine. By the time serum creatinine is elevated, the person may already have lost 50% of kidney function. Anemia may rarely occur at this stage.
- Urinalysis: proteinuria and/or hematuria.
- Blood pressure: some people start having high blood pressure even in early chronic renal failure.

### Advanced chronic renal insufficiency (Stages 3 to 4)

- Physical symptoms: may begin to experience one or more of the following symptoms:
  - ✓ tiredness or fatigue
  - ✓ puffiness or swelling (obvious in the hands or feet and ankles, but the puffiness will often first be seen around the eyes).
  - ✓ back pain. Usually felt as a dull ache anywhere in the mid-to-lower portion of the back, on one side or the other - this is sometimes referred to as flank pain, or loin pain)
  - ✓ pararexia
  - ✓ digestion: poor digestion (varying degrees of gastroparesis)
- Serum creatinine will be higher (indicating less than 30% kidney function)
- Urine: changes in urination (amount, color, frequency). Urine may in fact look exceptionally clear at this point, rather than abnormal. Previously high proteinuria and/or hematuria may actually improve
- Blood pressure: hypertension

### End-stage renal failure (or late chronic renal insufficiency)

- anemia (may begin earlier than this), easy bleeding and bruising
- headache, fatigue and drowsy feeling, weakness
- mental symptoms such as lowered mental alertness, trouble concentrating, confusion, seizures
- thirst, nausea, vomiting, hyporexia, poor digestion (varying degrees of gastroparesis), diarrhea
- muscle cramps, muscle twitching, numb sensation in the extremities
- itchy skin, itchy eyes, skin color changes (grayish complexion, sometimes yellowish-brownish tone)
- swelling and puffiness
- difficulty breathing (due to fluid in the lungs, anemia)
- hypertension
- decreased sexual interest, changes in menstrual cycle (and difficulty getting pregnant)
- nocturia, oligo-, may be polyuria (water as urine, but not wastes)

### Uremia

It is a clinical syndrome of progressive renal failure, characterized by a variety disorders of metabolism and function of many organs.

#### Clinical manifestations:

**Early symptoms are non-specific:** weakness, fatigue, and insomnia

**Digestive system** (anorexia, nausea, vomiting, diarrhea, glossitis, stomatitis, colitis, gastro, hepatitis, ulceration, is largely due to the excretion of nitrogenous wastes through it)

**Cardiovascular system** (AH, HF, arrhythmia, pericarditis, cardiac arrest)

**Respiratory system** (shortness of breath, coughing, pulmonary edema, Kussmaul breathing)

**System of blood** (anemia, leukocytosis, thrombocytopathy)

**Hemostasis** (bleeding into the skin, from the mucosa of GIT, uterine, nasal bleeding)

**Immune system** (inhibition of humoral and cellular immunity, decreased resistance to infections)

**Skin** (yellowish color, a film of urea on her, scratches, petechial rash)

**CNS, PNS** (headache, memory loss, confusion, mental depression, coma, convulsions, tremor, muscle twitching, itching, polyneuritis)

**Osteoporosis, osteomalacia**

### Uremic toxins

- urea and its metabolic products, aminoethanamide, dimethylamine
- parathyroid hormone. High level if parathyroid hormone is observed in chronic renal failure → accumulation of calcium in cells → dissociation of oxidation and phosphorylation, ATP deficiency, impairments of energy supply
- inadequate microelement concentration in blood, intercticium and cells ( $Mg^{2+}$ ,  $Zn^{2+}$ ,  $Cu^{2+}$ ,  $Cr^{2+}$ )

### Tubulopathy

It is a disease caused by disturbance of transport functions of epithelial tubules in the absence or qualitative changes of transport proteins, certain enzymes, receptors for hormones or degenerative processes in the wall of the tubule.

#### Classification:

##### By etiology:

- primary (hereditary)
- secondary (acquired)

##### By localization of defect:

- proximal:
  - ✓ phosphaturia (absence of a transport protein for phosphate: hypophosphatemia, rickets changes in the bones (hypophosphatemic vitamin D-resistant rickets)).
  - ✓ renal glycosuria (↓renal threshold for Gl by reducing the maximum capacity of the tubules to reabsorb it. Gl in blood N or ↓)
  - ✓ hyperaminoaciduria (absence of transport carrier-proteins for reabsorption of AA)
  - ✓ proximal renal acidosis
- distal:
  - ✓ renal water diabetes (lack of response to ADH of kidney → violation of water reabsorption in the distal tubules and collecting ducts. Symptoms: polyuria (up to 30 l / day), polydipsia, kidneys lose their

ability to concentrate urine, relative density <1005 hyposthenuria)

- ✓ pseudo hypoaldosteronism
- ✓ distal renal tubular acidosis

### **Nephrolithiasis**

The main pathogenic chain is a uric acid stone formations in the urinary system.

- It is observed in 1–3% of population
- It occurs after the inflammatory diseases in 30–40% of cases
- It is observed in 20–50-year-old man

*Its occurrence is promoted by:*

- the present-day conditions (physical inactivity results in calcium phosphate metabolism impairments), nutrition character (presence of proteins in food),
- the predisposing factors (age, sex, race, profession, climate, geographic conditions, housing conditions, genetic factors),
- factors of local character (infections of urinary system, pH of urine, urine relative density, anatomic and pathologic changes in the upper urinary tract resulting in the obstruct urine outflow, metabolic and vascular impairments in organism and kidney)

Mineralogical classification of stones: calcium stones, uric acid stones, protein stones (cystinuria), struvite stones

### **Questions for self-control of knowledge:**

1. What are main functions of kidneys?
2. What are pre-renal, renal and postrenal causes of renal dysfunction?
3. What is mechanism of kidney "activation" in hypertension?
4. Give definition of "nephrotic syndrome". What are its manifestations?
5. What are infectious-allergic mechanisms of development of acute diffuse glomerulonephritis?
6. Give evidence in favor of immuno-allergic mechanism of development of acute diffuse glomerulonephritis.
7. What are mechanisms of acute renal failure?
8. Describe main pathological links of chronic renal failure.
9. Describe main disorders of excretory renal function.

### **Tasks for self-managed student work:**

1. Role of immune system in development of kidney disease.
2. Berger's Disease
3. Mechanisms of uremia.
4. Principles of therapy of kidney pathology

### **Literature**

#### **Basis literature:**

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4. Научная электронная библиотека eLIBRARY.RU [Электронный ресурс] / Научная электронная библиотека. – М., 2005. – Режим доступа: <http://www.elibrary.ru>. – Дата доступа: 26.08.2017.

#### **Compiler:**

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