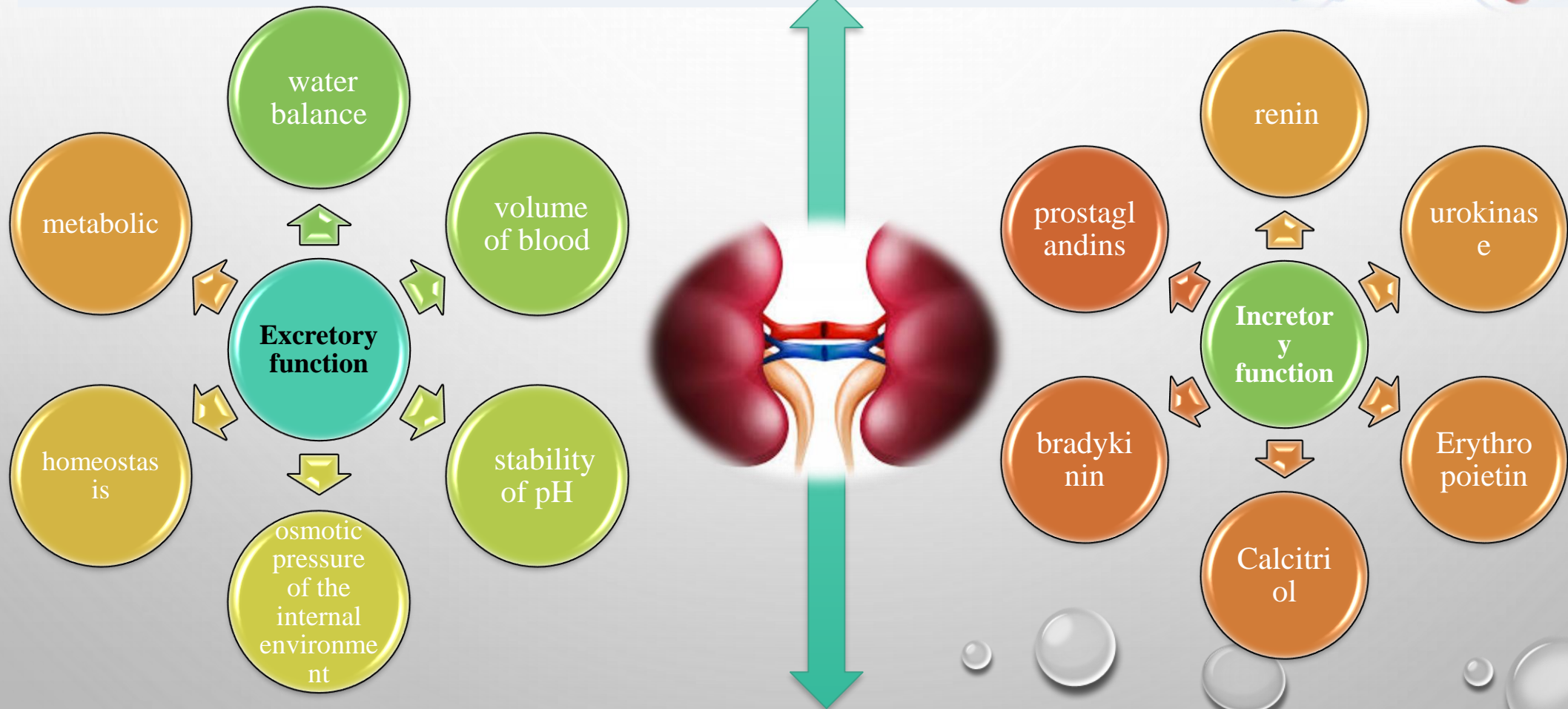
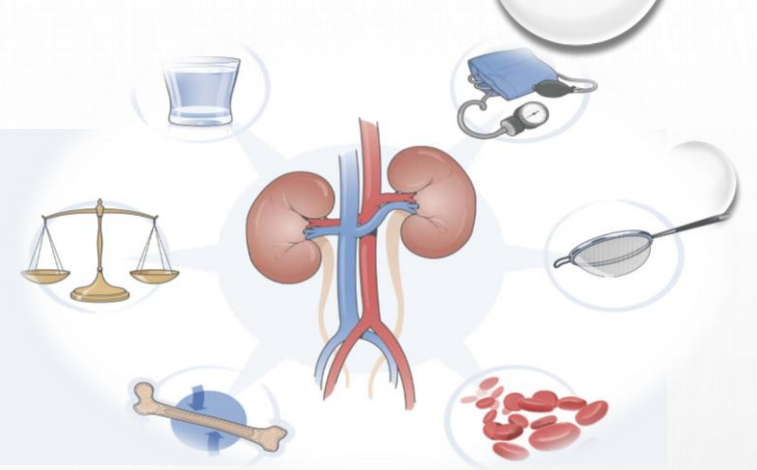


# LABORATORY DIAGNOSIS OF KIDNEY DISEASES



# FUNCTIONS



# LABORATORY DIAGNOSIS

- GENERAL EXAMINATION OF URINE
- URINE CULTURE
- GENERAL AND BIOCHEMICAL EXAMINATION OF BLOOD



# EXAMINATION OF URINE



**Table 5. Urinalysis Results for Case 3**

Component	Result	Reference range
<b>Dipstick urinalysis</b>		
Color	Dark yellow	—
Clarity	Turbid	—
pH	7.0	—
Specific gravity	1.010	—
Glucose	Negative	Negative
Blood	Negative	Negative
Ketones	Negative	Negative
Protein	Negative	Negative
Urobilinogen	Negative	Negative
Bilirubin	Negative	Negative
Leukocyte esterase	Positive	Negative
Nitrite	Positive	Negative
<b>Urine microscopy</b>		
White blood cells	20 per high-power field	0 to 5 per high-power field
Red blood cells	2 per high-power field	0 to 4 per high-power field
Squamous epithelial cells	None	None
Bacteria	Many	—

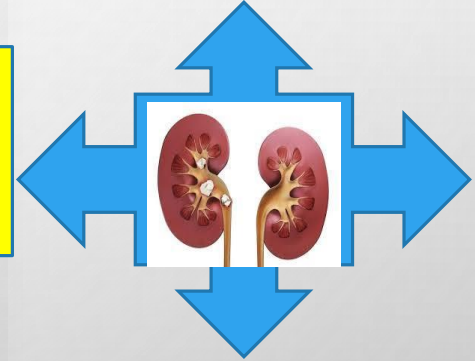


• PHYSICAL EXAMINATION OF URINE

• CHEMICAL EXAMINATION OF URINE

• URINE CULTURE

• EXAMINATION OF URINARY SEDIMENT



# PHYSICAL EXAMINATION OF URINE

- Volume (800-2000 ml)
- Color (yellow)
- Transparency (transparent)
- Odor (faint odor)
- Specific gravity (1008-1025)
- pH (5,0-7,0)



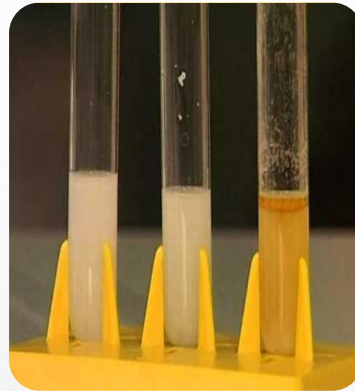
# COLOR OF URINE



"Meat red"



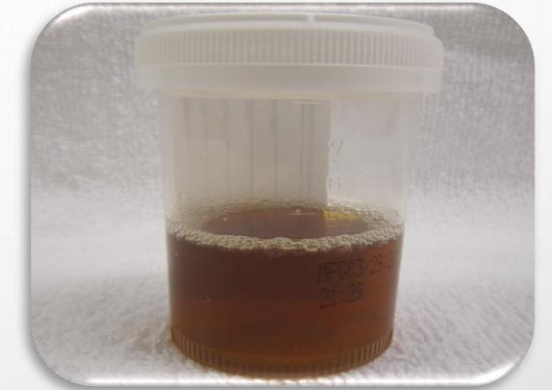
"Beer"  
colored urine



Milky



Black urine



Brown color



Glomerulonephritis



Parenchymatous  
and mechanical  
jaundice



Hiluria occurs when  
there are fat drops or  
lymph in the urine



Alkaptonuria,  
melanosarcoma



Bile pigments

# TRANSPARENCY OF URINE



- ❑ TURBID URINE IS HEATED ( $60^{\circ}\text{C}$ ) – URIC ACID AND URATES
- ❑ ADDITION 10% ACETIC ACID – PHOSPHATE SALTS
- ❑ ADDITION HCL ACID – OXALATE SALTS
- ❑ ADDITION OF ETHER AND ETHANOL – FATS IN THE URINE

# THE ODOR OF URINE

Smell	Cause	Occurrence
<b>Ammonia</b>	bacteria producing urease	Old urine sample Infections of urinary tract Diseases with chronic urine Adenoma of prostate
<b>Acetone (overripe apples)</b>	excretion of acetone in ketoacidosis	Diabetes mellitus Starvation
<b>Maple syrup</b>	branched chain carboxylic oxoacids (especially 2-oxoisocaproic, 2-oxoisovaleric acids)	Leucinosi (maple syrup disease)
<b>Hydrogen sulfide</b>	bacterial decomposition of proteins releases H <sub>2</sub> S from sulfur-containing amino acids	Infections of urinary tract associated with proteinuria Cystinuria, homocystinuria
<b>Mouse</b>	phenylacetate	Phenylketonuria
<b>Fish</b>	tyrosin	Tyrosinemia







# SPECIFIC GRAVITY OF URINE

**HYPERSTHENURIA**

**HIPOSTHENURIA**

**ISOSTHENURIA**

**DECREASED**

Diuretic drugs  
Intake of a large volume of water

Diabetes insipidus  
Unsalty, protein-free diet

**INCREASED**

Diabetes mellitus  
Dehydration

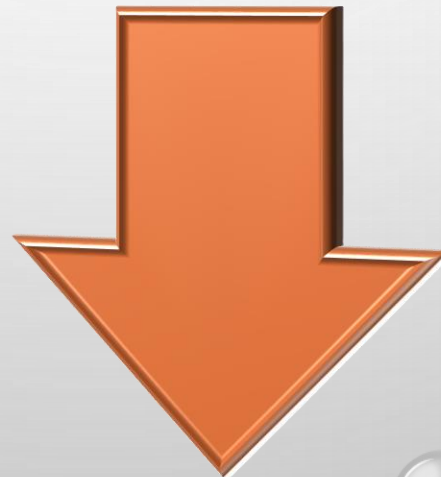
Nephrotic syndrome  
Decrease in water intake

Parkhon's syndrome

# REACTION OF URINE (PH)



Vomiting, metabolic alkalosis, chronic urinary tract infection, cystitis and etc.



Metabolic acidosis, diabetic coma, heart failure, acute kidney failure, acute nephritis, renal tuberculosis and etc.

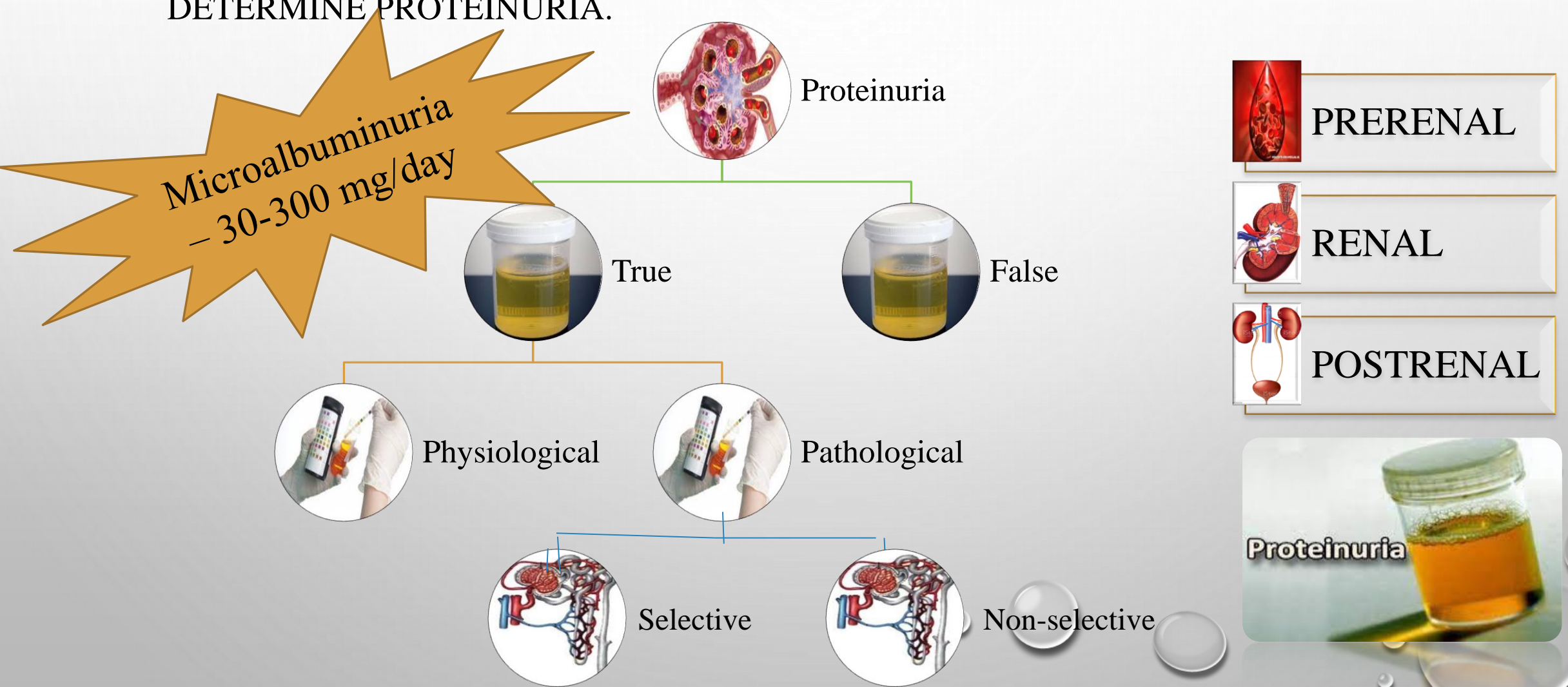
# CHEMICAL EXAMINATION OF URINE

- PROTEIN
- GLUCOZE
- KETONE BODIES
- BLOOD
- BILIRUBIN
- UROBILINOGEN
- etc.



# PROTEIN IN THE URINE

- QUALITATIVE (SULFOSALICYLIC ACID AND NITRIC ACID TEST) AND QUANTITATIVE METHODS BASED ON PROTEIN DENATURATION AND PRECIPITATION ARE USED TO DETERMINE PROTEINURIA.



# GLUCOSE IN URINE

- ❑ THE CONCENTRATION OF GLUCOSE IN BLOOD PLASMA EXCEEDS 10 MMOL/L
- ❑ DEFICIENCY OF ENZYMES (HEXOKINASE AND GLUCOSE-6-PHOSPHATASE)

## PHYSIOLOGICAL

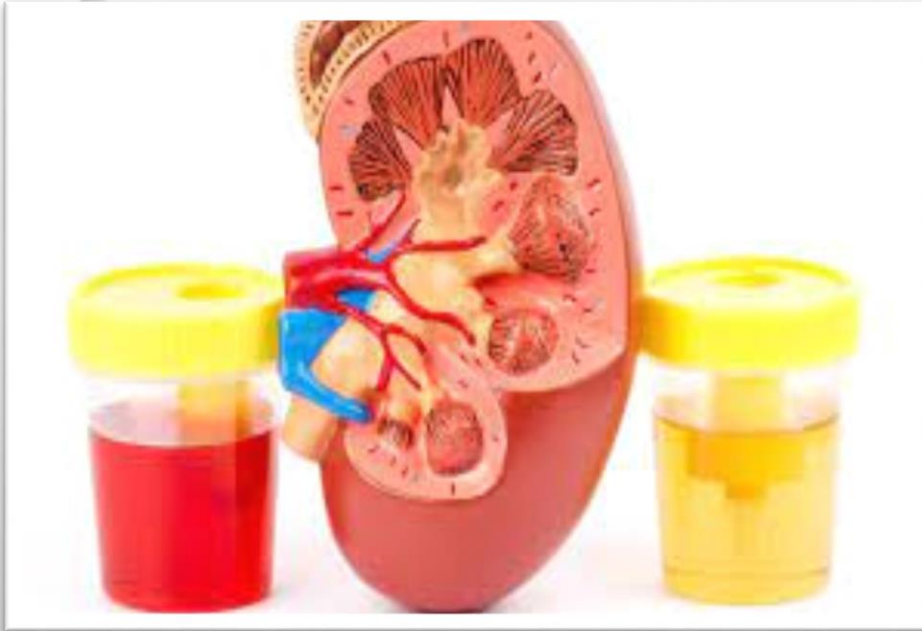
- Excessive intake of carbohydrates
- Emotional overstrain, certain medications

## PATHOLOGICAL

- Diabetes mellitus
- Cushing's syndrome, thyrotoxicosis



# HEMATURIA



## Renal



- glomerulonephritis, kidney tumor, tuberculosis, traumatic injury, hydronephrosis,,
- polycystic kidney disease, urinary tract diseases

## Non-renal



- hemophilia, thrombocytopenia, DIC-syndrome
- treatment with anticoagulants

## Glomerular



- Glomerular hematuria is the result of renal glomerular pathology, it is stable and often accompanied by proteinuria and erythrocyte casts. During the microscopic examination of the sediment, erythrocytes with changed morphology (dysmorphic erythrocytes) are detected.

## Non-glomerular



- The causes of non-glomerular hematuria are renal pelvis stones, bladder and ureter stones, malignant tumors of the bladder and tuberculosis. It is mainly intermittent in nature (periodically increases and decreases).

# THE THREE-CUP TEST

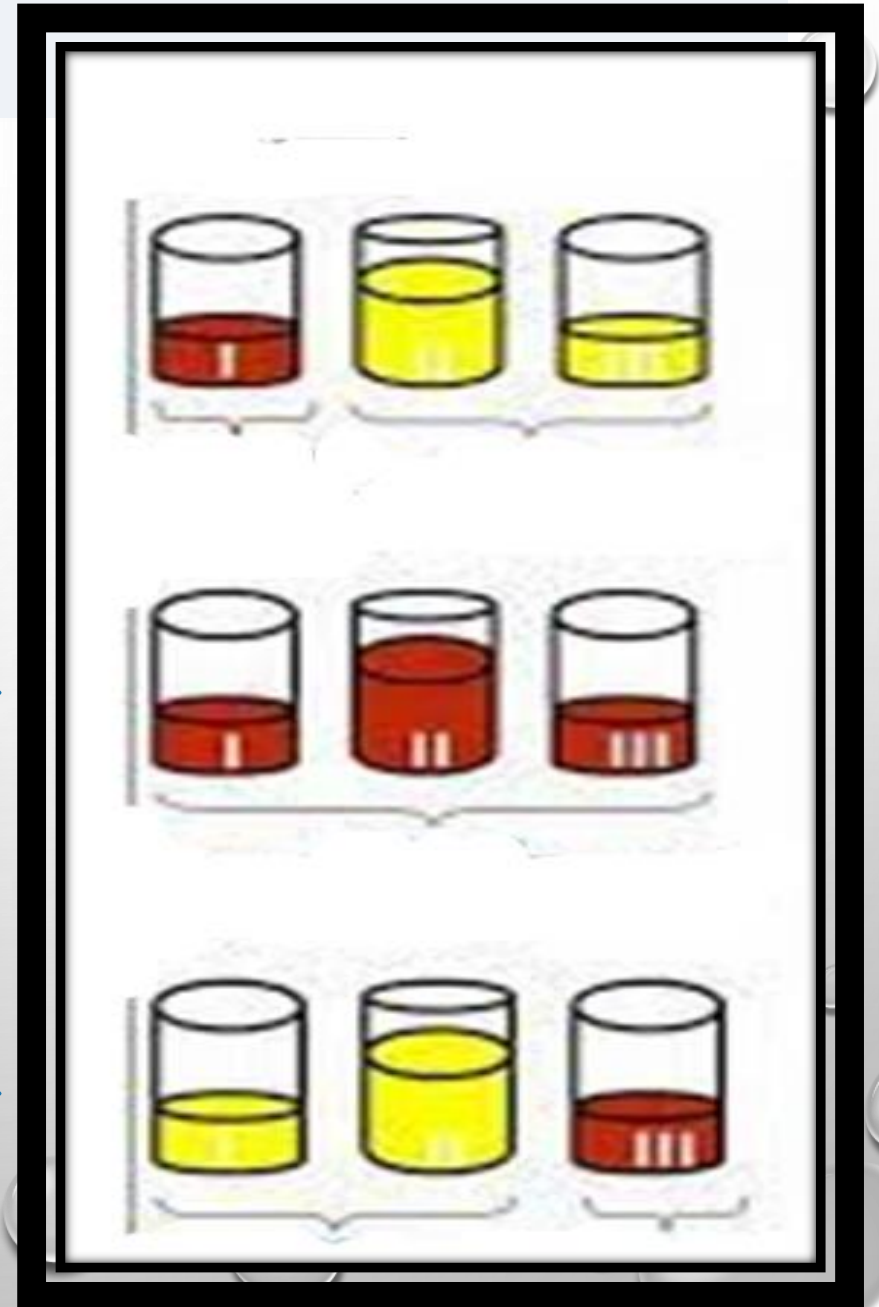
Initial hematuria – the source of hematuria: urethra



Total hematuria – the source of hematuria: bladder, upper urinary tract and kidneys

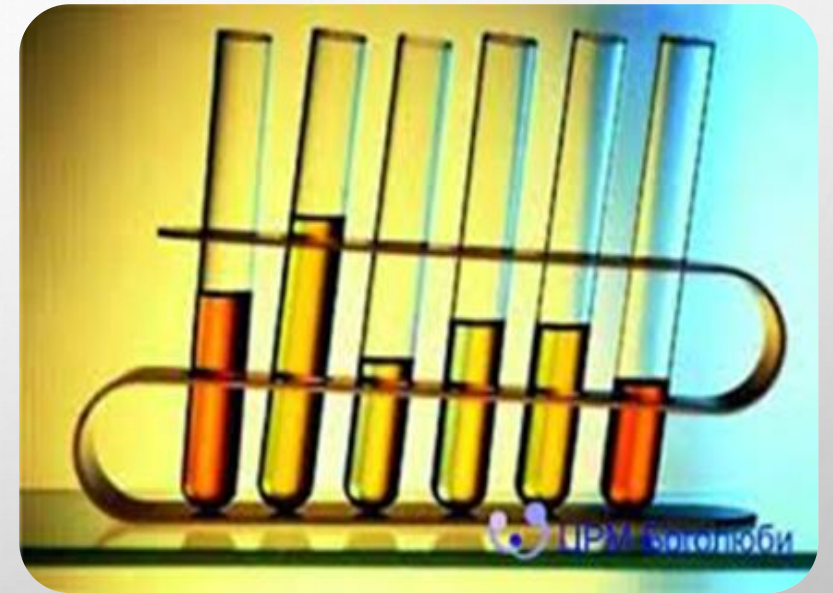


Terminal hematuria – the source of hematuria: uretral bladder neck



# KETONE BODIES, BILIRUBIN AND UROBILINOGEN IN THE URINE

- **KETONURIA** - OBSERVED DURING DIABETES MELLITUS, STARVATION, ALCOHOL INTOXICATION, LONG-TERM FEVER, CARBOHYDRATE-FREE BUT FAT-RICH DIET, HORMONAL DYSFUNCTIONS (THYROTOXICOSIS, CUSHING'S DISEASE, ACROMEGALY AND ETC.), PREGNANCY TOXICOSIS.
- **BILIRUBINURIA** - OBSERVED IN PARENCHYMATOUS AND MECHANICAL JAUNDICE
- **UROBILINOGENURIA** - OBSERVED ON HEMOLYTIC CONDITIONS, DAMAGE TO THE LIVER PARENCHYMA AND INTESTINAL PATHOLOGIES.





# MICROSCOPIC EXAMINATION OF URINE SEDIMENT



## ORGANIC

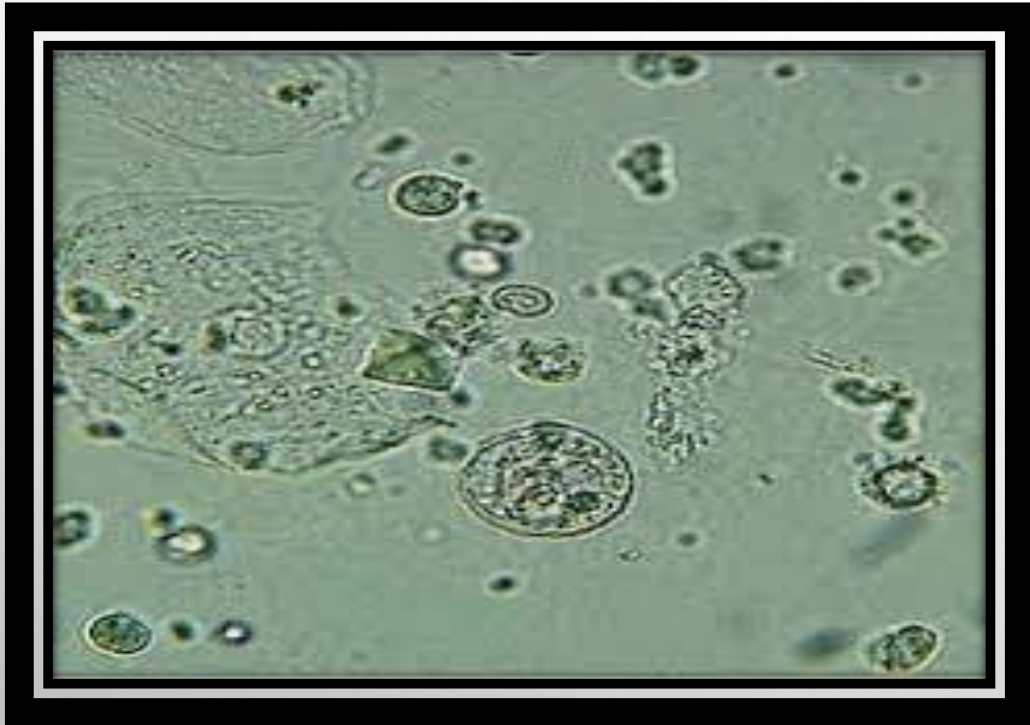
- Epithelial cells
- Leukocytes
- Erythrocytes
- Cylinders
- Mucus and bacteria

## INORGANIC

- Crystals
- Salts



# EPITHELIAL CELLS



*Squamous epithelial cells* are large, wide, round and oval, mononuclear and have small granules in the cytoplasm. It enters the urine through the uterus, external genitalia and urethra.

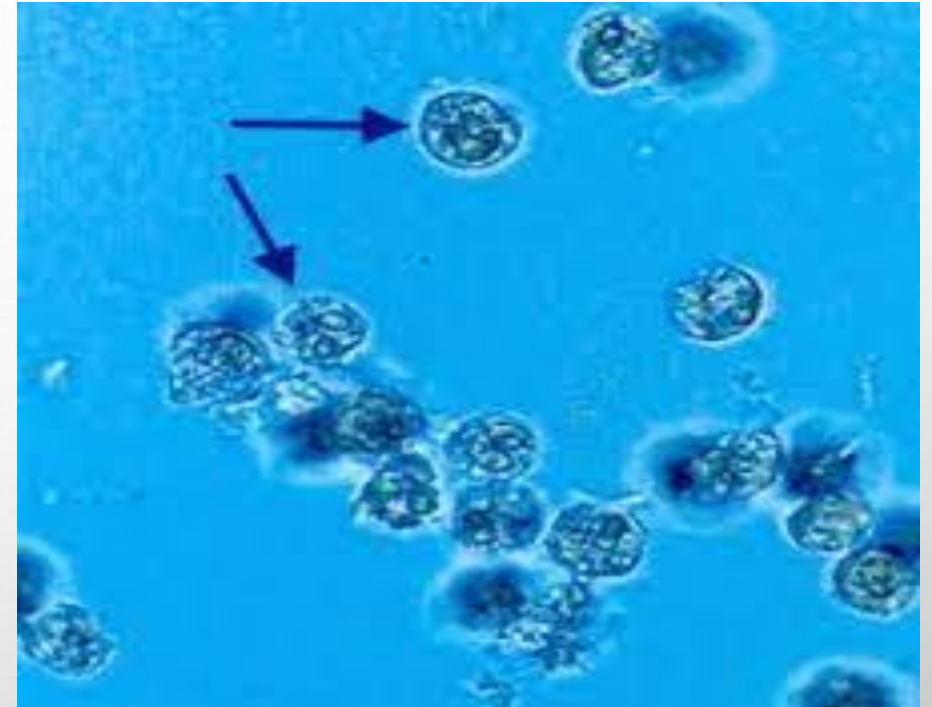
*Transitional epithelial cells* are cells with a yellowish tinged round nucleus. It enters the composition of urine as a result of the rupture of the mucous membrane of the urinary canal, bladder and renal pelvis.

*Kidney epithelial cells* are small, round or cube-shaped cells, the nucleus is large, the cytoplasm is slightly granular and vacuolated. It enters the urine through the epithelium of the urethra.

# LEUKOCYTES

Normally, during the examination of urine sediment, 0-2 leukocytes in men and 4-6 leukocytes in women in the field of vision and mainly neutrophils are detected. In the cytoplasm of leukocytes *in a weak acidic urine*, granularity is clearly noticeable and as a result, the nuclei are difficult to distinguish. In hypotonic *urine with an alkaline reaction* (pH 8.0 - 9.0), leukocytes swell, Brownian movement of neutrophil granules is detected in the cytoplasm. Such leukocytes are called *activated* or *Sternheimer-Malbin cells*. It is detected in inflammatory processes of the kidney and urinary tract. On the contrary, *in urine with an acid reaction*, the size of leukocytes decreases, polymorphic nuclei are clearly visible inside of the cytoplasm, granularity and Brownian movement are lost in the cytoplasm.

In case of leukocyturia, the two-cup test is used to determine the source of inflammation. In the morning, the initial portion of urine is collected in the first cup, and the next portion is collected in the second cup. If leukocytes are found in the first portion of urine, the source of inflammation is considered to be the urethra, if it is found in the second portion, the source of inflammation is considered to be the prostate gland, and if it is found in both portions, the source of inflammation is considered to be the urethra and kidneys.



If the number of leukocytes in the urine sediment exceeds 5-6 in the field of vision, it is called leukocyturia, and if leukocytes cover the entire field of vision, it is called pyuria.

# ERYTHROCYTES

In normal urine sediment, there does not exist erythrocytes or it is found singly. The color and shape of erythrocytes depends on the pH and relative density of urine. In the urine sediment, erythrocytes are yellowish-green or reddish color, appear as a small circle cells, are in the form of a two-contour ring, and are not granular. In urine with weak acid reaction and normal relative density, erythrocytes appear light yellow, retain their shape and pigment for a long time, and do not change. Such erythrocytes are called "*fresh*" or *unchanged erythrocytes*.

Erythrocytes can take the form of stars in solid urine with an acid reaction. The sizes of erythrocytes found in urine with a weak alkaline reaction are larger than those of normal erythrocytes. In urine with an alkaline reaction, they break down quickly, and if it remains in urine with a low relative density for a long time, erythrocytes lose their pigment and as a result, it turns into colorless, different-sized, sometimes jagged contoured and thin-membrane cells. Such erythrocytes are called "*alkalized*" or *altered erythrocytes*.

Usually, in the case of hematuria caused by the damage of the kidney glomeruli, changed erythrocytes are detected in the urine, and in the case of hematuria caused by the damage of the ureters, unchanged erythrocytes are detected.



# CASTS

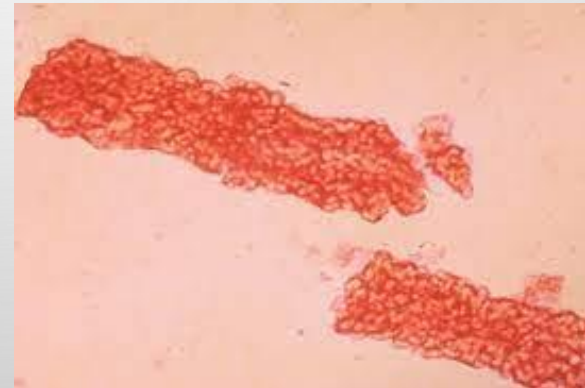
- *Hyaline casts* – observed in kidney diseases accompanied by proteinuria (eg, nephrotic syndrome). They are pale colored transparent particles formed from coagulated proteins. The finding of hyaline cylinders in the urine sediment indicates the increase of glomerular capillary permeability.
- *Granular casts* – consist of altered (destroyed and fragmented) renal epithelial cells. A characteristic feature is that their surface has a granular appearance. If there are blood pigments in the urine, it will be red-brown, and if there are bile pigments, it will be yellow. It is found in glomerulonephritis, pyelonephritis, renal amyloidosis, diabetic glomerulosclerosis and other pathologies.
- *Erythrocyte cylinders* – consist of protein derivatives covered by modified erythrocytes. It is formed during hematuria of renal origin, it indicates the damage of renal glomeruli. They are found in glomerulonephritis, kidney tumor, heart attack, thrombosis of renal veins and etc. The blood that coagulates in the renal tubules and takes a cylindrical shape is also referred to erythrocyte cylinders.
- *Leukocyte cylinders* - covered with leukocytes, formed of coagulated protein, having a cylindrical shape. It is mainly characteristic for purulent-inflammatory processes of the kidneys accompanied by leukocyturia and pyuria.



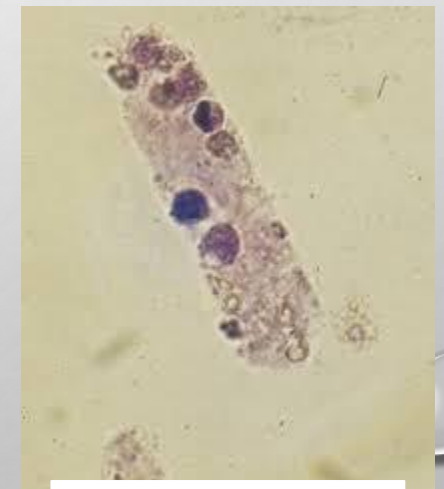
Hyaline



Granular



Erythrocyte



Leukocyte

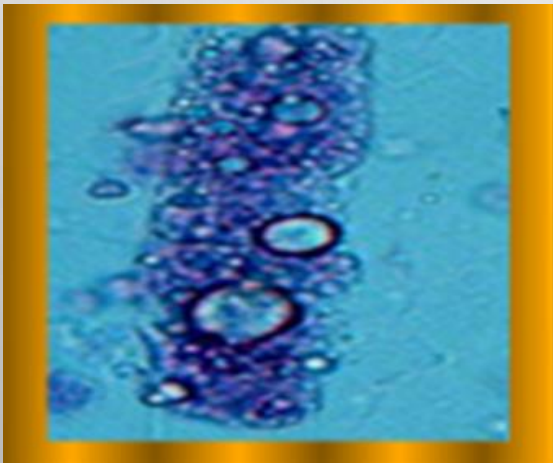
# CASTS



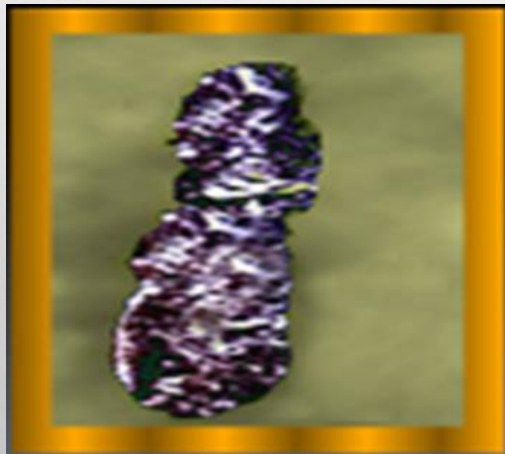
Wax-like



Epithelial



Fatty



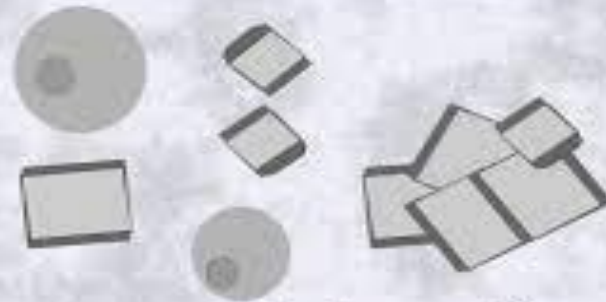
Pigment cylinders

- *Wax-like cylinders* – light yellow particles with rough contours. They are larger than hyaline cylinders. It is detected during severe acute and chronic damage of kidneys.
- *Epithelial cylinders* – are epithelial cells of kidney tubules and cylinders composed of protein. Finding them in the urine sediment indicates damage of the renal tubule. It is observed in tubular necrosis, poisoning with heavy metal salts and salicylates, nephrotic syndrome.
- *Fatty cylinders* – formed from epithelial cells that have undergone fat degeneration.
- *pigment cylinders* – are the accumulation of hemoglobin
- *false cylinders* - they look like a cylinder, but they have longitudinal stripes. False cylinders include bacteria, myoglobin and uric acid salts.

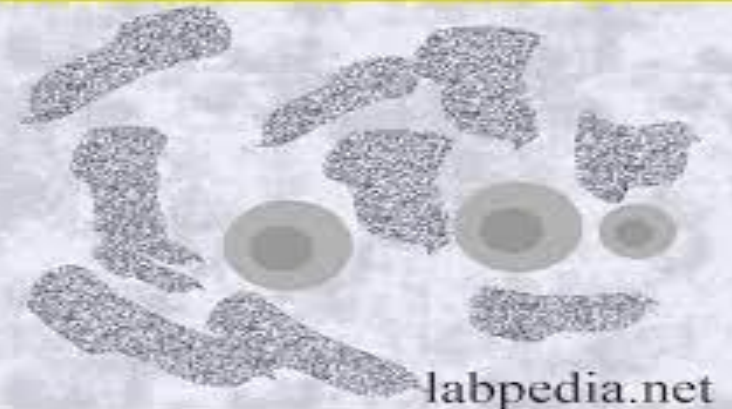
# INORGANIC URINE DEPOSITS

Inorganic elements of urine sediment include crystals and salts. Their characters depend on the colloidal state of urine, pH and other characteristics. Uric acid crystals and urates are observed in the contain of acidic urine. Crystals of oxalic acid, calcium and magnesium salts of phosphoric acid, and ammonium salts of uric acid are found in urine sediment with an alkaline reaction.

Uric acid crystals



Amorphous phosphate crystals



# URINE CULTURE



Urine culture is determined in the following cases:

- ▼ Finding the cause of urinary tract infection
- ▼ prescribing the correct treatment based on the sensitivity of microorganisms to antibiotics;
- ▼ to determine the treatment effect in urinary tract infection.



# LABORATORY DIAGNOSTICS OF THE EXCRETORY FUNCTION OF THE KIDNEYS

## Glomerular filtration

- GFR

Creatinine clearance: 80-120 ml/min in men;  
70-110 ml / min in women

## Tubular reabsorption

- TR

Tubular reabsorption is normally 96-99%

## Secretion in tubules

- ST

Based on the determination of phenolroth clearance.  
94% of phenolroth is removed from the body through secretion

# GENERAL AND BIOCHEMICAL EXAMINATION OF BLOOD IN UROLOGY DISEASES

CREATININE - 80-115  $\mu\text{mol/l}$  in men, 53-97  $\mu\text{mol/l}$  in women, 18-35  $\mu\text{mol/l}$  in newborns, 35-110  $\mu\text{mol/l}$  in children up to 14 years old.

UREA - 4,2-8,3  $\text{mmol/l}$  in newborns, 1,4-4,3  $\text{mmol/l}$ , 1,8-6,4  $\text{mmol/l}$  in children up to 14 years old.

RESIDUAL NITROGEN - 14,3-28,6  $\text{mmol/l}$ .



# ACUTE GLOMERULONEPHRITIS

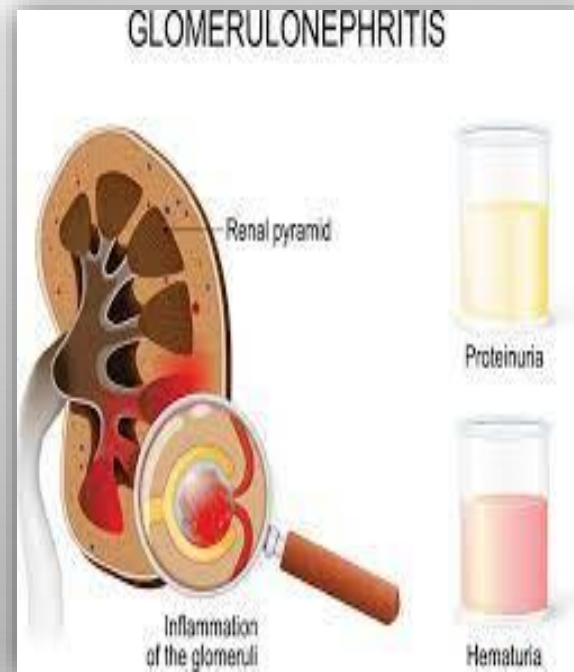
## ETIOLOGY

- $\beta$ -hemolytic streptococci
- rarely pneumococci, staphylococci
- 

## PATHOGENESIS

- Type II allergic reactions
- Type III allergic reactions

**Clinic.** Patients complain of edema on the face, under the eyes, then on the trunk and limbs, decreased urination, impaired vision, pain in the lower back.



## LABORATORY DIAGNOSTICS



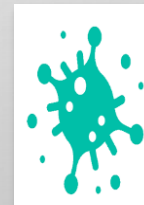
**Urine examination:** the presence of urine in the color of "meat juice", the specific gravity of urine does not change, but it may increase during the period of increased edema, proteinuria, hematuria



**Urine sediment:** erythrocyturia, cylinduria, mainly hyaline cylinders, erythrocyte cylinders, renal epithelial cells, rarely leukocyturia.



**Blood examination:** the amount of C-reactive protein, fibrinogen,  $\alpha$ 2-globulin increase, ESR increases.



**Immunological examination:** decrease in the amount of anti-O-streptolysin, streptococcal antigen, decrease in the amount of complement

# ACUTE RENAL FAILURE

## Etiology

- *Prerenal factors* include shocks, sedation, dehydration, hemolysis, etc., *renal causes* include acute necrosis of tubules, acute glomerulonephritis, acute pyelonephritis, etc., and *postrenal factors* include urinary tract pathologies, prostate gland diseases.

## Pathogenesis

- The basis of the disease is the disruption of kidney blood circulation, filtration in the glomeruli and reabsorption in the tubules due to the effect of etiological factors.

**Clinic.** Symptoms are mainly observed in the oligoanuria stage of the disease: decreased diuresis, nausea, vomiting, adynamia, dimming of consciousness, lowering of AP, muscle spasms, drowsiness, edema, respiratory and heart failure, etc.

## LABORATORY DIAGNOSTICS

*Urine examination:*  
proteinuria, cylindruria,  
erythrocyturia,  
leukocyturia (if it  
develops against the  
background of an  
inflammatory process)

*Blood examination:*  
leukocytosis, increased  
creatinine,  
hyperkalemia,  
hyponatremia,  
hyperphosphatemia,  
hypercalcemia,  
metabolic acidosis.