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**TUBERCULOSIS OF THE URINARY TRACT AND  
ORGANS MALE GENITAL SYSTEM.  
ACUTE AND CHRONIC RENAL FAILURE**

EDUCATIONAL MANUAL

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The educational manual for independent work of students IV courses of the II international faculty, majoring in "Medicine", presents theoretical material by topic, practical tasks and test control of knowledge.

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## CONVENTIONAL ABBREVIATIONS

|              |                              |
|--------------|------------------------------|
| ANN -        | acute renal failure          |
| EU -         | excretory urography          |
| CT -         | computer tomography          |
| MRI -        | magnetic resonance imaging   |
| PET -        | positron emission tomography |
| RRG -        | radiorenography              |
| UBM -        | ureteric-bladder mouth       |
| Ultrasound - | ultrasound examination       |
| CRF -        | chronic renal failure        |
| CBS -        | cup-bowl system              |

## GLOSSARY

*Angiography* is the study of blood vessels, parenchyma and cavity system of kidneys with the help of a radiopaque substance. Retrograde tranfemoral aortography according to Selzinger is used.

*Antegrade urethrocytography* - a radiopaque image of the bladder and urethra obtained by the method of excretory urography.

*The primary affect* is a limited inflammatory process that occurs in the tissues at the site of the initial impact of pathogenic microorganisms.

*Hypernephroma* is a malignant kidney tumor.

*Densitometry* - determination of tissue density using CT.

*Excretory urography* is a method of contrast X-ray diagnostics that allows obtaining data on kidney function.

*Infusion urography* is a modification of excretory urography, which is especially desirable when kidney function is reduced.

*CT-enhancement* - conducting tomography after IV administration of a contrast agent to the patient.

*Computed tomography (CT)* is a layer-by-layer x-ray examination based on computer reconstruction of the image obtained when scanning the object with a narrow beam. X-ray irradiation.

*Magnetic resonance imaging (MRI)* is a modern method of radiation diagnostics based on the phenomenon of magnetic nuclear resonance on the ability of the nuclei of some atoms (H) to behave as magnetic dipoles.

*Multidrug-resistant tuberculosis (MR TB)* is a form of tuberculosis when the patient secretes MBT resistant at least to isoniazid and rifampicin, and often to a larger number of first- and second-line antituberculosis drugs, which was confirmed by a laboratory method in the TMCH.

*Survey radiography of the kidneys* is a method of non-contrast X-ray diagnostics, which allows you to detect morphological changes in the kidneys and calcareous formations in the abdominal cavity, spine, large lumbar muscle.

*Radiography* is a radionuclide method of diagnosing kidney function disorders.

*Renovasography* is a method of radiographic examination of phases of renal blood circulation.

*Retrograde (ascending) pyelography* is a method of studying the morphology of renal calyces, bowls and ureter after introducing 8-10 ml of 20-30% contrast material solution into them through a urinary catheter.

*Tuberculosis relapse (TB)* is a confirmed case of tuberculosis in a patient who previously successfully completed a full course of antimycobacterial therapy and was considered cured, or who completed the main course of treatment with the result of "treatment completed" and has a recurrence of bacteremia.

*Scan* - a quick overview.

*Sonography* - two-dimensional US - image.

*Tubercles* (lat., gulka) are nodules in the tissues of the body, caused by the tubercle bacillus.

*Tuberculin* is a specially processed extract from the culture of the causative agent of tuberculosis (Koch's bacillus). It is used to diagnose tuberculosis.

Tuberculosis (Lat., gulka) is a common infectious disease of humans and some animals that mainly affects the lungs, bones, skin, joints, and intestines. The causative agent is a special microbacterium, i.e. Koch rod. Another name is dryness.

*Tuberculosis of the kidneys* is a specific inflammation of the disease of a secondary nature due to the introduction of mycobacteria by the hematogenous route.

*MDR-TB (DR-TB)* is a form of tuberculosis in which the patient excretes MDR resistant to at least isoniazid and rifampicin, any fluoroquinolone, and at least one of the three drugs amikacin, kanamycin, and capreomycin.

*Ultrasound examination (USE)* is a modern method of examining the kidneys and bladder.

*Ascending urethrography* is a technique of radiography of the urethra after retrograde filling of it with a 60-70% urographin solution using a Jeanette syringe.

is a form of tuberculosis in which the patient secretes MBTs that are resistant to one or more anti-tuberculosis drugs, which was confirmed by a laboratory method in TMCH.

*Cystography* is an X-ray examination of the bladder filled with a radiopaque substance. It is retrograde and descending.

## PREFACE

The scientific and technical progress of the 21st century requires specialists of various specialties to take a new approach to mastering the achievements that mankind has made. Today, the employees of III and IV level higher institutions face the task of training specialists of the future.

Educational and methodological manual "Tuberculosis of the urinary tract and organs of the male reproductive system. Acute and chronic renal failure" is intended for students of IV courses of the II international faculty, majoring in "Medicine". The purpose of the manual is for students to master the principles and methods of diagnosing patients, the specifics of treatment, dispensation of patients and examination of working capacity, as well as to study the basic concepts and classifications, as well as for students to acquire knowledge on the clinical interpretation of the obtained data.

The guide provides help to students to correctly collect anamnesis, to have an idea of the pathological anatomy of the process, to carry out classification, diagnosis and treatment, as well as to reveal solutions to problems associated with acute and chronic renal failure and to prove to students the causes of the occurrence, course and prevention of this disease.

In the manual, most of the illustrative material is created by the authors, otherwise - a link to the source is added. Tasks for test control can be used during extracurricular and classroom training.

Educational manual "Tuberculosis of the urinary tract and organs of the male reproductive system. Acute and chronic renal failure" for students of IV courses of the II international faculty, majoring in "Medicine", fully meets the requirements of the work program in the discipline "Urology".



## SECTION 1

### TUBERCULOSIS OF THE URINARY SYSTEM

**The purpose of training:** to teach students to properly examine patients with urogenital tuberculosis, to pay special attention to the early manifestations of the disease, to methods of early diagnosis, features of treatment, examination of patients and examination of work capacity, as well as the study of basic concepts and classifications.

**Didactic purpose:**

1. Consider the etiopathogenesis of urogenital tuberculosis.
2. Students should know the early symptoms of kidney and ureter tuberculosis.
3. To study the methods of diagnosing tuberculosis of the kidneys and ureter.
4. 3 to master the schemes of conservative treatment of tuberculosis of the kidneys and ureter.
5. Determine indications for surgical treatment.
6. To study the symptomatology, methods of diagnosis and treatment of tuberculosis of the genital system in men.
7. To study the peculiarities of dispensation of patients with urogenital tuberculosis and examination of working capacity.

**Glossary of terms:**

*Tubercles (lat., gulka)* are nodules in the tissues of the body, caused by the tubercle bacillus.

*Tuberculin* is a specially processed extract from the culture of the causative agent of tuberculosis (Koch's bacillus). It is used to diagnose tuberculosis.

*Tuberculosis* (Lat., gulka) is a common infectious disease of humans and some animals, which mainly affects the lungs, bones, skin, joints, and intestines. The causative agent is a special microbacterium, i.e. Koch rod. Another name is dryness.

## **UROGENITAL TUBERCULOSIS**

Urogenital tuberculosis can rightfully be classified as the most common pathology of the genitourinary system. This is evidenced by statistical data of both domestic and foreign authors. Tuberculosis of the kidneys makes up 13-15 percent of the total number of all surgical diseases of the upper urinary tract, second in frequency only to kidney stone disease, and tuberculosis of the external genitalia in men - 20-25 percent of diseases of the scrotum.

Modern methods of X-ray examination make it possible to recognize tuberculosis of the genital organs in the early stages of its development, and thus to start rational treatment in a timely manner.

**Gender, age, side of lesion.** Tuberculosis of the kidney occurs with the same frequency in persons of both sexes. It usually affects people of younger and blooming age. The largest number of patients occurs between 20 and 40 years of age. It is generally accepted that it occurs less frequently in children under 10 years of age and in the elderly. According to the literature, it is mostly impossible to establish kidney damage on one or the other side. Bilateral kidney damage was noted by a number of authors in 33-35 percent of cases.

**Pathogenesis.** According to many authors, kidney tuberculosis is a secondary process in the body and it arises as a result of hematogenous metastasis from the existing primary complex in the lungs, intestines or lymph nodes. Thus, kidney

tuberculosis is considered as a local manifestation of a general tuberculosis infection in the body.

Tuberculosis mycobacteria, as a rule, penetrate into the kidney by a hematogenous route, without causing a liquid lymphogenic route from adjacent organs affected by tuberculosis (intestine, mesenteric lymph nodes, spine).

However, in many patients with kidney tuberculosis, during a difficult clinical examination, it is not possible to establish a specific process in other organs from which hematogenous transmission of the infection was possible.

BY. Lebedeva (1952), A.I. Myants (1954), A.N. Chystovich (1960) recognize that tuberculosis mycobacteria can enter the kidney simultaneously with lung damage or from the lymph nodes during primary infection of the body. However, while tuberculosis mycobacteria find favorable conditions for their development in the lungs or intestine, these conditions are less favorable in the kidney parenchyma, where special sensitization of the kidney tissue, its susceptibility, is necessary for their vital activity.

According to A.I. Mayantsa, this sensitization sometimes occurs 3-10 years after the initial clinical manifestation of tuberculosis in the body. As soon as the infectiousness of tuberculosis was proven, features were found that distinguish it from other infectious diseases.

It was established that the nature of the course and morphological manifestations of TB in many cases depend on how the tubercle bacillus first penetrated the body, or the process takes place in an organism that has already been exposed to tuberculosis infection. Depending on this, 2 periods were distinguished in the development of TB: 1) the primary complex 2) the result of reinfection.

In 90-95 percent, the primary infection is through the respiratory tract.

The main feature of primary tuberculosis is its development against the background of active foci of primary infection - a constant source of sensitization of the body.

The primary focus is the source of hematogenous tuberculosis. It is characterized by the presence of foci in various organs and is characterized by increased activity of the body. At the same time, the occurrence of a focus - metastasis in the kidney and the development of a progressive process in them do not coincide in time: metastasis occurs in childhood, and the development of tuberculosis in the kidney begins after 10-15-20 years, when tuberculosis is no longer present in other organs.

Hematogenous involvement in the kidney usually occurs during bacillemia. Sometimes this happens when a tuberculosis focus is aggravated in any location, in other cases new tuberculosis foci appear. The body as a whole and its individual organs are sensitized to tuberculosis infection. Clinical studies have established that hematogenous transfer of tuberculosis infection to the kidney occurs in 5-14 percent of pulmonary tuberculosis, and in 2-12.7 percent of bone tuberculosis. These data should be taken into account during preventive examinations of the population.

More recently, some studies have recognized the possibility of the infection entering the kidney by an ascending route (urinogenically) from the bladder affected by tuberculosis. Today, this way of tuberculosis infection entering the kidney is considered a rare phenomenon. It is possible only in some cases as a result of backflow of urine infected with mycobacterium tuberculosis and bladder into the ureter with spasmodic contractions of the bladder and insufficiency of the vesical mouths of the ureters due to their specific lesions. This phenomenon was called vesicoureteral reflux. The upward path of spread of tuberculous infection from the affected bladder to the kidney is possible, both with a banal infection through the

opening of the ureter, and through the lymphatic pathways located in the tissue surrounding it.

The penetration of mycobacterium tuberculosis into the parenchyma of the kidney does not always cause the development of a specific process in it. The nature of the changes that occur depends on the degree of natural or acquired immunity from these conditions, the following options for the development of the pathological process in the kidney are possible: a) tuberculosis mycobacteria that have penetrated the kidney are reduced by the body's protective forces and die; b) tuberculosis mycobacteria in the kidney in a latent state, without causing any deviations from the parenchyma; c) tuberculosis mycobacteria find favorable conditions for their life activity, as a result of which a specific tubercular process develops in the kidney.

Until recently, there was an opinion of the authors that in most patients, the tuberculous process first affects one kidney and that the simultaneous impression of both kidneys is noted only in 15-20 percent (R.M. Fronshtein, I.M. Epshtein, etc.). However, today such a point of view is denied by many authors. So A.I. Mayants and A.N. Chistovych and others. it is believed that in most patients with tuberculosis, two kidneys are affected at the same time, but the degree of damage in each of them is not equally intense. In one of the kidneys, the tuberculous process is in a latent state and is not clinically noted, and in the future it is even eliminated, while in the other kidney, a blooming focus develops with the progression of the process. The validity of these statements is confirmed in practice - in the rapid outbreak of a tubercular process in a number of patients in the other kidney after nephrectomy, although the previous clinical examination of this kidney before the operation did not establish a tubercular process in it.

Veselovskyi (1955) believes that all modern diagnostic methods are not sufficient to assert that a kidney considered healthy is not affected by tuberculosis and that renal tuberculosis in its initial form is always bilateral.

For a long time, there was an almost unanimous opinion, however, that the hematogenous deposition of a specific embolus carried into the kidney occurs in the medullary layer at the top of the papillae, since the most pronounced specific changes in the parenchyma of the removed kidney are found in this layer. However, at the autopsy of the corpses of patients who died from pulmonary tuberculosis, the main mass of tubercles in the cortical layer of the kidneys and, to a lesser extent, in the medullary layer, were found in the early stages of the development of a specific process in the kidneys. In later periods, specific changes prevail in the brain layer.

The tuberculous process developing in the parenchyma of the kidney, during its spread, reaches the tops of the cups, and, undergoing disintegration, breaks into the lumen of the urinary tract. There is a permanent connection of the focus with the cups, bowl and bladder.

Tuberculosis infection of bowls and cups, according to rich authors, takes place through contact. A.I. Mayants does not rule out the spread of tubercle bacilli through lymphatic vessels located in the submucosal layer of the pelvis or ureter.

With further progression, the tuberculous process spreads to the urinary bladder, which is affected by tuberculosis only with a long process in the kidneys, from where it spreads downward.

In the presence of a tuberculous process in the urinary bladder, significantly pronounced destructive changes are usually noted in the parenchyma of the kidneys. However, in some cases, when a widespread ulcerative process of the mucous membrane of the urinary bladder is detected, only a specific infiltrate without signs of decay is detected in the affected kidney.

For a long time, the bladder remains intact until tuberculosis infection. According to V.D. Ground, such a condition is explained by the fatigue of the nerve trunks passing through its wall.

**Pathological anatomy.** Touching on the acute miliary form of renal tuberculosis, it should be said that it is clinically unrecognizable and can be detected only on section. Therefore, chronic tuberculosis of the kidney should be discussed in more detail.

When mycobacterium tuberculosis settles in the parenchyma of the kidney, the presence of the necessary allergic state of the body and, in particular, sensitization of the renal parenchyma, the tuberculous process (tuberculosis nodule) begins to develop.

Further, diverse dynamics of the tuberculosis process are possible.

In some cases, tubercles disintegrate, and disintegration cavities (caverns) are formed from the fusion of adjacent disintegrated tubercles in the kidney parenchyma, sometimes these caverns are single, in other cases there are many of them, occupying large areas of the renal parenchyma (polycavernous process) if the tubercles disintegrate occurs on the mucous membrane of the pelvis or ureter, then specific tuberculous ulcers are formed here. The wall of the cavern is covered with necrotic tissue, and its cavity is filled with caseous contents or thick pus. In the center of specific foci, nonspecific inflammatory changes develop. Zones of perifocal inflammation are sometimes closed over a long period of time.

Under favorable protective conditions in the body, the reverse development of the formed tubercles, their resorption, scarring, and petrification is possible. The dynamics of these changes are especially pronounced in relation to the kidney, where, along with the costal tubercles, they can be found in the medulla. Petrification of tuberculous changes is possible not only in nodules, but also in separate caverns and even in massive kidney lesions - a frozen kidney.

As a result of scarring and specific ulcerative changes from antibacterial treatment or under the influence of the body's protective reactions, a narrowing of the

lumen (passage) of the ureter often develops, with consequent impaired motility of the pelvis. As a result of the expansion of the bowl and calyces and stagnation of what can be contained in the caverns, a specific tuberculous pyonephrosis develops in a number of patients: the parenchyma of the kidney thins, and the organ itself turns into a purulent bag. In some cases, with complete obliteration of the lumen of the ureter, exclusion of the entire kidney-autonephrectomy may occur. False healing occurs: urine becomes transparent, dysuric phenomena disappear.

Sometimes, instead of pyonephrosis, atrophy of the kidney develops, in which only a lump of sclerosing tissue is determined at the level of its location, in the center of which there are remnants of the renal parenchyma with the phenomena of a specific tubercular process. In some cases, the atrophied parenchyma of the kidney is replaced by hyperplastic, growing adipose tissue and turns into a lump of fat with scant remnants of renal tissue. These changes were called fatty replacement of the kidney. Extremely rarely, in the presence of pyuria and mycobacterium tuberculosis, the urogenital examination of the removed kidney does not establish specific changes characteristic of tuberculosis. The histological picture of the kidney in these patients corresponds to a chronic inflammatory process such as nephrosclerosis. This form of kidney tuberculosis S.P. Fedorov called it Koch's nephrocirrhosis. The lesion of paranephritis manifests itself in two variants. In some cases, it is accompanied by purulent melting of adipose tissue, in others, which occurs much more often, sclerosing paranephritis develops, as a result of which the kidney is surrounded by a scar shell. Such changes in the perirenal tissue force the surgeon to use the subcapsular method when removing the kidney.

When the process spreads to the urinary tract, the lacrimal bowl, ureter, and bladder develop specific tubercular changes - tubercles, and as a result of their disintegration, one or another severity of ulcerative changes develops. Simultaneously



with these changes in the mucous membranes, a sclerosing process develops in the adipose tissue surrounding the pelvis and urethra.

Scarring of specific ulcers on the mucous membrane of the ureter, as well as the periurethral tissue, contributes to the formation of narrowing of the lumen (passage) of the ureter, and on this basis, a motility disorder of the pelvis and ureter occurs. Changes occurring in a tuberculous lesion of the urinary bladder are identical to those described in the pelvic lesion, tubercles, specific ulcers, sclerosing process in the peribladder tissue.

As a result of scarring of ulcerative changes and infiltration of peribladder tissue, the development of a shrunken so-called "small bladder" is possible, which is usually accompanied by insufficiency of the bladder mouths of the ureters. The last circumstance can cause a very serious complication - ureteral reflux.

**Classification.** To date, there is no proven and generally recognized classification of kidney tuberculosis. Most of the proposed classifications of tuberculosis of the kidneys and urinary tract are based on the principle of staging. They are based on the pathomorphological course of the tubercular process: from the initial infiltrative changes in the depth of the renal parenchyma to the development of polycavernous tuberculosis of the kidney or tuberculous pyonephrosis.

Before the antibacterial era, the classification of renal tuberculosis was mainly clinical. For example, S.P. Fedorov identified two forms of kidney tuberculosis: 1 – acute or subacute, miliary form, 2 – chronic kidney tuberculosis. He, in turn, divided chronic kidney tuberculosis into two subgroups: a) with specific tubercular changes, b) without specific changes - according to the type of chronic nephritis.

In the past, when the main method of treatment was early nephrectomy, any classification was considered to solve the main diagnostic task - recognition of

diseases at an early stage. At present, the following classification is the most acceptable and sufficiently appropriate for practical purposes:

I. Clinical and radiological forms of renal tuberculosis:

- 1) tuberculosis of the renal pelvis (without existing X-ray changes)
- 2) tuberculosis of the renal papilla (papellitis)
- 3) cavernous tuberculosis of the kidney
- 4) tuberculous pyonephrosis

II. Phase of the tuberculosis process:

- 1) Open tubercular process
- 2) Exclusion
- 3) Total segmental scarring
- 4) Disclosure of one cup

III. Bacillary: BK + BK-2

IV. Functional state of the kidney:

- 1) The function is broken
- 2) The function is reduced
- 3) The function is missing

V. Complications:

Pyelonephritis, nephrolithiasis, hypertension, amyloidosis, etc.

Each of these forms is easily determined by classical methods, has its own clinical and radiological characteristics, its prognosis and methods of treatment.

**Semiology.** In the clinic of tuberculosis of the kidneys and urinary tract, unfortunately, there are not enough specific pathognomonic symptoms, quite often, tuberculosis of the kidneys can appear under the guise of a completely different disease.

Brief clinical assessment of individual symptoms.

a) Bacillus

This term should be understood as the excretion of tuberculosis mycobacteriuria in the urine in the absence of pyuria. Bacilluria is a symptom of great practical importance, as it can be the first manifestation of latent, flowing tuberculosis of the kidneys. Occurring without any clinical manifestations, bacilluria appears suddenly and indicates that there is damage to the parenchyma of the kidneys, mycobacterium tuberculosis (Kilpoitner) does not pass through. However, he established that mycobacterium tuberculosis under certain conditions can pass through the renal filter without causing changes in the kidney. He claims that bacilluria is a temporary phenomenon and occurs periodically. His opinion is that bacilluria should be recognized only when it manifests itself due to some complications or aggravation of the tubercular process in the lungs (hemoptysis, pyothorax, accompanied by a large influx of tuberculosis mycobacteria into the blood). Emphasizes that the usual research methods are not enough for this - it is necessary to examine the urine daily for several weeks. These data were confirmed by M.M. Chaussovsky

B) Pyuria.

Manifestation of pyuria - proved the connection of the tuberculosis center with the excretory urinary tract. At the first stage of the symptom, due to the small lumen of the course, due to the connection, the intensity of pyuria is quite insignificant. It can occur periodically and even disappear for a while. As the passage connecting the

bowl and calyces with the tubercular focus expands, the amount of pus in the urine increases accordingly - pyuria becomes sharply pronounced. In cases where, in the course of antibacterial treatment, scar processes in the neck of the calyx or ureter cause their lumen to become overgrown, progress from the tuberculous focus stops and pyuria is not determined. Since pyuria or kidney tuberculosis passes for a long time in the absence of pain, it is often incorrectly interpreted as chronic pyelitis or pyelonephritis, which is why patients undergo long-term and unsuccessful treatment. Pyuria is the main and most constant symptom of kidney tuberculosis. The amount of pus in the urine can vary widely: from barely expressed turbidity to the formation of a strong sediment, similar to what is observed in pyelonephritis. The intensity of pyuria depends both on the degree of the destructive process in the kidneys and on the conditions of emptying the tuberculosis focus. In cases of "asymptomatic" pyuria, the possibility of kidney tuberculosis should be considered and a long-term urological examination should be carried out.

### C) Hematuria

Of clinical interest is the total hematuria occurring in the initial stage of the disease, of course, as the first symptom of a kidney tubercle, which is associated with their disintegration and the involvement of the walls of blood vessels in this process. Sometimes they are so intense that sometimes they force urologists to perform nephrectomies. Its intensity depends on the caliber of vessels affected by tuberculosis (tuberculosis manifested).

Sudden onset, irregular flow, intense nature, rapid disappearance - make these hematurias similar to those in kidney neoplasms.

Total hematuria can also be noted in the later stages of a tuberculous impression of the urinary system - when the process spreads to the mucous membrane of the urinary bladder (this is due to the presence of bleeding ulcers). Therefore, in the

presence of only total hematuria, one should also think about kidney tuberculosis, especially in young people.

#### D) Urinary disorder.

Dysuric disorders occur when the bladder is damaged and those pathological changes on the part of the mucous membrane of the bladder that are observed in tuberculosis. However, urination disorders can be noted even in the absence of changes on the part of the mucous membrane of the urinary bladder. In such cases, they should be considered as a manifestation of the renal-bladder reflex of I.M. Epstein believes that similar phenomena occur under the influence of tuberculosis intoxication.

The intensity of diuresis of these disorders in renal tuberculosis depends on the nature of specific changes in the bladder, the depth of the lesion and the localization of the process. Urinary disorders include: imperative urges, often nocturnal and then daytime urination, pain during and at the end of urination, urinary incontinence.

However, dysuria is not an early sign of the disease. Urinary disorder in kidney tuberculosis progresses rapidly, becomes painful and exhausting for patients. The frequency of urination fluctuates in a wide range: the majority of patients hold urine for an average of 1.5-2 hours, sometimes the frequency of urination is significantly increased - the urge occurs after 10-20 minutes (bladder neck, area of the Lieto triangle, a sharp decrease in urinary volume bladder, insufficiency of the closing apparatus).

#### E) Pain syndrome.

Pain in kidney tuberculosis can be either dull in the area of the affected kidney or sharp in the nature of renal colic.

Dull pains are caused by an increase in the kidney and its pinching in a poorly pliable capsule. They are localized in the hypochondrium or in the corresponding half of the lumbar region. They develop gradually, have a long-term course. Pains that are aching in nature can have a typical irradiating pain in the groin, thigh, scrotum, not related to the position of the patient's body or physical exertion. Sometimes pain can radiate to a healthy kidney.

In 21 percent of cases with kidney tuberculosis, acute pains such as renal colic are noted. They are no different from those in other kidney diseases. These pains are based on the following three factors: spastic contractions of the wall of the bowl or ureter, cicatricial changes in the lumen of the ureter and blockage of the lumen of the ureter by pus with fecal matter or blood clots - renal colic can end with its discharge. Cases of kidney tuberculosis with renal colic as the only symptom of the disease are of considerable interest.

#### F) General condition of patients.

The general condition of patients with a tubercle of the renal organs can be satisfactory even with a significant destructive process in the kidney. Sometimes patients have an increased appetite, are in excellent condition, without loss of working capacity, are engaged in physical education and sports.

Disturbance of the general condition occurs when the process spreads to the urinary bladder with nodular rash in the kidney parenchyma and does not go parallel to the intensity of the destructive process. With small foci, a poor general condition can be noted, and with large ones, good. The severe general condition of the patient should cause suspicion of the possibility of bilateral kidney damage.

#### G) Temperature of the patient.

Fever is not a characteristic symptom of kidney tuberculosis. The second appearance can be related to many points:

- 1) an additional infection that joined the tubercular process
- 2) nodular rash in the parenchyma with an ascending infection of the kidney
- 3) retention of the contents of the caverns in case of insufficient emptying of them with subsequent absorption of toxic products into the blood
- 4) the influence of extrarenal tuberculosis foci, mainly the process in the lungs.

In most cases, observing an increase in temperature has the character of a low-grade fever. Often, a high temperature (running along a hectic curve) is the first symptom of a process in the kidney that has already progressed far. The duration of the febrile period can be limited to one or several days, in other cases it lasts for weeks and months, which is accompanied by an exacerbation of the process in the kidney, increased pain and dysuric disorders.

In those cases when the cause of high temperature cannot be established, with a proven unilateral process in the kidney with significant destructive changes in the parenchyma, it is necessary to perform a nephrectomy: the operation quickly brings him out of the state of intoxication and preserves his life.

### **Combined kidney damage with tuberculosis and stones.**

Combined kidney damage by tuberculosis and stones cannot be considered as a case study. According to L.P. Kreiselburg, this condition is observed in 8 percent of cases. Until now, the following issues of this combination are considered debatable:

Is there any dependence between these processes or these lesions should be considered as a random combination of two independent diseases

Which of these processes is primary in the kidney and whether one of them can be recognized as a predisposing factor.

### **Forms of combined lesions according to Hotstein**

Tuberculosis of the kidney and stones in the kidney of the same name.

Tuberculosis in one, and stone in the other kidney.

Stones in both kidneys, tuberculosis in one.

Tuberculosis in both kidneys, and stones in one.

Tuberculosis and stones in both kidneys.

Most often, there is the first group, in second place is the second.

**Diagnostics.** Diagnosis of cavernous tuberculosis of the kidneys when using modern methods of endoscopic examination and contrast radiography of the urinary system does not present any particular difficulties. Difficulties in diagnosis mainly arise in the initial stages - the infiltrated form of tuberculosis of the kidney in the absence of destructive changes determined on an X-ray.

The main reason for this problem is insufficient familiarity of general practitioners with the clinical manifestations of tuberculosis of the urinary system, weak study of analysis and insufficient use of all possible methods of bacteriological and biological examination of urine for the presence of tuberculosis mycobacteria.

Diagnosis of kidney tuberculosis is designed to solve the following tasks:

1) to confirm the presence of a tuberculous lesion in the urinary system or to rule out the specific nature of the purulent-inflammatory process. It must be supported by evidence;

2) determine the spread of the process in the urinary system: is there damage to one or two kidneys, damage to the ureter and bladder;



3) to establish the volume of destructive changes in the kidney parenchyma, since their severity determines the choice of a conservative or operative treatment method.

4) Determine the functional state of the diseased and healthy kidney, as the acceptance of operative treatment largely depends on this. In addition, it is necessary to rule out the presence of tuberculosis foci in the systems, in particular, in the genital organs in men.

The diagnosis of kidney tuberculosis should be based on indirect signs, since these indicators do not determine the nature of the disease and cannot become elements of a specific diagnosis. But sometimes the presence of such non-specific criteria (aseptic pyuria, history of tuberculous lungs or other organs, chronic cystitis that does not respond to conventional therapy). It is often a valuable diagnostic aid, and sometimes the only possibility to make a diagnosis.

Palpation of the area of the location of the kidneys in the issue of specific diagnosis of tuberculosis of the urinary system is not of great importance. Positive results in some cases determine the condition of the diseased kidney (its enlargement, mobility) and perirenal tissue, which can be taken into account in predicting the complexity of surgical intervention. An enlarged kidney is palpable only with pyonephrosis. As long as the membranes are not involved in the tubercular process, the kidney is mobile. When the perirenal tissue is involved, the mobility of the kidney is sharply limited, sometimes a dense infiltrate is determined at this level. In some cases, when kidney tuberculosis is suspected, when it is not possible to establish specific symptoms of this disease, palpation of the thickened ureter during vaginal examination in women can to a certain extent speak in favor of tuberculosis.

Urine study. The data of the general analysis of urine do not make it possible to make a diagnosis of kidney tuberculosis in patients. They testify only to the

presence of a purulent-inflammatory process in the urinary organs without determining its etiology. The only proven diagnosis of kidney tuberculosis is the finding of mycobacterium tuberculosis in purulent urine.

Not being specific symptoms, some deviations found in the urine of these patients, however, give the right in some cases to speak in favor of kidney tuberculosis.

#### A) Persistent acidic reaction of urine.

This symptom is recognized by a number of authors as pathognomonic for kidney tuberculosis. According to H.M. Epshtein, the acidic reaction of urine with tuberculosis of the urinary tract can sometimes persist for up to 3 months. According to L.P. Kreisalburg's alkaline reaction of urine occurs in only 1.3 percent of patients. However, in his opinion, the acidic reaction of urine is not specific for tuberculosis of the urinary tract, as it can be noted in other diseases. However, a persistently pronounced acid reaction of purulent urine, which does not contain microbes, if even then mycobacterium tuberculosis is not found, is highly suspicious for tuberculosis of the urinary organs.

#### B) Proteinuria

According to R.M. Kronshtein, proteinuria should lead to thinking about kidney tuberculosis.

The diagnostic value of proteinuria is considered from several points of view:

- 1) albuminuria as an early symptom of kidney tuberculosis, which can occur long before the disease manifests
- 2) cylinderless albuminuria as a specific sign of renal tuberculosis
- 3) albuminuria as a manifestation of accompanying nephritis of the second kidney.

In the last category of patients, the amount of protein exceeds 1-2 percent and is usually accompanied by the presence of enzyme elements characteristic of nephritis. The amount of protein in the urine of patients with kidney tuberculosis, as a rule, does not reach a high level, it rarely exceeds one percent. In some cases, a large amount of protein should be attributed to accompanying hematuria.

### C) Tuberculosis mycobacteria in urine.

Clear evidence of tuberculosis kidney disease is the detection of mycobacterium tuberculosis in purulent urine. But their absence in urine does not deny this diagnosis. The fact is that the existing methods of their determination do not provide an absolute opportunity to detect tuberculosis mycobacteria in urine in all cases.

There are three methods of detecting tuberculosis mycobacteria in urine:

- 1) bacterioscopic - in fixed and specially colored smears from urine sediment
- 2) bacteriological - by sowing urine on special media
- 3) biological - infection of a guinea pig with infectious urine of a patient.

These three methods complement each other.

It should be noted that the biological method is also not always correct. A positive result can sometimes be obtained even in the absence of tuberculosis in the kidneys. S.D. Fedorov described a case from his practice when a nephrectomy was performed based on a positive result. Histology revealed a kidney tumor.

Sometimes inoculation does not give a negative effect, if few tuberculosis bacteria were found in the urine used for urine analysis or they are not virulent enough, in particular, under the influence of antibacterial treatment. A positive result of a biological test to detect the specific nature of the disease is determined within 84-90%.

#### D) Aseptic pyuria.

In the antibacterial period of aseptic pyuria, great attention was paid. Nowadays, the question of the diagnostic significance of aseptic pyuria in renal tuberculosis is being revised. Clinicians have observed the frequent combination of kidney tuberculosis with non-specific pyelonephritis. Most likely, as a result of the use of antibiotics, their bacteriological effect is reflected in the weakening of the activity of tuberculosis mycobacteria, which entails the growth of pus of harmful microbes.

True aseptic pyuria should include those cases where the absence of flora was detected by cultures.

Aseptic pyuria is not a specific sign of renal tubercle, it is also possible in other diseases. However, in case of kidney tuberculosis, aseptic pyuria is stable, permanent, while in other diseases it is determined only periodically. It is determined in 85% of patients with kidney tuberculosis (L.P. Kryselburg).

**Blood test.** Changes in the blood, which are noted in patients with tuberculosis of the urinary organs, are not specific only for this disease. They are important mainly in assessing the effectiveness of antibacterial treatment, prognosis and in establishing indications and contraindications for surgical intervention in exhausted and weakened patients.

A) *Specific blood changes* (bacteremia, serological reactions). The source of bacillemia can be any active tuberculosis focus in the body. Of course, it is not possible to determine which source caused this condition.

The opinions of different scientists differ regarding the meaning of bazallemia. We also received definitions and serological reactions of the type of complement rejection.

Acceleration of ROE, leukocytosis, lymphopenia are also not specific for renal tuberculosis.

**B) *Protein functions of blood plasma.***

Recently, in the diagnosis of tuberculosis of the urinary organs, the determination of the level of protein functions in the patient's blood plasma by the electrophoresis method is widely used. In the exudative stage of the disease, in the presence of tissue decomposition processes, the globulin reaction increases. Acute exudative inflammatory processes are characterized by an increase in the globulin fraction. Currently, the study of the state of protein fractions in the blood plasma is carried out after the previous administration of tuberculin in 24-48 hours. Based on this, the indicators found are more characteristic of tuberculosis infection, allowing differential diagnosis with non-specific processes in the urinary organs.

**C) *C-reactive protein.***

In recent years, research articles devoted to the study of C-reactive protein in tuberculosis infection have been published. Normally, this protein is not detected in humans.

Not being specific for any infection, it serves as an early indicator of developing acute inflammatory and destructive processes in the patient's tissues. C-reactive protein in patients with kidney tuberculosis appears early, is the most stable compared to changes in the blood protein formula, and can be used as one of the early signs of the disease.

Endoscopic studies. Cystoscopy is one of the leading methods for diagnosing tuberculosis of the urinary organs. It can detect specific elements of tuberculosis infection in the bladder and confirm the diagnosis by the same documenting method. At the same time, it is possible to find out the side of the infection, and in combination with the indigo carmine test, the functional state of the sick and healthy

kidney. Sometimes the bladder is so reduced that cystoscopy becomes impossible, the minimum capacity of the bladder is sufficient for cystoscopy (40-50).

Specific changes in the bladder can be expressed in the form of tubercular tubercles and ulcers. This is enough to determine the diagnosis. Tubercles have the appearance of yellowish formations the size of a pinhead or hemp seed, surrounded by an auxiliary rim located along blood vessels, are concentrated more often in the area of the mouth (mouth) of the ureter of the affected kidney. Moderate progression of the process in the bladder, tubercles are much less common.

Tuberculosis ulcers are a product of the decay of tubercles, they correspond to a later stage of bladder damage. Their peculiarity is that they are linear according to the localization of former tubercular nodules.

The mouths of the ureters affected by tuberculosis often undergo a number of changes. Accompanied by infiltration of the mouths and scarring processes, they are so characteristic that many authors give them the meaning of specific: gaping of the mouth, its retraction, star shape with uneven edges, bullous edema. The mucous membrane of the urinary bladder can be covered with a fibrinous plaque in some areas. Cystoscopy usually reveals changes in the mucous membrane of the urinary bladder on the side of the kidney lesion.

**Chromocystoscopy** sets itself the task of:

Identify the side of kidney damage in the absence of subjective complaints.

Determination of the functional state of each kidney, which is very important when deciding on the choice of a treatment method, and in particular, an operative one.

However, the intensity of indigo carmine excretion does not always correspond to the severity of kidney damage. Sometimes, when the excretory function is preserved, massive destructive changes are observed and vice versa.

Then, in addition to the above-mentioned methods, according to S.P. Fedorov, it is necessary to perform catheterization of the ureters. This achieves:

1) obtaining urine directly from the kidneys to confirm the source of pyuria and the possibility of detecting tuberculosis mycobacteria.

2) checking the patency of the ureter of the affected side and thereby identifying narrowings that are important in the diagnosis of kidney tuberculosis.

### **X-ray examination.**

X-ray examination for tuberculosis of the kidneys and ureter occupies a central place among other diagnostic methods.

X-ray diagnostics for tuberculosis of the urinary organs should solve the following tasks:

- a) to confirm or exclude the tubercular nature of the lesion.
- b) determine the volume of destructive changes in the urinary organs
- c) specify the prevalence of the process in the urinary system
- d) determine the functional state of both the diseased and healthy kidney

Only after that, you can decide on the method of treatment. In the diagnosis of tuberculosis of the kidneys, the following can be used: inspection radiography, ascending pyelography, internal urography, tomography, urocytography and renal angiography.

Survey radiography in the diagnosis of urinary organs can become a very valuable method for recognizing tuberculosis of the kidneys. On the pictures, you can

find petrifications, the presence of silted areas, the presence of accompanying concretions, etc.

Ascending pyelography gives a complete picture of destructive changes in the kidney (one or many cavities), changed pelvic cups, etc.

When the process spreads to the ureter, characteristic specific changes occur in this organ.

a) straight direction of the ureter - it loses its curvature and takes the form of a stretched string between the bladder and the kidney

b) narrowing of the course of its lumen above which a number of consecutive expansions appear. In some cases, the anatomical condition of the entire ureter is revealed, its lumen sometimes reaches a significant width.

Excretory urography is less important for recognizing kidney tuberculosis than ascending pyelography, especially in the early stages of the disease. However, it becomes the only invaluable method in diagnostics when ascending pyelography is technically impossible to perform in the presence of an ulcerative lesion of the urinary bladder with a decrease in its capacity and in the case of insurmountable obstacles along the course of the ureter, which have developed as a result of tuberculous strictures.

It indicates the destructive changes in the kidneys and the degree of preservation of kidney functions.

Indications for excretory urography in renal tuberculosis can be divided into two groups: absolute and relative. Those cases in which it is not possible to perform an ascending pyelography should be classified as absolute (strictures of the urethra, small capacity of the bladder, ureter obstruction), and all cases of kidney tuberculosis are relative.



TOMOGRAPHY - can be performed to obtain auxiliary data.

a) performance of the shadow of the kidney, when it is not achieved by inspection radiography or excretory urography.

b) detection of specific changes in the parenchyma of the kidney and in the bowls, when ascending pyelography is impossible, when the usual excretory urography does not give a clear contrasting shadow of the bowls or cavities

c) to detect in the kidney parenchyma large caverns covered with a shadow, expanded cups or bowls.

UROKIMOGRAPHY - reflects a violation of the motor and evacuation function of the affected organs (ureter, character, frequency, rhythm and direction of the palsy).

CYSTOGRAPHY - allows you to detect the limitation of distension of the urinary bladder and the presence of vesicoureteral reflux (if present). It is possible to establish the side of the lesion when cystoscopy and catheterization of the ureters are impossible.

Renal angiography. The disease can be established only with a developed process. The number of vessels at the level of the tuberculous focus decreases, their branching becomes diverse, contours are uneven. The terminal vessels of the branch disappear, roughly breaking off near the very focus of the pathological process. Renal angiography acquires great diagnostic value in the case of extensive so-called tubercular infiltrates of the kidney.

Renal angiography can be very useful for clarifying the limits of a specific process in patients who are proposed to undergo kidney resection.

**Treatment.** Until relatively recently, in relation to the treatment of unilateral tuberculosis of the kidney, there was a unanimous opinion that the only rational method is the early removal of the affected kidney.

S.P. Fedorov R.M. Fronshtein in all his writings claimed that in the case of confirmation of unilateral tuberculosis of the kidney, it is necessary to perform nephrectomy. At the same time, it is recommended to remove the kidney as early as possible, before the tuberculous process has spread to the bladder or the second kidney.

The discovery of antituberculosis drugs and their introduction into the clinic created a new era in the treatment of tuberculosis.

The latter are divided into two groups:

I) main antibacterial drugs (1st line) and 2-reserve drugs (second line). First-line drugs include streptomycin PASK and hydrazyl derivatives of isonicotinic acid (ftivazid).

Treatment begins, as a rule, with the use of first-line drugs. using two or three drugs of this series at the same time. Such combined use of the drug increases the effectiveness of therapy and prevents the development of drug resistance in bacteria.

Streptomycin is administered intravenously, usually 0.5 g per day.

Ftivazid is initially prescribed up to 0.1 g three times a day, intravenously, and then, if the drug is well tolerated, 0.3 g three times a day. Ftivazid is well tolerated by patients and does not cause side effects. Only some patients have peresthesias of the intercostal nerves and nerves of the limbs, convulsions and angiotic attacks. Therefore, ftivazide is contraindicated in coronary insufficiency, after a myocardial infarction, in epilepsy, heart defects, with decompensation and organic diseases of the central nervous system.

Ftivazid reduces blood coagulation.

Tubazid is prescribed internally at 0.15-0.2 g per day. Has significant toxicity. Duration of treatment is 3-4 months.

Saluzide 0.5 2-3 times a day (10% -5-10 ml intramuscularly, intravenously, subcutaneously)

Metazid 0.2-0.5 g 2 r.v.d. Less toxic.

Larusan 0.1-0.3 g 2-3 times per day.

Side effects: dyspeptic disorders, headaches, changes in white blood, in particular significant zosinophilia.

Contraindications: Liver disease, organic diseases of the central nervous system, glomerulonephritis.

Due to the great success of antibacterial therapy, indications for surgical treatment of tuberculosis of the urinary organs have narrowed significantly. Absolute indications for operative treatment today are polycavernous tuberculosis of the kidney, tuberculous pyonephrosis. A. I. Mayants refers to a non-functioning kidney that is osmolated as an indication for nephrectomy.

The main method of surgical intervention for kidney tuberculosis is its removal. In addition to this radical operation, organ-sparing ones such as kidney resection and cavernotomy have also been used today.

**Contraindications to nephrectomy.** Absolute: a) tuberculous cavernous process in the second kidney with bilateral damage; b) significant insufficiency of the second (healthy) kidney; c) generalization of the tuberculosis process in the body.

Relative: severe general condition of the patient, tuberculous process in the lungs in the stage of an infiltrative outbreak, pregnancy in the second half or passing

with complications, exhaustion of the patient, if it is associated with the tuberculous process in the kidney, is not only not a contraindication to nephrectomy, but dictates its implementation.

**Plastic operations on the ureter.** Replacement of the ureter in case of narrowing of the pelvic-ureteral segment by a loop of the small intestine (A.P. Frumkin).

2) Technique of surgery for structures in the pelvic segment of the ureter according to Bang-Huk-Boar.

Bladder plastic surgery.

a) Intestinal plastic surgery (loop of the ileum of the small intestine) (S.D. Holigorskyi, A.M. Gasparyan) or segment of the sigmoid colon (Kyuss, A.P. Frumkin).

Tuberculosis of genital organs.

We considered the pathogenesis above. What factors contribute to the development of a tuberculous focus in the organs of the reproductive system in men.

a) Injury. As recognized by A.I. Mayantz, hemorrhage and disruption of innervation mobilizes the essential infection. Currently, it is recognized that trauma gives an impetus to the development of an already organized patent focus.

b) Transferred gonorrheal inflammation of the urethra and gonads. However, this is a debatable issue.

c) Sexual excesses. Theoretically, the influence of an active sexual life on the development of genital tuberculosis is completely permissible. The predominance of the disease in young people during the period of increased sexual function is a confirmation of the stated assumption.

Tuberculosis of the genital system in men is rarely localized in one of its organs. In most patients, a tubercular process can be detected in a number of gonads during clinical examination.

They are most often found in the combined lesion of the epididymis in combination with tuberculosis of the prostate gland and the vas deferens.

More recently, external organs - testicles and epididymis - were recognized as the primary localization of tuberculosis in the genital area. However, Oppenheim's experiments and Lev's confirmations proved that the primary focus in the reproductive system is the prostate gland and seminal vesicles, hence the process of hematogenous, or more often via the seminal pathways antiperistaltically spreads to the testicle and epididymis.

The third group of authors B. N. Holtsov, Gohen admit the possibility of primary infection of any organ of the sexual sphere without any selective tendency of one or another of them.

### **Patho-anatomical picture of the prostate and seminal vesicles.**

Specific changes in the prostate gland develop both in the follicles themselves and on the walls of the excretory ducts. As a result of the fusion of tuberculous nodules and their curdled decay in the parenchyma of the gland and seminal vesicles, decay cavities (caverns) can form. In the future, the contents of these cavities break through and enter the urethra or the near-prostatic tissues, forming persistent purulent fistulas on the perineum or buttocks. In rare cases, the contents of the caverns open into the lumen of the rectum. In rare cases, petrification of caseous contents is possible, which should be distinguished from prostate stones in the differential diagnosis.

**Semiology.** The clinical course of tuberculosis of the external genital organs does not always develop in the same way.

There are two clinical forms of the disease: acute and chronic. Why there is such a division is unknown to this day. The acute form begins with sudden, pulling pains in the scrotum with irradiation along the spermatic cord and in the sacrum. 3 swelling of the affected side of the scrotum will appear. The skin reddens, becomes shiny and tense. The spermatic cord and especially the vas deferens thickens and becomes sharply painful, the body temperature rises, the patient's condition is extremely depressed. Usually, by the end of the second week, the acute symptoms subside and the disease turns into a chronic form.

The chronic form develops gradually, and is not rarely determined during a random examination of the patient. Painful, dull compression along the course of the seminal canal, in the body and tail of the epididymis, a bumpy dense formation is noted. The boundary between the appendix and the testicle is delineated, which does not happen in the acute form, in which the appendix and the testicle merge into one conglomerate.

In the future, the density merges and is subject to melting. Abscesses grow together with the skin of the scrotum, the skin reddens, thins, the abscess comes out with the formation of a persistent, long non-healing fistula. Tuberculosis mycobacteria are rarely found in pus. In some patients, fistulas sometimes heal with a dense retracted scar.

**Diagnostics.** Diagnosis of epididymitis or orchoepididymitis does not cause special difficulties. The main task is to clarify the nature of the lesion of the appendix, that is, to differentiate tuberculosis of the external genital organs from gonorrheal processes and especially from the category of so-called non-specific epididymitis, which is often encountered in our country and in our time.

The rational choice of the treatment method depends on the establishment of such a diagnosis, and in particular, the solution to the issue of indicators for surgical treatment-removal of the appendix.

It is necessary to know that non-specific epididymitis in most patients has a sluggish, torpid, chronic course and it is very difficult to distinguish them from specific (tubercular) lesions. Unconditional evidence of tubercular damage to the appendix or testicle is the presence of fistulas in the scrotum. The finding of the tubercular process in two organs and especially in the urinary system can be very essential in determining the tubercular nature of the lesion of the appendix.

Is a specific diagnosis of tuberculosis of the external genital organs possible?

A positive Perke reaction in adults indicates only the presence of a tuberculosis focus in the body, without determining the degree of its activity and, of course, localization.

Serological reactions-rejection of complement according to Bordet-Chat (however, it is not universal).

Determination of tuberculous bacteremia by bacteriological examination of blood for the presence of the causative agent of tuberculosis (Lakhtenstein).

**Treatment.** Specific therapy (tuberculin treatment) is not justified. Patients in whom the diagnosis was made early from the onset of the disease and in whom there is a tendency to predominate productive changes, should be treated only conservatively (but at least 7-9 months).

**X-ray therapy.** If in the 1930s X-ray therapy was used quite widely, today, with the availability of antibiotics, the wide use of X-ray therapy is inappropriate.

**Surgical treatment.** Surgical treatment is the main and most rational method of treatment. It should be recommended for the following reasons.

a) tuberculosis-affected appendix as an organ has lost its functional significance.

b) a tubercular focus in the epididymis has a constant threat of spreading the process in the testicular parenchyma.

c) the presence of a unilateral tuberculosis lesion of the external genital organs very dangerously reflects on the opposite side, both in terms of the possibility of its damage by a specific process, and in terms of the toxic effect on the function of the not yet affected organs of the genital system of the opposite side.

Four types of surgical intervention:

- 1) removal of only the epididymis
- 2) removal of the appendix together with the testicle
- 3) organ-preserving operations, resection of the testicle, opening and treatment of the cavern
- 4) radical operations on pelvic genital organs.

Prostate tuberculosis.

Unfortunately, tuberculosis of the prostate cannot be considered a rare disease (about 10 percent of cases). Always hit the seminal vesicles.

Most patients with tuberculosis of the prostate gland are completely asymptomatic. However, when the process spreads to the thickness of the organ, the following occur: frequent, painful or difficult urination, terminal hematuria. Dull pains in the perineum are possible. When opening the cavity in the posterior urethra, pyuria is manifested. Ulcers are formed, indicating the presence of tuberculosis in the prostate. The general condition of patients in the early stages of the disease is usually



satisfactory, with cavernous lesions general weakness, rapid fatigue, subfibrous or elevated temperature, weak appetite are noted.

Taking into account the fact that the prostate gland is more often affected in men with pulmonary tuberculosis, a mandatory rectal digital examination, as well as prostate secretion, is indicated in all these patients.

The prostate gland in such patients is enlarged. Its surface may be bumpy in some areas, the consistency is dense and elastic.

In all these patients, it is necessary to examine the urine in two portions - the presence of leukocytes in the second portion and the absence in the first. It is necessary to investigate the sperm culture for mycobacterium tuberculosis.

In some cases, the diagnosis can be made after ascending urethrography. The contrast mass penetrates the parenchyma of the prostate gland and reveals decay cavities.

Vesiculography can be of great help (insufficient filling of the testicles of the seminal vesicles, eaten contours of the urinary tract). Differential diagnosis is considered in detail in practical classes.

**Treatment.** Tuberculosis of the prostate gland and seminal vesicles is currently treated only conservatively, by using antibacterial chemopreparations actively and for a long time.

Effective antibacterial treatment of the prostate gland in sanatorium-resort conditions in combination with rational nutrition, vitamins, climate and the use of koumiss. The treatment is periodically repeated.

Surgical treatment is used only in limited cases - when a decay cavity is formed in the thickness of the prostate gland or when specific abscesses break into the paraprostatic tissue with the formation of spotting abscesses, purulent or urinary

fistulas. In these cases, caverns are opened with their subsequent drainage and the use of antibiotics and chemotherapy drugs.

A number of authors (A.I. Mayants, V.D. Grund) prescribe vitamin D in an alcohol solution - 25-30 units per day for 3 months, along with treatment with streptomycin, in the case of a normal state of the urinary tract.

Indications and contraindications for sanatorium-resort treatment of tuberculosis and the approximate length of stay of these patients at the resort:

1) Bilateral tuberculosis of the kidney without detection of destructive changes in the parenchyma on the x-ray. The period of stay at the resort is 2-3 months.

2) Unilateral tuberculosis of the kidney with destructive changes in the papillae, with one or two small caverns. Duration of medication. at the resort for 3-4 months.

3) Tuberculosis unites the kidneys. The period of stay at the resort is determined individually, but at least 3 months.

4) Patients referred after nephrectomy for kidney tuberculosis. It is advisable to send them to a sanatorium-resort treatment within 6-12 months after the operation.

The term of stay is 2-3 months. After returning, these patients must be observed in local anti-tuberculosis dispensaries for 2 years.

5) Patients with bilateral tuberculosis of the kidneys with moderately pronounced changes in each of them with satisfactory function. The term of stay is 4 months.

6) Patients with residual changes in the bladder after nephrectomy. Stay at the resort for at least 3 months.)

7) Tuberculosis of testicles and their appendages. These patients can be referred to the resort both before and after surgery.

8) Prostate tuberculosis. The duration of stay at the resort is 2 months, with repeated treatment after one year.

## INTRAVENOUS UROGRAPHY IVU

The IVU consists of a series of plain films taken after administration of an intravenous injection of a water-soluble iodine containing contrast medium.

### *Indications*

#### 1. Tuberculosis of the urinary tract

Traditionally the patient was prepared with a period of 4 h starvation and fluid deprivation and the bowel purged with a strong laxative. Occasionally the patient will feel nauseated after the IVU injection and rarely there will be a severe reaction with the need for cardiovascular and occasionally cardiopulmonary support. With this in mind, it seems reasonable to persist with avoidance of food for 2-4 h prior to the procedure.

### *Radiological anatomy*

The kidneys are typically located at the level of the upper lumbar spine with the right kidney slightly lower than the left. They generally lie with their axes along the psoas muscles with the upper pole slightly more medial than the lower. Alterations in position and orientation of the kidneys may be related to congenital anomalies such as pelvic kidneys or may be secondary to mass effect from an adjacent lesion.

The size of the kidneys is somewhat variable depending on age and sex of the patient, but on the intravenous urogram, the kidneys normally range from 11 to 14 cm. The right kidney is typically slightly smaller than the left.

The kidneys should be symmetric in size with a discrepancy greater than 2 cm requiring an explanation. There are a number of causes of abnormal renal size, ranging from incidental anomalies such as congenital renal hypoplasia to significant conditions such as renal artery stenosis (small kidney) or infiltrating renal neoplasm (large kidney).

The kidneys should have a reniform shape and a smooth contour.

The intrarenal collecting system consists of calyces, infundibula, and the renal pelvis. Normally, each kidney consists of 7 to 14 evenly distributed calyces.

The normal ureters exhibit continual peristalsis and on a single film, it is uncommon to demonstrate the entire length of both (or even either) ureters.

They will often demonstrate smoothly narrowed areas (especially at the pelviureteric junctions and as they cross the iliac vessels in the pelvis) and more relaxed capacious areas. This is normal. Proximally, the ureter passes over the psoas muscle and should generally lay just lateral to the lumbar spine. The midportions of the ureters course over the lateral sacrum with the distal portion gently curving laterally in the pelvis before entering the bladder.

The ureter should be inspected for filling defects, which can be caused by stones or tumor, and should be symmetric in size. Evaluation of the ureteral course is important. Deviations of the normal ureter generally suggest extrinsic diseases, such as mass lesions. However, in patients with large psoas muscles the ureters may be displaced laterally as an incidental result.

The bladder is an oval to rounded structure that normally lies just above the pubic symphysis on the IVU. In women, the dome of the bladder may normally be indented by the uterus. These normal findings must be differentiated from abnormal extrinsic mass effects. Bladder wall thickness can sometimes be visualized and assessed,

especially if thickened. Additionally, the bladder mucosa should be scrutinized for irregularity or filling defects that may suggest a mass.

### ***Patient preparation***

- blood urea and serum creatinine level should be within normal limits
- if patient is asthmatic premedication in the form of steroids is administered two days prior
- fasting after 10 pm (previous night) (as contrast injection sometimes induces nausea which might lead to vomiting and aspiration)
- patient should be well hydrated (dehydrated patients are prone for renal damage)
- bowel preparation is necessary, as gas and fecal matter filled bowel loops will obscure the kidney shadows
- low residue diet with plenty of oral fluids, the day previous to the IVU

### ***Contrast media***

Contrast materials currently in use are excreted almost exclusively by glomerular filtration, with subsequent concentration in the renal tubules and progressive opacification of the urinary tract.

They are two types:

1. ionic (urograffin, angiograffin)
2. non-ionic(omnipaque, ultravist)

Ionic contrast media have a higher incidence of reaction but they are cheaper as compared to the non-ionic contrast media.

### ***Procedure***

Patient is placed in supine position. The patient is asked to void the bladder before the procedure.

Contrast media is injected intravenously into a prominent vein in the arm. Test injection of 1ml of contrast is given and patient observed for 5 min for any contrast reactions. Then the rest of the contrast is rapidly injected within 30-60 seconds.

The dose of contrast media is 2 ml/kg body wt.

Intravenously injected iodinated contrast is excreted primarily by glomerular filtration in the kidney, opacifying the urinary tract as it progresses from the kidney through the ureter and to the bladder. Capturing this sequential "opacification" on radiographs is the fundamental basis of the IVU. There are many variations in the filming sequence for the urogram that are acceptable as long as it optimizes visualization of specific anatomy of the urinary tract during maximum contrast opacification. Optimal visualization of the kidney is accomplished very early in the examination. Within 1 to 3 minutes after injection, the contrast bolus is filtered by the glomeruli and fills the nephron, resulting in intense opacification of the renal parenchyma; this phase of contrast opacification is called the *nephrogram*.

The kidneys should be evaluated for:

- their position
- orientation
- size
- contour
- radiographic density.

Soon after the nephrographic phase, contrast begins filling the intrarenal collecting system including the calyces and renal pelvis. This portion of the study is termed the *pyelographic phase*.

### ***5-10 min film***

Shows nephrogram, renal pelvis

### ***15-20 min film***

A complete visualization of the pelvicalyceal system entire ureters is possible in this film, especially with the patient in prone position as the ureters will be antedependent in prone position.

### ***30-35 min film***

A complete visualization of the urinary tract: kidney, ureter, bladder can be done and bladder distension can be evaluated in the later film.

The series is varied according to the individual patient. Renal obstruction may require a delayed study up to 24 hours to outline the pelvicalyceal system.

### ***Advantages***

- IVU is low cost
- anesthesia is not needed
- detailed anatomy of the collecting system
- rapid overview of the entire urinary tract
- demonstration of calcifications
- demonstrate renal function and allow for verification that the opposite kidney is functioning normally
- it is sensitive for obstruction
- can show non opaque stones as filling defect
- IVP is an excellent modality to diagnose medullary sponge kidney and papillary necrosis .

### ***Disadvantages***

- contrast material must be avoided in patients with a history of allergy, hay fever or asthma until steroid cover has been given; those on metformin must stop this

drug for 24 h before any contrast. These groups cannot safely undergo an emergency IVU.

- the differentiation from a phlebolith is difficult, especially when there is no ureteric dilatation proximally
- contraindications renal insufficiency
- contraindications hepatorenal syndrome, thyrotoxicosis, pregnancy
- do not differentiate solid or cystic lesion
- requires contrast medium and radiation
- missing small stones
- quality of study may be limited by inadequate bowel preparation
- inconvenience of a long filming sequence

### ***Retrograde and antegrade pyelography***

Direct injection of water-soluble iodinated contrast material is a useful method of examining various regions of the urinary tract. The advantage of this method of evaluation is the direct control over the contrast injection rather than reliance on secondary excretion from the kidney.

## CYSTOGRAPHY

Imaging of the bladder is performed with a cystogram.

### ***Indications***

- the extent of vesicoureteral reflux
- urinary stress incontinence can be assessed
- urinary tract infections
- suspected obstruction
- suspected bladder trauma or rupture
- detection tumor
- detection diverticula



- detection stones
- to investigate suspected fistulas involving the bladder (usually into the gastrointestinal tract, occasionally elsewhere such as the vagina)

### ***Procedure***

A catheter is placed into the bladder and contrast material is then injected. The contrast material is optimally injected under fluoroscopic observation but occasionally is performed with only static conventional radiographs, such as in the trauma setting. Anatomic considerations and evaluation are similar to the IVU with a few caveats.

The method is useful for outlining tumors of the bladder when intravenous urography has been unsuccessful or equivocal.

One advantage to cystography is that vesicoureteral reflux can be evaluated during the conventional cystogram unlike during IVU.

### ***Cystography can be classified into three groups:***

- micturiting cystourethrography (MCUG)
- dynamic cystography
- simple cystography.

The MCUG is primarily performed for an investigation of childhood.

Dynamic cystography is part of the urodynamics investigation of the lower urinary tract.

Simple cystography is a relatively frequently performed and straightforward investigation in the adult.

### ***Patient preparation***

For two days before the examination, medical experts recommend limiting intake of products that provoke flatulence. On the eve of the research (in the evening), as well as immediately before cystography (morning) held enema.

### *Advantages*

- these imaging tests provide the basic anatomy of the bladder and urethra
- show urethral movement
- low cost
- wide availability
- general familiarity

### *Disadvantages*

- cystography is contraindicated spend in acute inflammation of the bladder, urethra, scrotum, prostate and seminal vesicles (If the research is still necessary, the doctor can perform a downward cystography)
- the catheter could damage the urethra, bladder or nearby structures
- they require catheterization
- the images contain no information about the pelvic musculature and adjacent soft tissue structures
- only structures in direct contact with the urethral and bladder lumen opacify with contrast.

### **Contraindication.**

- 1) Unilateral polycavernous tuberculosis of the kidneys.
- 2) Tuberculous pyonephrosis.

In addition, the presence of marked changes on the part of the urinary bladder is also a contraindication for the referral.

**Practical task:** describe x-ray images of virtual patients.



Fig. 1. Tuberculosis. Cavern (tubercular track). JPG



Fig. 2. Tuberculosis. Cavern (tubercular track). JPG

## X-RAYS OF KIDNEY TUBERCULOSIS



Figure 1. Urinary stone disease. Bowl stone. Caverns of the right kidney. JPG

## TEST TASKS

1) What causes tuberculosis of the genitourinary system?

- a) streptococci
- b) staphylococci
- c) mycobacteria
- d) fungal lesions

2) Who is most often affected by tuberculosis?

- a) children up to 10 years old
- b) young people aged 20-45
- c) elderly people
- d) long-lived people

3) The main signs of kidney tuberculosis:

- a) hematuria
- b) cylindruria
- c) leukocyturia
- d) glycosuria

4) What are the changes in urine in patients at the beginning of tuberculosis of the genitourinary system?

- a) proteinuria more than 1.0-2.0 g/l
- b) proteinuria in the range of 0.033-0.99 g/l
- c) persistent isosthenuria

d) specific gravity fluctuates within the norm

5) Use of radiography for the purpose of:

a) decisive method in the diagnosis of tuberculosis

b) auxiliary method of diagnosis of tuberculosis  
6) The main signs of tuberculosis of the ureters:

a) deformation due to strictures

b) intact evacuation function

c) distinct anatomical narrowings

7) Where does a stricture in the ureter most often occur?

a) in the upper third

b) in the middle third

c) in the lower third

d) there are no changes

8) Main signs of tuberculosis of the urinary bladder:

a) reduced bladder (microcyst)

b) neurogenic bladder

c) unchanged detrusor

d) there is no bladder destruction

9) The main signs of tuberculosis of the prostate gland:

a) not increased

b) very painful during palpation

- c) the presence of fistulas on the perineum
- d) doesn't care about anything

10) With which diseases should a differential diagnosis be carried out first of all?

- a) prostate cancer
- b) prostatitis
- c) stone-prostate disease

11) The main signs of tuberculous epididymitis:

- a) absence of pain
- b) the appendix is enlarged
- c) the appendix is determined only by palpation
- d) the appendix is painful and prevents walking

12) The main signs of tuberculous orchitis:

- a) the testicle is not enlarged
- b) the testicle is enlarged, painful
- c) the testicle is enlarged, there are fistulas
- d) the testicle is reduced, not painful

13) Treatment of nephrotuberculosis is only conservative when:

- a) significant destruction of the kidney (polycavernosis)
- b) a single cavern
- c) with a large number of strictures of the urethra
- d) with a functional kidney



14) The most effective antibiotics that must be used to treat nephrotuberculosis:

- a) vancomycin
- b) isoniazid
- c) ceftriaxone
- d) kanamycin

15) Indications for operative treatment are:

- a) polycavernous form of nephrotuberculosis
- b) a single cavern
- c) the first signs of the disease (hyperthermia, chills)
- d) dysuria (night and day), polyuria

Correct answers to test tasks on nephrotuberculosis:

| № question | Correct answer | № question | Correct answer | № question | Correct answer |
|------------|----------------|------------|----------------|------------|----------------|
| 1          | <b>c</b>       | 6          | <b>a</b>       | 11         | <b>d</b>       |
| 2          | <b>b</b>       | 7          | <b>c</b>       | 12         | <b>b</b>       |
| 3          | <b>a</b>       | 8          | <b>a</b>       | 13         | <b>b</b>       |
| 4          | <b>c</b>       | 9          | <b>b</b>       | 14         | <b>b</b>       |
| 5          | <b>a</b>       | 10         | <b>a</b>       | 15         | <b>a</b>       |

## SECTION 2

### ACUTE AND CHRONIC RENAL FAILURE

**Purpose of training:** To teach students to timely diagnose, provide primary care and treat acute renal failure (ARF), clearly define indications and contraindications for extracorporeal hemodialysis in ARF, diagnose and treat chronic renal failure (CRF), determine the doctor's tactics when combined with ARF and pregnancy and gynecological diseases, diagnose and treat neurogenic hypertension.

**Didactic purpose:**

1. Perenal, renal and postrenal factors of acute renal failure
2. Pathogenesis of acute renal failure
3. Stages of acute renal failure, their clinical manifestation
4. Stages of chronic renal failure, their clinical characteristics
5. Methods of treatment of acute renal failure
6. Methods of treatment of chronic kidney failure

**List of skills:** to know the basic concepts, to master the tactics of diagnosis and treatment of acute and chronic renal failure

1. Symptoms of nephrological diseases.
2. Examination of urine: daily amount, relative density, presence of protein, elements of urine sediment.
3. General blood analysis: number of erythrocytes, hemoglobin, leukocytes.
4. Biochemical research: residual nitrogen, urea, creatinine, serum potassium.
5. Methods of palpation and percussion of kidneys, bladder.

6. Pain symptoms, their diagnostic significance.
7. Types of hematuria, their clinical significance. Tactics of the doctor.
8. Functional kidney tests.

## **ACUTE AND CHRONIC RENAL FAILURE**

Renal failure is a syndrome that develops as a result of impaired renal blood flow, glomerular filtration, tubular reabsorption and secretion, as well as the concentrating ability of the kidneys and is characterized by azotemia, impaired electrolyte-water balance and acid-base balance.

According to the course, acute and chronic renal failure are distinguished. Acute renal failure occurs suddenly as a result of acute, often reversible kidney damage. Chronic kidney failure develops gradually as a result of progressive irreversible loss of functioning kidney parenchyma.

Acute kidney failure can occur at any age, but most often in 20-40 years.

**Etiology.** The causes of acute renal failure are disorders of general and renal hemodynamics, acute effects on the kidneys of exogenous poisons, infectious agents, acute occlusion and damage of renal vessels, obturation and compression of the urinary tract, rarely kidney injury.

Etiological factors of acute renal failure can be combined into groups:

- 1) prerenal factors - a sharp decrease in the volume of circulating blood and a decrease in blood pressure during shock or bleeding, loss of a significant amount of water and electrolytes during profuse diarrhea, prolonged vomiting, burns, as a result of (especially prolonged) use of diuretics.

2) Renal factors - exogenous poisoning with nephrotic poisons (salts of heavy metals, chlorinated hydrocarbons), alcohols, strong acids, poisons of plant origin, for example, mushroom poison, insect poison, some drugs in their overdose), as well as damage to the kidneys in the case of renal occlusion vessels (thromboembolism), with acute inflammatory diseases (acute glomerulonephritis, acute pyelonephritis), some acute infectious diseases.

3) postrenal factors - occlusion of the urinary tract by a stone, compression of the ureters by a tumor, hematoma, ligature, etc.).

Acute renal failure can also occur after bilateral nephrectomy for vital indications and with traumatic loss of both kidneys (the so-called Arenal condition). About 90% of cases of ARF are associated with kidney damage as a result of hemocirculatory disorders and poisoning with nephrotoxic poisons.

**Pathogenesis.** Necrotic changes in tubules in acute renal failure due to hypoxia develop as a result of hemodynamic disturbances or histotoxic effects. Violation of renal blood flow and reduction of glomerular filtration are the most important mechanisms of acute renal failure. In the origin of oliguria or anuria, a significant role is played by indiscriminate absorption of glomerular filtrate through the wall of the damaged renal tubule and interstitial edema. Compression of renal tubules and their blockage by pigment cylinders are only additional factors in the development of oliguria. A major role in the pathogenesis of acute renal failure belongs to humoral influences, especially from the renin-angiotensin system, which can cause spasm of kidney vessels and ischemia. There is an opinion that damage to the tubules entails a violation of sodium reabsorption and an increase in its concentration in the area of the dense spot, to which the Juxtaglomerular complex responds by increasing the secretion of renin.

High activity of renin causes spasm of lead glomerular arterioles (bringing vessels) and reduction of glomerular filtration. Vasoactive substances such as histamine, serotonin, vasopressin are of certain importance in the pathogenesis of acute renal failure, and some steroids increase tissue sensitivity to hypoxia. It has been established that in this form of the disease there is shunting of renal blood flow, that is, blood passing through the cortical vein of the kidney enters the system of direct arterioles of the cerebral vein through the glomeruli of the renal corpuscles of the juxtamedullary nephrons. The well-known spasm of interlobular arteries and cortical ischemia of the kidneys also has a certain role in the pathogenesis of cortical necrosis.

**Pathological anatomy.** Morphological changes of the kidneys in acute renal failure depend on the etiology of the period of the disease, the timeliness, nature and scope of treatment measures.

The macro- and microscopic picture of ischemic changes in the kidneys, or the so-called shock kidney, is more often observed in traumatic shock, blood loss and their nature for processes of exo- and endotoxic origin. Electron microscopic studies indicate severe and deep damage to the renal tubules. Rupture of renal tubules (tubolorhexis) is considered typical in shock. Shock is accompanied by hemolysis and myolysis, morphological changes in the kidneys correspond to hemoglobinuric and myoglobinuric nephrosis. After infusion therapy, the morphological picture of kidney tubule lesions may change - stretching of the brush border of nephrocytes occurs.

Thrombosis of glomerular capillaries, as a manifestation of disseminated intravascular blood coagulation, is observed more often in bacteremic shock and pathological pregnancy. This also includes symmetrical cortical necrosis of the kidneys.

There are total, subtotal, segmental and small forms of necrosis. In the total form, the renal pyramids are preserved, in the subtotal form, islands are found in the cortical vein, not a tissue lesion, in the segmental foci of necrosis, they look like infarcts surrounded by hemorrhages, small forms can be detected only by microscopic examination. With long-term acute renal failure, the necrotic cortical tissue of the kidney thins.

Poisoning with ethylene glycol (antifreeze) leads to symmetrical cortical necrosis of the kidneys, which is combined with glycolic nephrosis, and in the case of pregnancy pathology - with acute hemoglobinuria, apoplexy and necrosis of the adrenal cortex, necrosis of the anterior lobe of the pituitary gland. Histologically, coagulation necrosis of the cortical vein of the kidney is noted.

Fibrin thrombi are found in glomerular capillaries and arterioles.

The zone of necrosis is limited by a demarcation line of polymorphonuclear leukocytes. In the future, necrotic areas are subject to organization, fibrosis, and calcinosis.

The nature of kidney damage in case of poisoning by some chemical substances, medicinal products of plant and animal origin largely depends on the properties of poisons and their metabolites.

The nephrotoxic effect of poisons of the thiol group (compounds of heavy metals, etc.) is manifested by a characteristic coagulation necrosis of the epithelium of the renal tubules. A classic example of necronephrosis is the so-called sulem kidney. With glycol nephrosis, the kidney is enlarged, on wet section, balloon dystrophy of the tubular epithelium of the proximal and distal parts of the nephron with oxalate crystals in their lumen and inside the cells is histologically determined. Nephrocytes in the state of balloon dystrophy may not be rejected for a long time, slowing down the regeneration of kidney tissue.

Damage to the kidneys in case of poisoning by chlorinated hydrocarbons has some features. In case of dichloroethane poisoning, fatty dystrophy of the nephrocytes of the renal tubules of the proximal and distal parts of the nephron is noted.

Acute hemoglobinuric nephrosis develops in case of poisoning with hemolytic poisons (acetic acid, arsenic hydrogen, copper sulphate, amino and nitro compounds). At the same time, morphological changes reflect the transport of hemoglobin through the nephron system.

**Clinical picture.** In the course of acute renal failure, 4 periods are distinguished: the initial effect of the etiological factor, oliguria or anuria, restoration of diuresis, recovery. In the period of initial action, etiological factors are observed in the manifestation caused by a specific cause of the disease, for example, with an incomplete infected abortion, anaerobic sepsis, bacteremic shock, intravascular hemolysis and acute renal failure develop.

General signs of the disease: chills, increase in body temperature, decrease in blood pressure, pallor, cyanosis of the skin in combination with rapidly increasing jaundice, urine acquires a dark color, protein, erythrocytes, leukocytes, cylinders, blood pigment and detritus appear in it.

Regardless of the cause of acute renal failure, changes in hemodynamics always prevail during this period, sometimes with a significant drop in blood pressure, only after recovery from shock (collapse) do signs of kidney dysfunction begin to prevail.

The period of anuria or oliguria is characterized by a decrease in diuresis (500 ml / day) and a violation of kidney functions - in the blood, the level of the products of protein metabolism, non-volatile K-t increases, the water-electrolyte balance changes.

Patients complain of weakness, loss of appetite, drowsiness, listlessness. Nausea increases, vomiting appears. As the level of urea and urinary creatinine increases, the concentration of sulfates and phosphates increases, hyperkalemia is observed, and the concentration of sodium, chlorine, and calcium in the plasma decreases. The more pronounced these changes are, the more pronounced are the signs of uremia. Signs of damage to the nervous system appear: anisocoria, nystagmus, decreased corneal, tendon, and periosteal reflexes, anisoreflexia, pathological reflexes, adynamia, decreased memory, sometimes excitement, convulsions, paralysis. Drowsiness can change to a comatose state.

During this period, mental disorders may occur, which are mainly characterized by exogenous reactions, more often in the form of various delirious states. Delirium usually has a fantastic content. Ament syndrome, stupor, the development of which indicates acute renal failure, are less common. There may be epileptic seizures up to status epilepticus.

Violations of the function of the urogenital system and hemodynamics are different, but almost always there is tachycardia, expansion of the borders of the heart to the left, and a systolic murmur at the top of the heart. High blood pressure is a non-permanent symptom, but significantly affects during this period. It can cause pain in the heart, heart failure, and an attack of eclampsia. Sometimes pericarditis occurs, while the patient notes pain in the region of the heart, there is a noise, friction of the pericardium, as the effusion in its cavity increases, the noise decreases, but the symptom of heart failure increases, the borders of the heart expand, and the RO pattern characteristic of pericarditis develops. Hyperkalemia, which disrupts the electrolytic activity of the myocardium, is especially dangerous in acute renal failure.

Depending on the degree of hyperkalemia, the electrocardiogram reveals varying degrees of pronounced slowing of atrioventricular and intraventricular



conduction, increased amplitude and a sharper shape of the T wave, and a decrease in the potential of the R wave.

Rapid breathing is often associated with acidosis, with severe acidosis (acidemia) breathing becomes noisy. Sometimes pulmonary edema occurs against the background of which pneumonia often occurs. The main causes of pulmonary edema are hyperhydration, left ventricular failure, decreased blood oncotic pressure, and increased capillary permeability. Constant acute renal failure is observed with anemia as a result of inhibition of hematopoiesis in the bone marrow, blood loss and hemolysis. Damage to the intestinal tract, in addition to nausea and vomiting, is manifested by abdominal pain, diarrhea. Due to increased bleeding, blood is often found in vomitus and feces. Sometimes serous peritonitis occurs. The period of oliguria or anuria lasts an average of 2 weeks, but there are known cases of longer oliguria (5-6 weeks).

The period of restoration of diuresis has 2 phases of initial diuresis when about 500 ml of urine is released per day, and polyuria - diuresis exceeds 1800 ml. Symptoms in the phase of initial diuresis. Acute renal failure does not change significantly or continues to grow. This phase lasts only 2-3 days and is replaced by polyuria. The phase of polyuria sometimes develops violently, the amount of urine can reach several liters, as a result of which dehydration occurs, the patient loses weight, the skin becomes dry, the tongue is scaly, the tongue is dry, there is thirst, weakness, pain in the heart.

Extrasystole and pain in the heart are most often signs of hypokalemia, which on the electrocardiogram is manifested by a decrease in the ST segment, a decrease and inversion of the T wave, and the appearance of a U wave. Polyuria is accompanied by a constant decrease in azotemia, the content of creatinine and urea in the plasma, and the normalization of its electrolyte composition. The period of recovery of diuresis lasts an average of 20 days, but it can be longer.

The normalization of creatinine and urea in the blood is considered the beginning of recovery. This period, during which the renal blood circulation, glomerular filtration and concentration capacity of the kidney is restored, lasts from 6 months. up to 2 years.

The most frequent complication of acute renal failure in the period of anuria is pulmonary edema, as well as pyelonephritis, which can occur at any period of the disease.

**Diagnosis.** It is based on the clinical picture, observations of diuresis and the data of laboratory studies (acid-alkaline composition), plasma electrolytes, biochemical changes in nitrogen metabolism). X-ray examination is carried out to diagnose disorders of the heart and lungs. If obstruction of the upper urinary tract is suspected, retrograde ureteropyelography is indicated.

**Treatment.** The nature of medical measures is determined by the cause of acute renal failure. In case of impaired blood circulation, shock (collapse), anti-shock measures are carried out, in case of acute poisoning, along with anti-shock therapy, measures to remove poison from the body: gastric lavage, infusion therapy, forced diuresis, hemodialysis, hemosorption, hemofiltration, peritoneal dialysis, etc.

With massive hemolysis, exchange blood transfusion, plasmaphoresis with replacement of albumin solution or plasma is indicated. If the cause of acute renal failure is bacteremic shock, in addition to anti-shock measures, antibacterial therapy is prescribed.

At the beginning of the period of oliguria or anuria, diuresis is stimulated by intravenous administration of furosemide 160 mg x 4 times a day (up to 1000 mg per day).

If diuresis increases at the same time, the use of furosemide is continued. Further therapy, for example, to regulate homeostasis.

Prescribe a high-calorie diet with an increase in the content of carbohydrates and fats (with a restriction of protein and potassium). The volume of injected fluid should exceed diuresis and the amount of water lost by the patient with vomiting and bowel movements, no more than 500 ml, while 400 ml of 20% glucose solution with 20 units of insulin must be injected.

With hyperkalemia, in addition to glucose, 10-20 ml of 10% calcium gluconate solution and 200 ml of 5% sodium bicarbonate solution are prescribed intravenously. Anabolic steroids are also used. Tranquilizers and antipsychotics are indicated for developing mental disorders.

If oliguria continues and symptoms of uremia increase, the patient should be transferred to a hemodialysis unit, where an artificial kidney or peritoneal dialysis can be used. Indications for extrarenal blood purification are an increase in the content of urea in the blood more than 200 mg / per 100 ml (33.3 mmol / l), potassium in the plasma more than 6.5 mmol / l, acidemia with a deficiency of bases

10mmol/l, hyperhydration with clinical and R-logic manifestations of pulmonary edema, symptoms of acute uremia.

**Forecast.** Acute renal failure is largely determined by the cause and severity of structural changes in the kidneys. The post-traumatic and postoperative period with acute renal failure has a more difficult prognosis (mortality of about 50%) than acute renal failure due to other causes (mortality of 10%).

After suffering from acute kidney failure, in most cases there is a full recovery. Only some patients retain altered kidney function, which leads to arterial hypertension, sometimes chronic pyelonephritis or nephrocalcinosis develops. After a large ischemic heart attack, symmetrical cortical necrosis of the kidney is possible, leading to chronic renal failure.

### **Chronic kidney failure**

It occurs most often in young and middle-aged people.

**Etiology.** Chronic renal failure is the result of many long-term (from 2 to 10 years) diseases of the kidneys and urinary tract with a gradual decrease in the functional capacity of the kidneys. They include chronic glomerulonephritis, chronic pyelonephritis, interstitial nephritis, systemic diseases accompanied by kidney damage, diabetic glomerulosclerosis, kidney stone disease, kidney tuberculosis, hydronephrosis, atony and obstruction of the urinary tract, cystic dysplasia, hypoplasia, congenital tubulopathies, amyloids, etc.

**Pathogenesis.** Chronic renal failure is characterized by progressive damage to most or all nephrons, often replaced by connective tissue. According to the theory of intact nephrons, proposed in 1969. Bricker, with chronic renal failure, part of the nephrons dies, while the part remains intact, hypertrophies and homeostasis is maintained for a long time, and the preserved nephrons function like the nephrons of a healthy kidney. However, research by M.Ya. Ratner, V.V. Serova, and N.A. Tomilina in 1977. revealed the heterogeneity and variability of the affected nephrons and what processes they undergo during the development of chronic renal failure.

**Pathological anatomy.** Morphological changes of the kidneys depend on the nature of the disease leading to the development of chronic renal failure. This is mainly a decrease in the mass of functioning nephrons as a result of their atrophy and sclerosis of the parenchyma of the organ.

Pathological changes in uremia are various uremic encephalopathy is expressed by neuron dystrophy, hemorrhages and edema. With long-term and severe uremia, the inheritance of bodies is demyelination of nerves due to the action of toxins.

In uremic cardiomyopathy, hypertrophy and dystrophy of the myocardium, dilatation of the heart cavities, atherosclerosis of coronary vessels, subendocardial

hemorrhages, pericarditis, signs of chronic pneumonia and pulmonary edema are found.

In chronic renal failure, chronic pharyngitis and esophagitis with micro and macrohemorrhages are observed. Hemorrhages and erosions are found in the stomach and intestines along with edema of the mucous membrane and submucosal base. Dystrophy, hemoptysis, hemorrhages also develop in the liver, which is often enlarged. Chronic uremia is characterized by hyperplasia of the parathyroid glands and osteodystrophy.

**Clinical picture.** The most common clinical classifications of chronic renal failure are built taking into account the severity of its course and the data of laboratory studies, E. M. Tareev in 1973 proposed to distinguish 2 stages - conservative and terminal - in the clinical course of chronic renal failure caused by diseases of a non-surgical profile. In urological diseases characterized by intermittent flow, the most recognized classification is Lopatkin N. A., 1972. according to which 4 stages of chronic renal failure are distinguished: latent, compensated, intermittent, terminal. Chronic renal failure develops gradually, which is associated with a slow increase in the content of creatinine, urea, guanidine derivatives, sulfates, phosphates, and other metabolites in the blood.

In the conservative stage, when diuresis is preserved (polyuria is often observed), the level of sodium, chlorine, magnesium and potassium in the plasma does not increase. Persistent hypocalcemia is associated with impaired vitamin D3 metabolism and absorption of calcium in the intestines. Polyuria can lead to hypokalemia. Anatomical acidosis is often observed. A set of humoral disorders determines the symptoms of chronic uremia.

Patients complain of rapid fatigue, reduced work capacity, headache, decreased appetite, unpleasant taste in the mouth. Nausea and vomiting appear. Pallor

of the skin, dryness, flabbiness is noted. Muscles lose their tone, their small twitches appear, tremors of the fingers of the hands. Sometimes there are pains in the bones and joints. Anemia develops, leukocytosis increases. Increased bleeding. Arterial hypertension was often observed, leading to heart disorders. The boundaries of the heart are expanded, its tones are muffled, changes are noted on the electrocardiogram (sometimes they are associated with dyskalemia). In the early stages of CKD, asthenia develops with a predominance of irritability, autonomic disorders, and hypochondriacal complaints.

The conservative stage can last several years. The condition allows the patient to work, but increased physical exertion, mental stress, errors in diet, restriction of drinking, infectious diseases, operations can lead to deterioration of kidney function and increase in uremic symptoms.

When glomerular filtration drops below 10 ml/min, conservative correction of homeostasis is not possible. The terminal stage comes: oliguria gradually develops, azotemia, acidosis of hyperhidrosis increases, hyponatremia, hypochloremia, hypermagnesemia, hyperkalemia are possible. The terminal stage is characterized by emotional lability, inhibition, inadequacy of behavior, mental disorders, the nature of which depends on the degree of uremia.

Asthenia occurs in the early stages. Its feature is the predominance of sudden fatigue, hyperesthesia, sleep rhythm disturbances (insomnia at night, drowsiness during the day). But as the severity of the condition increases, an adenoma appears, asthenia in some cases is replaced by stupor of varying degrees, up to coma, in others psychosis may develop. Hypnagogic delirium occurs most often. Its feature is mundane content, hallucinations, contemplativeness, lack of motor excitement. Amnesia and dusky obscuration of consciousness develop less often. The development of psychoses and the terminal stage of chronic renal failure always indicates an unfavorable prognosis of the underlying disease. Somatic changes are

diverse, the face is puffy, gray-yellow in color. There is itching of the skin on the skin of the comb, the hair is dull and brittle. Dystrophy is increasing, hypothermia is characteristic, the voice is hoarse. An ammonia smell is felt from the mouth. Aphthous stomatitis occurs, sometimes Paraty, the tongue is coated, the stomach is distended, frequent vomiting, retching, often diarrhea, foul-smelling, dark-colored stools. Anemia and hemorrhage are increasing.

Muscle twitches become frequent and painful. With the long-term development of uremia, pains appear in the bones and joints, the fragility of the bones is noted. Loud breathing is often due to acidosis, pulmonary edema, pneumonia. Uremia is complicated by pericarditis, pleurisy, ascites, pseudoperitonitis, encephalopathy and uremic coma develop.

The terminal stage lasts from several weeks to several months.

**Diagnosis.** It is established on the basis of the history of chronic kidney disease, characteristic symptoms - uremia, azotemia and other biochemical disorders.

**Treatment.** In the conservative stage, medical measures are aimed mainly at restoring homeostasis, reducing azotemia, and combating manifestations of uremia. When glomerular filtration falls below 50 ml/min and creatinine rises above 2 mg/100 ml (177  $\mu$ mol/l), it is advisable to reduce the amount of protein entering the body to 30-40 g per day, and with glomerular filtration below 20 ml/min, a low-protein diet is prescribed (protein content 20-24 g) per day. The diet must be high-calorie (2-3000 kcal) and contain non-replaceable amino acids (potato-egg diet without meat and fish). Food is prepared with a limited amount of salt up to 2-3 g, and for patients with high hypertension - without salt. Calcium gluconate and Vit. D in large doses at up to 100,000 IU / day. However, it should be remembered that the use of vit. D in large doses with hyperphosphatemia can lead to calcification inside the organs, therefore, to

reduce the level of phosphorus, Almagel should be taken 1-2 hours per spoonful 4 times a day. The level of calcium and phosphorus in the blood is regularly monitored.

Depending on the degree of acidosis, 100-200 ml of 5% sodium bicarbonate solution is administered intravenously. When diuresis is reduced, furosemide (Lasix) is indicated in doses that cause polyuria. In order to reduce blood pressure, conventional hypotensive agents are used in combination with furosemide.

The treatment of anemia is complex: prescribe 5% solution of testosterone propionate 1 ml IV daily or other anabolic steroids, iron preparations, blood transfusions, Vitamin B12, folic acid, antibiotics and chemotherapeutic drugs are used cautiously. Only erythromycin is prescribed in usual doses, the doses of penicillin, ampicillin, methicillin, ceporin (cephaloridine) and sulfonamides are reduced by 2-3 times.

The use of streptomycin, monomycin, neomycin, polymyxin even in reduced doses in chronic renal failure can cause neuritis of the auditory nerve, etc. Complication. Nitrofurantoin derivatives are contraindicated.

With the development of pulmonary edema as a result of hyperhydration, you can prescribe sorbitol (50 g in a solution internally), it causes diarrhea and the release of a significant amount of fluid. Glycosides for heart failure in chronic renal failure are used in reduced doses, especially in hypokalemia. In case of pericarditis, small doses of prednisolone are prescribed or hemodialysis is performed, which is more effective, in severe cases surgical treatment is required. Tranquilizers can be used as symptomatic agents, in case of neuroleptic psychoses. Effective neotropic drugs. Hemodialysis is indicated for exacerbation of chronic renal failure. After the improvement of the patient's condition, it is possible to restore and for a long time carry out the usual conservative therapy.



In the terminal stage, when conservative treatment and surgical intervention on the kidneys and urinary tract are ineffective, the patient is transferred to regular treatment (2-3 times a week) with hemodialysis, peritoneal dialysis. Indications for extrarenal blood purification in chronic renal failure usually occur in cases where creatinine clearance falls below 1 mg/min, and its plasma level exceeds 10 mg/100 ml (884  $\mu\text{mol/L}$ ).

**Forecast.** The use of hemodialysis and kidney transplantation improved the results of treatment of patients with chronic renal failure, allowed them to prolong their lives and achieve rehabilitation for many years. Thanks to hemodialysis, by 1981, the lives of 150,000 patients with chronic kidney failure were saved in the world. With the timely application of new operative methods (hemodialysis and kidney transplantation) in the late stages of chronic renal failure, mental disorders are reduced, but asthenic symptoms and psycho-organic syndrome phenomena persist. In unfavorable cases, with long-term use of hemodialysis, the so-called dialysis uremic dementia with lethargy and apathy may develop. Long-term stay of the patient in a state of uremia, pronounced dystrophy, encephalopathy worsen the results of hemodialysis treatment and prevent kidney transplantation.

**Kidney failure in children.** It occurs as in adults, in acute and chronic form. The main etiological factors, pathogenesis and pathological anatomy, clinical picture are similar to those in adults. Treatment is carried out with the same means in terms of kg of mass. The prognosis of acute kidney failure in children is very serious, especially in the first two days of life. This is due to relatively low tolerance to acute renal failure and imperfect children. Complex treatment of exacerbation of chronic renal failure is carried out with the use of large doses of diuretics or temporary use of hemodialysis, which often allows to restore kidney function and transfer the process to a compensated stage. According to special indications, a kidney transplant is performed.

### ***MAG3 renography***

<sup>99m</sup>Tc-MAG3 (mercaptoacetyltriglycine) has become the agent of choice for dynamic radionuclide imaging of the renal tract in most centers. It was first developed as an alternative to hippuran, but has a plasma clearance 50–65% slower than that of hippuran. Nevertheless, it gives images comparable to <sup>123</sup>I-hippuran. Following intravenous injection, it remains loosely bound to serum proteins, and only a small proportion undergoes glomerular filtration. Clearance is predominantly by tubular secretion. The 30-minute excretion of <sup>99m</sup>Tc-MAG3 is approximately 70%, and by 3 hours 90% of the tracer is cleared by the kidneys. Renography can be combined with administration of a diuretic (usually furosemide) to produce a high urine flow diuresis renogram. The <sup>99m</sup>Tc-MAG3 is an adequate tool for the assessment of urinary uptake, transit, excretion, and split renal function. In addition, simple conversion methods will allow reproducible estimations of ERPF from the <sup>99m</sup>Tc-MAG3 activity curve.

#### ***Indications***

- assessment of whole or relative kidney function
- before and after surgical intervention (e.g., pyeloplasty, partial/total nephrectomy)
- investigation of acute or chronic renal failure
- assessment of the transplanted kidney
- assessment of renal function following trauma
- assessment of kidney drainage in obstructive uropathy (e.g., uretero-pelvic junction obstruction, renal stones)
- assessment of congenital renal abnormalities (e.g., duplex, horseshoe, absent, ectopic, or cystic kidneys)
- identify vesico-ureteric reflux.

### ***Patient preparation***

- adequate hydration (500 mL of oral fluid 15–30 min before examination) is vital to ensure good diuresis (urine flow of 1–3 mL/min). Avoid study if patient appears clinically dehydrated

### ***Procedure***

The patient may be placed supine or erect, reclining against the camera. Assess need for catheterization. If not, patient must void before study.

Position patient in either supine or sitting up position. The posture of the patient may have an effect on the renography curve .

A typical adult dose of 50–120 MBq is carefully injected intravenously to avoid local extravasation. An image is taken every 10–20 seconds for up to 40 minutes following administration of radiopharmaceutical.

Analog images are taken every 5 minutes and the hard copy must include several serial analog images as well as the renogram curve. The first 12 and last 12 frames may be summed to exhibit the kidneys more clearly. If the kidney fails to empty by 20 minutes, frusemide may be administered (F+20). Data is collected for 30–45 minutes (or 20 min following diuretic injection). The patient is asked to void at the end of the procedure (minimizes radiation to the bladder as well as allowing assessment of urine production rate). The renogram curve demonstrates change in kidney activity over time.

The radiation activity detected by the gamma camera is first stored as a computer image. Regions of interest (ROI) are mapped out for the kidneys and bladder, in addition to a background region to enable precise measurement of activity count for each time frame. The background region is usually chosen just lateral to the kidneys, but care must be taken to avoid the liver, due to its high tracer uptake.

Additional ROIs (different moieties of a duplex kidney) may also be delineated.

The renogram curve can be obtained following subtraction of the background count from the kidney and bladder ROI count and is displayed as a percentage of the injected dose (y-axis) against time (x-axis). The relative function of each kidney is calculated by comparing the percentage dose at 2 and 3 minutes' uptake. Following the procedure, the patient is informed about the possibility of a prolonged diuresis. Frequent voids will help reduce bladder irradiation.

### *Diuresis*

If diuresis renography is indicated, an intravenous bolus of frusemide (dose 1 mg/kg in infants, 0.5 mg/kg in children aged 1–16 years, and 40 mg in adults) is commonly used. Ensure there are no contraindications to diuretic therapy. Frusemide will produce a maximal diuretic response within 5–10 minutes and rationale is to increase the sensitivity of the dynamic renal study by increasing urine flow rates to stress the system, such that minor degrees of obstruction are unmarked. The timing of diuretic administration is a matter of local policy but the various techniques have distinct advantages. The traditional technique (F+20) involves frusemide administration 20 minutes after injection of the radiopharmaceutical. The study must continue for at least 20 minutes following frusemide injection. This enables study of initial unmodified renal handling, followed by the response to increased urine flow rate.

The F-15 technique (frusemide given 15 min before radiopharmaceutical) ensures maximal diuresis at commencement of data acquisition, thereby revealing minor levels of obstruction. Administration of the tracer simultaneously with frusemide (F+0 technique) has been practiced in pediatric units and has the advantage of significantly reducing examination times. The F+0 technique is not recommended in patients with significant renal failure (GFR < 15 mL/min per kidney) and renal units with significant hydronephrosis.

The timing of diuretic does not significantly alter split renal function result, but centers should standardize practice to enable meaningful comparisons (e.g., before and after surgical intervention). The F-15 technique will separate the majority of equivocal curves on F+20 renography in to either unobstructed or obstructed, and therefore is preferred in patients with equivocal results or with gross hydronephrosis.

### *Factors influencing MAG3 renography*

**1. Renal function:** a GFR of <15 mL/min per single kidney will result in urine flow rates of <10 mL/min, with poor subsequent tracer washout. This may result in an “obstructed” (false positive) curve. Frusemide is usually insufficient to increase diuresis significantly and perfusion pressure-flow studies (Whitaker test) ought to be considered. Renal disease affecting the parenchyma (e.g., acute tubular necrosis) may diminish the response to diuretics.

**2. Hydration:** minor levels of obstruction may be masked in dehydrated individuals. In addition, diuretic administration may be perilous if the patient is already dehydrated. Oral hydration (500 mL of water 30 min before study) will usually suffice although on occasions intravenous fluids may be required.

**3. Collecting system capacity:** in the massively dilated system, urine flow may be inadequate to prevent tracer accumulation in the renal pelvis. In such cases, a false-positive “obstructed” curve may be the end result. The F-15 technique will help minimize the effects of a capacious system.

**4. Collecting system compliance:** increased diuresis, within a normo-compliant system, should result in distension of the renal pelvis with no significant increase in pressure. However, poor compliance may cause rapid elevations within a non-dilated system, such that any obstruction is overcome and there is reasonable tracer flow distal to the obstruction (false negative). Conversely, a hyper-compliant upper tract

will result in renal pelvic tracer accumulation, in spite of the absence of obstruction, resulting in a false-positive curve.

**5. Bladder effects:** a full bladder may inhibit drainage from the ureters and cause artifacts. The patient must be asked to void prior to commencement and again before completion of data acquisition. Alternatively, in patients with chronic retention or a neurogenic bladder, catheterization should abolish any effects of a full bladder.

**6. Ureteric dilatation or obstruction:** in cases of gross ureteric dilatation, an ROI drawn around the kidney and renal pelvis may miss the distal obstruction, resulting in a false-negative study. Care must be taken to study the analog images and ROI must include the ureter proximal to the obstruction. Furthermore, multiple simultaneous levels of obstruction will not be apparent by MAG3 renography.

The maximal recommended activity per test is 100 MBq for MAG3 renography, which corresponds to an effective radiation dose of 1 mSv (equivalent to 6 months of background radiation).

### *Interpretation*

#### *Normal renogram curve*

The shape of the renogram curve (following subtraction of background activity) is dependent on—

1. MAG3 uptake from blood into kidney
2. MAG3 elimination from kidney into bladder

Classically, the normal MAG3 renogram curve has **three phases:**

- ***The first phase:*** steep upward rise following intravenous contrast injection; this is indicative of the speed of tracer injection and its delivery to the kidneys (i.e., renal vascular supply).

- ***The second phase:*** a more gradual slope which represents renal handling of MAG3 (renal uptake by tubular secretion and glomerular filtration) and peaks between 2 and 5 minutes. Time taken for the curve to peak following tracer injection is referred to as T<sub>max</sub>. This may be delayed in patients with renovascular insufficiency, renal failure, and obstruction
- ***The third phase:*** commences after the peak. Associated with the emergence of tracer in the bladder. Represents elimination (but also delivery) of tracer from the kidney. After 3 minutes both elimination and uptake are in competition, but the former subsequently dominates. It is this elimination curve that is dependent on the upper tract urodynamics. The elimination curve may have a smooth or stepwise (variant of normal) pattern and when normal, excludes the presence of obstruction.

A delayed upward deflection may indicate intermittent obstruction or vesico -ureteric reflux.

### ***Split renal function***

This is expressed as the ratio of the area under the renogram curves of the two kidneys obtained during the period 40 seconds to 2 minutes 40 seconds after tracer injection. The shortest transit time for filtrate in the Bowman's capsule to the renal pelvis is 2.5 minutes, and therefore it can be safely assumed that the MAG3 will not be found in the collecting system within 2.5 minutes of injection. The initial 40 seconds are excluded to prevent artifact errors. The relative function in a pair of normally working kidneys may vary between 40% and 60%. Similarly, relative functions in different moieties of a duplex kidney can also be calculated.

### ***Scarring***

Since 80% of MAG3 is metabolized by tubular secretion, the analog images can be analyzed for the presence of parenchymal scarring. Although DMSA renography

remains the gold standard for the investigation of scarring, MAG3 studies show good correlation between the two techniques.

***Renogram curve patterns:*** When interpreting MAG3 renography, five distinct patterns (based on the F+20 technique) are recognized. It is important not merely to assess the shape of the curve, but also to examine the sequential analog images to determine the level of obstruction, as the calyces, pelvis, and ureter may all be easily visible

***Type I—normal response***

This is characterized by a rapid uptake curve leading up to a peak within 2–5 minutes, followed by gradual (but sometimes stepwise) elimination of tracer. Administration of furosemide results in no appreciable difference in speed of elimination. A normal curve virtually excludes obstruction, although it may be argued that increasing urine flow rate (i.e., using the F-15 technique) may expose lesser degrees of impedance.

***Type II—obstructive response (high-pressure system)***

In the absence of any other factors affecting drainage (e.g., dehydration, renal impairment, etc.), an obstructive pattern is denoted by a rising curve. In addition, the lack of an exponential tracer elimination curve is also suggestive of a degree of obstruction. Typically, there is little or no response to furosemide. On the analog images, the affected kidney will often display good parenchymal uptake and accumulation of tracer above the level of obstruction (e.g., in the renal pelvis in patients with UPJO). The diagnosis of obstruction cannot be satisfactorily made (even in the presence of a rising curve) if the affected kidney has a GFR of <15 mL/min, since the rate of urine production may be insufficient to produce tracer washout (usually 1–3 mL/min urine production is required).



***Type IIIa—dilated but not obstructed (low pressure/hypotonic system)***

There is an initial accumulation of tracer in the kidney, resulting in a rising curve similar to an obstructive response, but there is prompt elimination following furosemide injection. The analog images usually demonstrate tracer accrual in a dilated system secondary to stasis rather than obstruction. The increased urine flow produced by the diuretic is adequate to effect free drainage.

***Type IIIb—equivocal response***

Following an initial “obstructed” rising curve, a furosemide injection produces a somewhat languid response. The curve demonstrates some tendency to washout, albeit incompletely. Examination of the analog images may help clarify whether this represents partial obstruction or inadequate tracer elimination (e.g., due to a dilated renal pelvis). In this situation, an F-15 study will help categorize the majority of equivocal curves into either obstructed or non-obstructed.

***Type IV—delayed compensation (Homsy’s sign)***

Described by Yves Homsy in 1988, the characteristic shape is a “double peak” response to diuretic. This pattern is seen in patients with subclinical intermittent obstruction. A repeat F-15 diuresis renography will often reveal an obstructed pattern. The first “peak” is due to an initial rising curve, which then exhibits a good response to furosemide. However, as the diuretic effect increases, the threshold is reached and tracer accumulation causes the curve to either flatten or rise.

***Modifications of the MAG3 renography. Deconvolution analysis***

This is a mathematical manipulation of the renogram to produce a theoretical curve that would be derived if the tracer had been injected in the renal artery (rather than a peripheral vein). This allows calculation of a range of transit times through the renal tubules, including mean parenchymal transit time as well as whole kidney transit time.

Transit times are increased in obstruction and renal failure. Attention to technical detail is paramount, and as yet deconvolution techniques have not gained widespread acceptance.

### ***Captopril-enhanced renography***

This modification is indicated for the investigation of renal artery stenosis. Patients should be instructed to stop any angiotensin converting enzyme (ACE) inhibitor or diuretics for at least 3 days prior to examination. Ensure patient is well hydrated. A baseline study is performed first. Following this, a further study is repeated (on the same day or consecutive days) with 25 mg of Captopril

(ACE inhibitor) given orally 1 hour before tracer injection. Renin converts angiotensinogen to angiotensin I, which in turn is converted (by an ACE) into angiotensin II. Angiotensin II effects efferent arteriole vasoconstriction and thereby maintains GFR.

Patients with renovascular (e.g., renal artery) stenosis have higher levels of angiotensin II. The captopril-enhanced renogram therefore will display reduced function (reduced gradient in the uptake part of the curve) and delayed transit, with a delay in T<sub>max</sub> (time taken for renogram curve to peak) (see figure 4.2). The overall sensitivity of this technique is 80–90% for the detection of renovascular hypertension, and patients with positive results can often be successfully treated.

### ***Advantages of MAG3 renography***

- provides sensitive indices of tubular function and urinary excretion
- virtually no contraindications
- non-nephrotoxic
- no significant risk of allergic reactions

- serial examinations possible (often required)
- side effects are rare (unless frusemide or captopril is used)

### ***Disadvantages***

- exposes patient to radiation
- length of study (can take up to 1 hour)
- prone to artifact errors (e.g., due to renal impairment, posture, bladder effect, etc.)
- limited anatomical information
- equivocal results require a repeat procedure (usually F-15 study)
- inaccurate outlining of ROIs can affect curve dynamics

### ***DMSA renography***

- $^{99m}\text{Tc}$ -DMSA (*dimercaptosuccinic acid*) has a high affinity for the renal cortex
- $^{99m}\text{Tc}$ -DMSA is the preferred radiopharmaceutical for static parenchymal imaging

Provides the most accurate assessment of relative renal function compared to other tracers.

Following tracer injection,  $^{99m}\text{Tc}$ -DMSA is mostly plasma protein bound, and therefore clearance by GFR is minimal. In the kidney, the cells of the proximal convoluted tubules (and the distal tubules to a lesser extent) extract the  $^{99m}\text{Tc}$ -DMSA by tubular secretion allowing slow concentration of radioactivity in the renal cortex. After 3 hours, about 50% of the injected tracer is concentrated in the kidneys, remaining there for up to 24 hours. The majority of the other 50% is excreted unchanged in urine. Increased hepatic accumulation, and subsequent biliary excretion is noted in patients in renal failure. Owing to the slow renal extraction of  $^{99m}\text{Tc}$ -

DMSA, the optimal time for imaging is between 2 and 4 hours after tracer injection.  $^{99m}\text{Tc}$ -DMSA scanning represents functioning tubular mass, yields excellent cortical images, and is an invaluable tool in the assessment of both adults and children.

### ***Indications***

- assessment of relative renal function
- detection of renal scarring with a sensitivity of 96% and specificity of 98% (due to urinary tract infections or reflux nephropathy in children)
- investigation of renal anomalies (e.g., horseshoe, solitary, or ectopic kidneys)
- examination of space occupying renal lesions

### ***Procedure***

The optimal time for DMSA scanning remains an unresolved issue. Many units perform the study in the acute phase (i.e., during or soon after a UTI) in order to determine the extent of parenchymal involvement. Critics of such practice point out that an acute abnormality does not necessarily represent a permanent scar and a repeat scan is often required after 3–6 months to determine longstanding injury. Deferring the DMSA scan for such a period of time may avoid the initial examination.

$^{99m}\text{Tc}$ -DMSA has no specific contraindication and no specific patient preparation is required, since uptake is independent of the hydration state

- A typical adult dose of 80–100 MBq (1 MBq/kg body weight) is injected into a peripheral vein.
- Images are acquired after 2–6 hours (usually after 3 h). Imaging must be avoided within the first hour due to the presence of free  $^{99m}\text{Tc}$  in urine.

- Regions of interest are created around both kidneys as well as a background area between the kidneys. Subtraction of the background area count from the overall kidney count will result in the correct kidney count.

To maximize the detection of scarring, various projections should be utilized to image the kidney. Posterior, right, and left posterior views are standard, but anterior views must be included if a pelvic or horseshoe kidney is suspected. Furthermore, in asymmetric kidneys (e.g., ectopic kidneys, scoliosis), anterior views must be obtained and split function expressed as a geometric mean of radioactivity in both posterior and anterior images.

A typical dose of 80 MBq for DMSA renography corresponds to an effective radiation dose of 1 mSv (equivalent to 6 months of background radiation).

### ***Interpretation***

Normal kidneys should have a homogenous parenchymal distribution with visible demarcation between the cortex and medulla. Preservation of cortical thickness is indicative of acute changes, while cortical thinning is in keeping with chronic damage. The size, shape, and location (normal or ectopic) of the kidneys is readily demonstrated. Scars or other deformities are seen as areas of decreased or absent activity within the parenchyma.

Artifacts may arise in kidneys with congenital fetal lobulations or due to splenic overlapping of the left kidney.

### ***Advantages***

- provides excellent cortical images
- accurate split renal function estimation
- non-nephrotoxic

- no significant complications
- allergic reactions are exceptionally rare

### *Disadvantages*

- involves radiation
- does not allow dynamic assessment of renal excretion

### *Obtaining glomerular filtration rate (GFR)*

#### *Indications*

General indications for GFR estimation include—

- any clinical situation requiring an accurate measurement of absolute renal function
- follow-up in patients with chronic renal disease
- prior to administration of nephrotoxic therapy (e.g., chemotherapy)
- GFR estimation is invaluable in the management of patients with or at risk of renal impairment
- GFR is defined as the volume of blood from which a solute is cleared by glomerular filtration through the Bowman's capsule per unit time (mL/min)
- GFR is regarded as a measure of global function

#### *The ideal radioactive tracer for this purpose would have the following properties:*

- be cleared solely, completely, and unmodified by glomerular filtration
- should not undergo tubular secretion or resorption (any tubular secretion will increase the resultant GFR value)
- be non-toxic, stable, and not bound to serum proteins
- be readily measured in blood or urine

- should have a constant clearance irrespective of plasma concentration

### ***Patient preparation***

No specific patient preparation is required, but ensure that the patient is well hydrated and empties the bladder prior to injection.

### ***Procedure***

- Doses used are 10 MBq for <sup>99m</sup>Tc DTPA and 3 MBq for <sup>51</sup>Cr

EDTA

- A pre-tracer injection venous blood sample is taken for background activity
- The tracer is injected and the exact time noted
- A heparinized blood sample is taken at 90, 150, 210, 270 minutes following injection and corrected (minus background activity) plasma tracer concentration can be plotted against time. Studies have shown that a minimum of four blood samples are required for accurate results
- Plotting the log of tracer concentration will result in a linear curve. Extrapolation of this line back to time zero will indicate the effective volume of distribution. The GFR is then calculated as the product of the distribution volume and the slope of the linear log curve, using the formula

$$\text{GFR} = V \lambda$$

where  $\lambda$  is the slope and V is injected tracer dose/distribution dose

The *Gates* technique for GFR estimation, though not in common use, involves analysis of tracer activity in the kidneys between the 2- and 3-minute intervals

following tracer injection. While the obvious advantage of this technique is the speed of the test and the absence of blood tests, its accuracy has been doubted.

This technique has therefore fallen out of favor and most centers use a serial venous sampling method.

An alternative to blood sampling is using three urine samples over 3 hours to measure urinary tracer concentration, but difficulties and inaccuracies in specimen collection make this method unattractive.

A typical dose of 10 MBq for  $^{99m}\text{Tc}$  DTPA and 3 MBq for  $^{51}\text{Cr}$  EDTA for GFR studies corresponds to reasonably small effective radiation doses of 0.1 mSv and 0.007 mSv, respectively. It is therefore feasible to perform serial studies safely if clinically indicated.

### ***Interpretation***

Although cumbersome, these single injection filtration markers techniques provide a more accurate GFR reading than that obtained with traditional creatinine based methods. The GFR value obtained may be used uncorrected to evaluate changes in renal function for an individual patient. However, since GFR varies with age, gender, and body mass, it is recommended that a normalized GFR based on the standard body surface area of 1.73 m<sup>2</sup> be used for comparisons.

- Normal values are 130 mL/min/1.73 m<sup>2</sup> (men) and 120 mL/min/ 1.73 m<sup>2</sup> (women) with a variation coefficient of 14–18%
- Normalized GFR for the newborn is almost half that of the adult, with a gradual increase to adult values by the age 2
- GFR declines by roughly 1% per year after age 40



- Other factors affecting the GFR are time of day (10% higher in the afternoon than at midnight); pregnancy (up to 50% higher in the first trimester); high protein meal (gradual rise in GFR within an hour), and exercise (a transient reduction occurs)

### *Advantages*

1) Accurate; 2) no need for 24-hour urine collections; 3) mandatory in clinical trials investigating progressive renal failure

### *Disadvantages*

- invasive repeated blood samples
- involves a small amount of radiation
- lengthy procedure
- artifacts can be caused by inaccurate recording of times, tracer extravasation at injection site, significant edema, or ascites (due to altered body compartment distribution).

**Practical task:** describe renoscintigraphs of a virtual patient

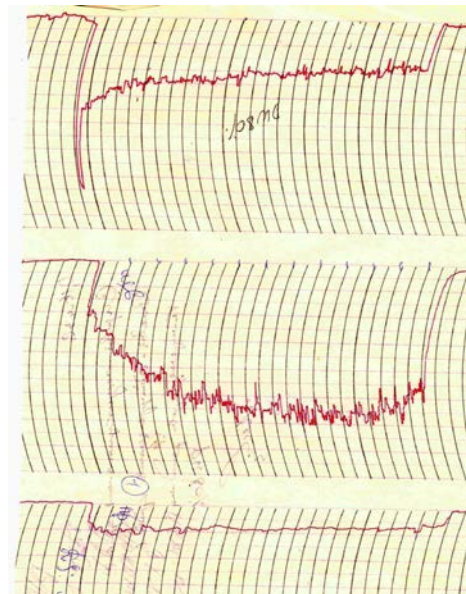


Figure 1. Urinary stone disease. Jpeg

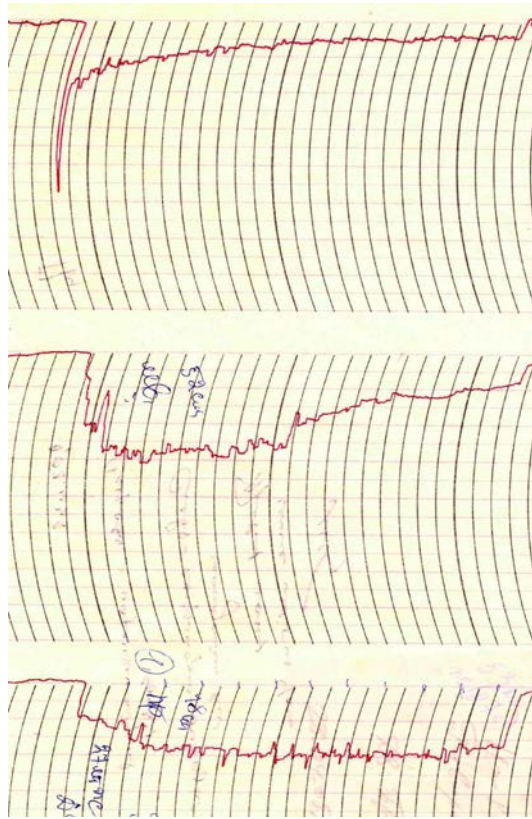


Figure 2. Right-sided nephroptosis. Jpeg

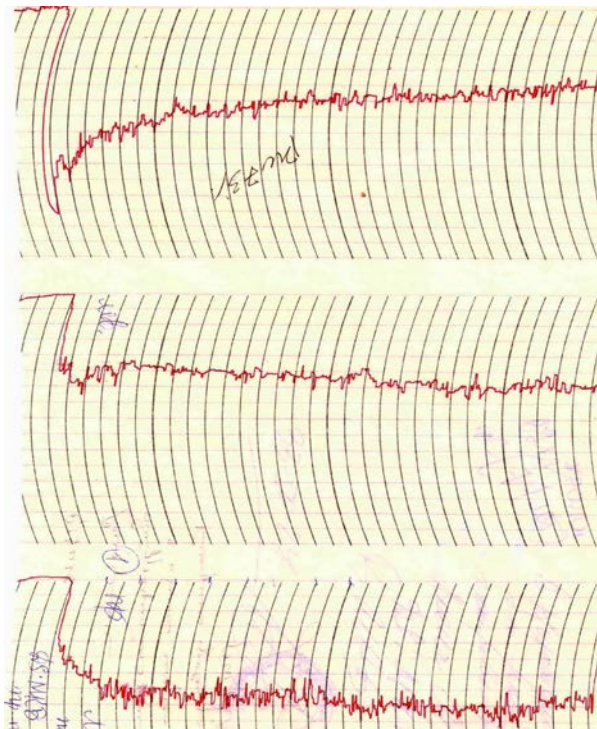


Figure 3. Urinary stone disease. Right kidney stone. Jpeg

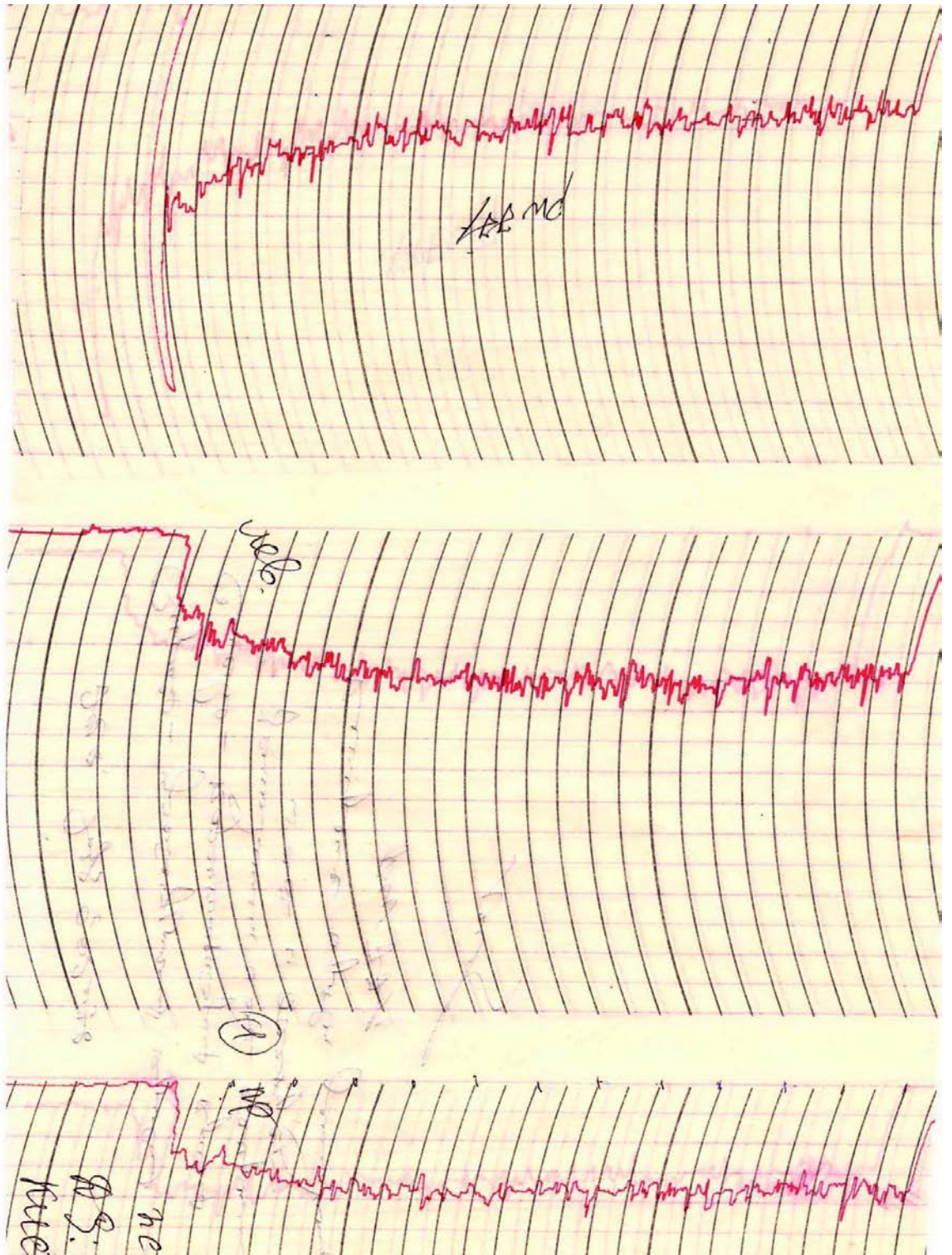


Figure 4. Urinary stone disease. Right kidney cyst. Jpeg

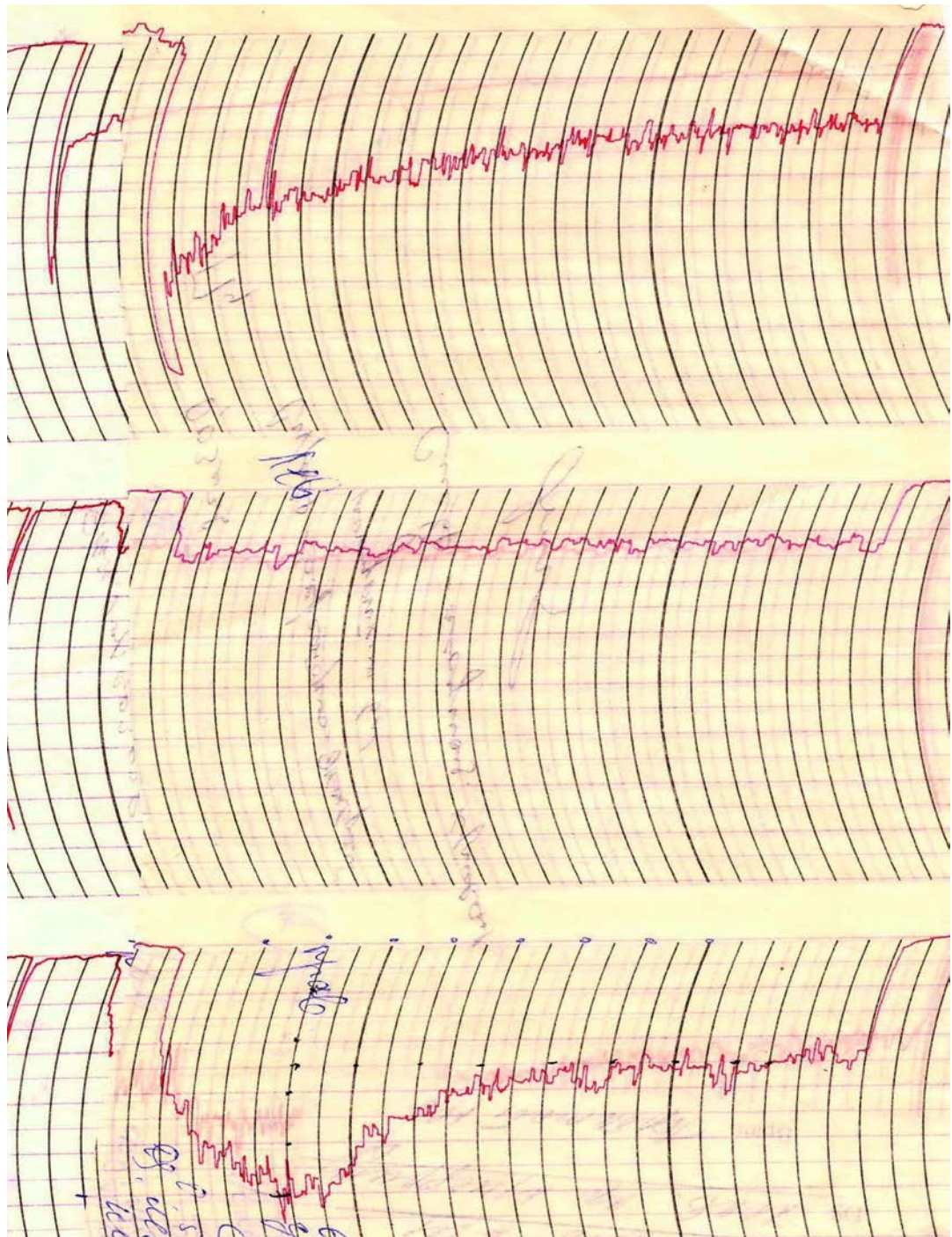


Figure 5. Urinary stone disease. Chronic pyelonephritis. Jpeg

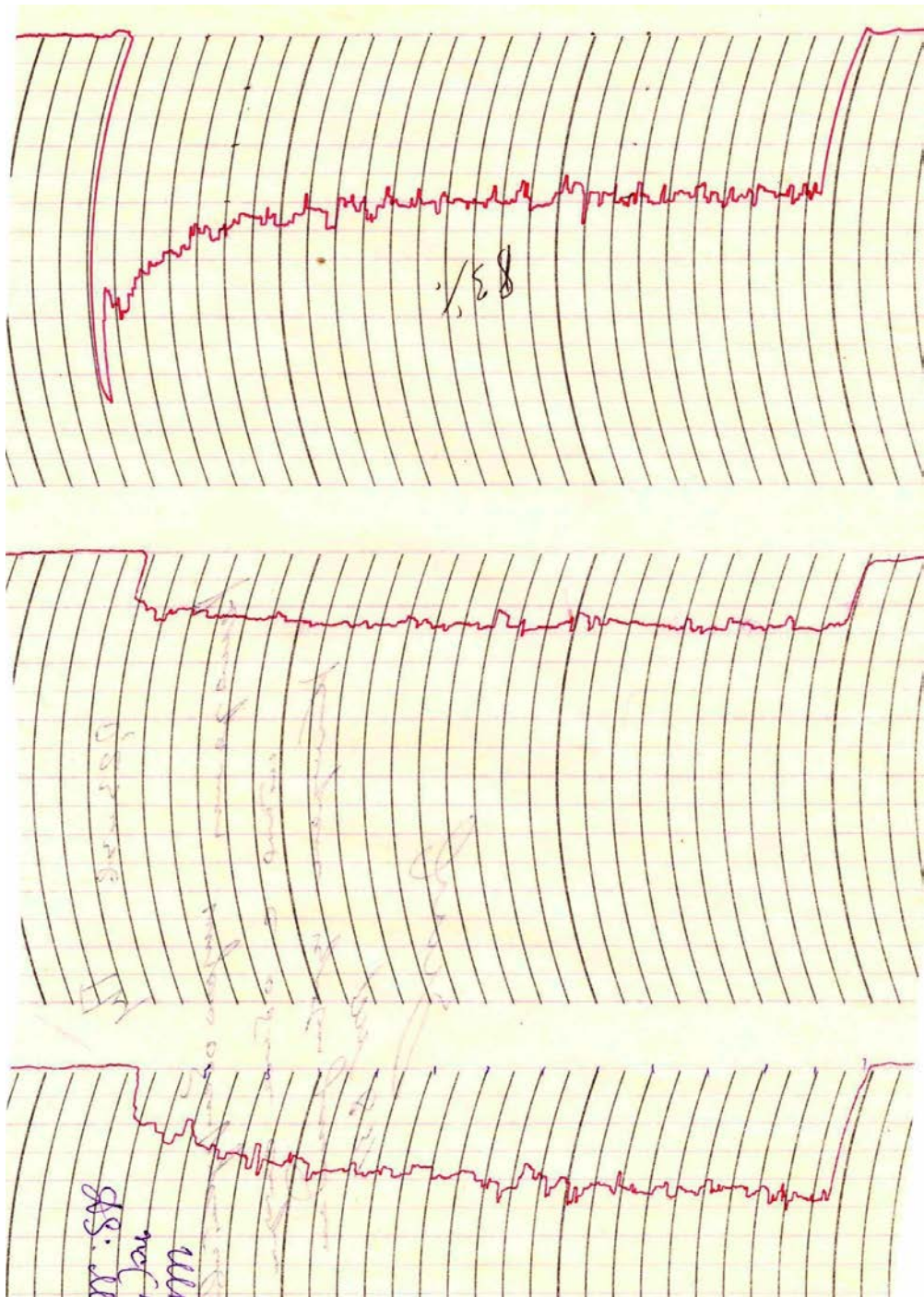


Figure 6. Urinary stone disease. Chronic pyelonephritis. Jpeg

## TEST TASKS

1. What are the causes of prerenal anuria?
  - a) hypertensive syndrome
  - b) bleeding
  - c) inflammatory process of the prostate gland
  
2. When do postrenal anuria occur?
  - a) obstruction of the ureter (ureters)
  - b) with urethritis
  - c) with pyelonephritis
  - d) with glomerulonephritis
  
3. When ureter is obstructed with calculi, should it be used first of all?
  - a) nephrostomy
  - b) ureterotomy
  - c) catheterization of the ureter
  
4. In case of acute renal failure, the stages occur earlier?
  - a) diuretic
  - b) oligoanuric
  - c) primary
  
5. How many stages of development are divided into acute renal failure?
  - a) in three stages
  - b) in four stages

c) in five stages

d) for six stages

6. At the diuretic stage of acute renal failure, do they prevail?

a) leukocyturia

b) erythrocyte

c) potassium

d) bilirubinemia

7. Prognosis from the treatment of acute renal failure?

a) prosperous

b) not prosperous

8. What diseases primarily affect the kidney glomeruli?

a) chronic pyelonephritis

b) chronic glomerulonephritis

c) chronic gastritis

d) chronic epididymitis

9. What diseases contribute primarily to damage to the kidney tubules?

a) chronic cholecystitis

b) mastitis

c) interstitial nephritis

d) cystitis

10. What is more effective to use in chronic renal failure?

- a) antibacterial therapy
- b) bladder catheterization
- c) hemodialysis
- d) kidney transplant

11. How many units of insulin should be administered if:

- a) introduce 5% - 500 ml of glucose - 4 units
- b) introduce 5% - 500 ml of glucose - 8 units
- c) introduce 5% - 500 ml of glucose - 12 units
- d) introduce 5% - 500 ml of glucose - 16 units

12. What is more effective after a kidney transplant?

- a) introduction of 0.9% - 300 ml of sodium chloride
- b) antibacterial therapy
- c) imuran
- d) diet therapy

Correct answers to test tasks on Acute renal failure and Chronic renal failure.

| № Question | Correct answer | № Question | Correct answer |
|------------|----------------|------------|----------------|
| 1          | B              | 7          | a              |
| 2          | a              | 8          | B              |
| 3          | c              | 9          | c              |
| 4          | c              | 10         | c              |
| 5          | B              | 11         | c              |
| 6          | c              | 12         | c              |



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МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ  
ЗАПОРІЗЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ  
КАФЕДРА УРОЛОГІЇ

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**ТУБЕРКУЛЬОЗ СЕЧОВИДІЛЬНИХ ШЛЯХІВ ТА  
ОРГАНІВ ЧОЛОВІЧОЇ СТАТЕВОЇ СИСТЕМИ.ГОСТРА  
ТА ХРОНІЧНА НИРКОВА НЕДОСТАТНІСТЬ**

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