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PYELONEPHRITIS AND LABORATORY DIAGNOSIS OF SOME OTHER KIDNEY DISEASES

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Summary: Pyelonephritis is one of the common diseases of the kidneys and urinary tract, which has a serious prognosis for the development of chronic renal failure, and therefore early diagnosis and adequate therapy are necessary. On the other hand, the danger of overdiagnosis of chronic pyelonephritis lies not only in the inadequacy of the therapy, which is fraught with the development of dangerous side reactions, but also in the fact that once the label of chronic pyelonephritis is applied, it leads the doctor away from the true cause of the disease, delaying the establishment of the correct diagnosis and appropriate treatment.

Keywords: pyelonephritis, laboratory diagnostics, glomerular filtration rate, chronic kidney disease.

Pyelonephritis is an infectious-inflammatory disease of the kidneys with localization of the inflammatory process in the interstitium and upper urinary tract.

Etiology. In 80% of cases in developed countries, the main causative agent of pyelonephritis is E. Coli. Uropathogenic microorganisms also include bacteria of the Enterobacteriaceae family, Enterococcus spp., Staphylococcus saprophyticus, P. aeruginosa. At the same time, microorganisms such as S. epidermidis, Gardnerella vaginalis, diphtheroids, lactobacilli, anaerobes, practically do not cause urinary tract infections, although they also colonize the rectum, vagina and skin covers [1].

In approximately 20% of cases of hospital-acquired pyelonephritis, more than one microorganism is isolated from the urine, especially in patients with an indwelling urinary catheter. During the course of the disease, a change in the causative agent of the infection may be observed; as a rule, multidrug-resistant forms of microorganisms appear, especially with uncontrolled and haphazard use of antibiotics. It should be noted that the patient's own urinary flora upon admission to the hospital is very quickly (within 2-3 days) replaced by nosocomial strains of bacteria, which are characterized by a higher level of antibiotic resistance. Therefore, pyelonephritis that developed in a hospital is characterized by a more serious prognosis and persistent course[2].

Classification

At the moment there is no common point of view on the classification of pyelonephritis.

Classification of chronic pyelonephritis:

1. primary (hematogenous) and secondary (urogenic)
2. non-obstructive and obstructive pyelonephritis
3. exacerbation or remission

4. Latent or recurrent.

Clinical symptoms

Chronic pyelonephritis is a consequence of a poorly treated acute process, although clinically this transition is not always clearly expressed. Particular difficulties in this regard are associated with the long period of latent course of the disease, when there are no symptoms, but the disease slowly progresses and worsens when exposed to provoking factors.

Risk factors for pyelonephritis:

- Infection
- Urodynamic disturbances
- Concomitant pathology (pregnancy, diabetes, non-infectious kidney pathology, immunosuppression) [3].

The latent course of pyelonephritis is characterized by a minimal number of symptoms; sometimes after a cold, during random studies, incoming leukocyturia may be observed. This course is observed in 20% of patients with pyelonenritis, but the disease progresses; after some time, under the influence of certain factors, the infection becomes more active and begins to manifest itself clinically. The main complaints are: dysuria, fever from subfebrile to febrile. Simultaneously with the increase temperatures indicate chills, which are not always adequate to the temperature; in some cases, chills are observed at normal temperatures. Typically, there is pain in the lumbar region. The pathogenesis of pain in pyelonephritis is associated with overstretching of the kidney capsule due to swelling of the renal parenchyma. Urinary stasis cannot be ruled out; in some patients, pain may be localized in the abdominal area. Hematuria may appear, which is associated with damage to the fornical parts of the calyces by the inflammatory process. 80-90% of patients have symptoms of chronic intoxication, such as general weakness, fatigue, and decreased performance. The development of dyspeptic symptoms, loss of appetite, nausea, and sometimes vomiting is extremely typical. It should be noted that these complaints appear before the development of uremia, and therefore cannot be explained by an increase in the level of nitrogenous bases. Some patients suffer from headaches, which can be explained by both arterial hypertension and chronic intoxication [4].

It is necessary to note the dependence of clinical symptoms on the severity of the process. Patients with more significant changes in the pyelocaliceal system have a greater number of complaints. On the contrary, with less pronounced changes in the pyelocaliceal system, clinical symptoms are minimal.

Changes in the cardiovascular system are associated with increased blood pressure. Arterial hypertension is detected in 50-70% of patients. It is important to note that hypertension in pyelonephritis is quite labile and responds well to antihypertensive and anti-inflammatory therapy. As a result, normalization of blood pressure is observed during dispensary observation in 90% of patients [5].

Laboratory and instrumental diagnostics:

Urinary syndrome:

- leukocyturia of varying degrees
- proteinuria (usually does not exceed 1g/day
- bacteriuria (>100,000 bw/ml for E.coli >10,000 bw/ml for pyogenic cocci)

Decreased relative density of urine

Rg examination data (intravenous urography; computed tomography)

Radioisotope studies (renography, scintigraphy)

Biopsy (less important)

Repeated urine tests are critical in making the diagnosis. In this case, moderate proteinuria, leukocyte - and erythrocyturia are detected. The protein content in urine usually does not exceed 1-3 g/l. With latent pyelonephritis, 75% of patients lose less than 1 g of protein per day and only 25% lose from 1 to 3 g. With a recurrent course of the process, proteinuria exceeds 1 g/day in 50% of patients.

It is extremely important to examine the urinary sediment. The most typical is the presence of leukocyturia. However, an increased number of leukocytes in the urine is not always detected, and in this regard, dynamic monitoring of patients is extremely important. Only with repeated studies can one form an impression of the true frequency of leukocyturia in patients with pyelonephritis. Erythrocyturia with a recurrent course of the process is detected in most patients. Approximately 5-10% of patients have gross hematuria [6].

I would like to point out the low information content of the Nechiporenko test, especially since when it is performed, the necessary conditions for urine collection are rarely met. Bacteriological examination is mandatory. Urine cultures should be repeated at least 3 times during observation. In this case, only positive samples are taken into account when more than 100 thousand bacteria grow in 1 ml of urine. X-ray examination plays an important, if not decisive, role in the diagnosis of chronic pyelonephritis. Currently, excretory urography is more often used, but in doubtful cases, the use of retrograde examination is also indicated. Already in the initial stages of the process, a slowdown in the removal of the contrast agent on the affected side is detected. A fairly characteristic and early sign is local spasms of the collecting system. More often they capture the upper calyxes, corresponding to the location of the Disse sphincter [7].

More typical is the development of deformation of the cups and pelvis, which is manifested by filling defects associated with edema and infiltration of the walls. With the long-term existence of the inflammatory process, the spasm is replaced by atony, including the ureters. Gradually, the initial sections of the calyxes become rounded, and the fornixes become deformed. Changes in the renal parenchyma, characterized by edema and infiltration, are manifested by the presence of calyces far removed from each other, usually already atonic. However, as the sclerotic process develops, the calyxes come closer together, the size of the buds decreases, their contours become uneven, and the root layer decreases [8].

Ultrasound and isotope research. Sonograms of the kidneys of patients with chronic pyelonephritis with a latent course and short duration of the disease differ little from sonograms of normal kidneys. With long-term disease and progression of sclerosing processes, heterogeneity of the pyelocaliceal complex (PCC) is noted; the calyxes are dilated and gape, which gives them a “moth-eaten” appearance. In the projection of the maxillofacial area, small echo-positive flares are located that do not give an acoustic shadow, which are foci of scarring. The thickness of the parenchyma remains within normal limits or decreases slightly, while the transverse size of the CLK prevails over the thickness of the parenchyma. As the process progresses, the size of the kidneys decreases, the unevenness of the contours increases, the heterogeneity of the CL increases, the thickness of the parenchymal layer decreases, which is due to sclerosing processes in the kidneys [13].

To characterize the functional state of the kidneys, it is proposed to use the following terms.

Chronic kidney disease (CKD) reflects the presence of kidney damage and/or GFR characteristics.

CKD criteria:

- Kidney damage ≥ 3 months, with decreased GFR or
- GFR < 60 ml/min/1.73 m^2 ≥ 3 months, with or without kidney damage.

Kidney damage is a structural or functional abnormality of the kidneys. They may initially appear when GFR is normal, but may lead to a decline over time. Markers of kidney damage include deviations from the norm in indicators characterizing kidney function: in the results of a biochemical blood test - the concentration of creatinine (Cr), serum potassium; urine analysis - erythrocyturia, leukocyturia, microalbuminuria (MAU), proteinuria; imaging studies - changes in the pyelocaliceal system, kidney cysts, stones, etc. with ultrasound (US), intravenous urography, computed tomography (CT), etc. All persons with kidney damage, regardless of the level of GFR, are considered to have CKD [9].

Thus, those with CKD include:

1. all patients with GFR <60 ml/min/1.73 m^2 for ≥3 months, regardless of the presence of kidney damage;
2. all patients with kidney damage, regardless of GFR.
3. end-stage chronic renal failure (ESRD) GFR < 15 ml/min/1.73 m^2

To assess kidney function, serum CR, GFR, and urinary albumin (Al) excretion are assessed. Assessing Al excretion in urine gives an idea of the state of the glomerular filter and dysfunction of the renal capillary endothelium.

The most accurate indicator reflecting the functional state of the kidneys is GFR. GFR can be measured using endogenous (inulin) and exogenous filtration markers, calculated from the clearance of endogenous filtration markers (Cr), or using formulas based on serum levels of endogenous markers (Cr, cystatin C). Determination of the clearance of endogenous and exogenous filtration markers. The gold standard for measuring GFR is the clearance of inulin, which is present in stable concentrations in plasma, is physiologically inert, is freely filtered in the glomerulus, and is not secreted, reabsorbed, synthesized, or metabolized in the kidneys. Determination of inulin clearance, as well as the clearance of exogenous radioactive tracers (125I-iotalate and 99mTc-DTPA) is expensive and difficult to obtain in routine practice. A number of alternative methods for estimating GFR have been developed. Reberg-Tareev test. Measuring 24-hour CCR (Reberg-Tareev test) requires collecting urine over a certain period of time [10].

Calculation methods for estimating GFR and KKr. Formulas for calculating GFR take into account various influences on CR production, they are easy to use, validated: their values quite accurately coincide with the values of reference methods for estimating GFR. In adults, the Cockroft-Gault formula and the MDRD (Modification of Diet in Renal Disease Study) formula are the most widely used.

Cockcroft-Gault formula (ml/min)

$$GFR = \frac{88 \cdot (140 - \text{age, years}) \cdot \text{body weight, kg}}{72 \cdot \text{Serum CR, µmol/l}}$$

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For women the result is multiplied by 0.85

MDRD formula (ml/min/1.73 m^2)

GFR = 186 • (Serum, mg/dL) -1.154 • (age, years) -0.203

for women the result is multiplied by 0.742

for people of the Negroid race, the result is multiplied by 1.210 [12]

Экскреция белка с мочой

Normal urinary protein excretion in adults is 50 mg/day, Albumin 10 mg/day. Persistent increased protein excretion is usually a marker of kidney damage. In urine one can detect both filterable proteins (α, -, α2-, β2-microglobulins, lysozyme) and those formed in the urinary tract (Tamm-Horsfall protein). Excretion of certain types of protein depends on the type of kidney injury. Increased excretion of Albumin is a sensitive marker of kidney damage in diabetes, glomerular damage and hypertension. In the absence of urinary tract infection and fever, increased urinary Al excretion, as a rule, reflects pathology of the glomerular apparatus of the kidneys [11].

The rate of Al excretion in urine increases significantly in an upright position, after physical activity (PE), with increased protein intake from food, during pregnancy, fever, in patients with urinary tract infection and HF, as well as some other diseases.

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