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DIAGNOSTCAL DIFFICULTIES OF RENAL TUBERCULOSIS

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Annotation: This review presents a systematic approach to diagnosis based on the integration of laboratory (including key urine PCR) and instrumental (primarily contrast-enhanced MSCT) methods. MSCT is considered the most informative method for assessing the extent and specificity of lesions (caverns, calcifications). For differential diagnosis with chronic pyelonephritis, papillary necrosis, spongy kidney, urolithiasis, and neoplasms, a clear algorithm is proposed based on a comprehensive analysis of the anamnesis, the results of three urine cultures, PCR testing, tuberculin diagnostics , and imaging data.
Key words: renal tuberculosis, nephrotuberculosis , diagnosis, differential diagnosis, urine PCR.

Introduction: The relevance of the problem of nephrotuberculosis . Renal tuberculosis (nephrotuberculosis) is an extrathoracic form of a specific infectious process caused by Mycobacterium tuberculosis (M. tuberculosis and other pathogenic species), in which the renal parenchyma is primarily affected, with possible involvement of the renal pelvis-calyceal system (RPS), ureters, and bladder . Despite overall successes in the fight against tuberculosis, nephrotuberculosis remains a serious clinical problem. It is the second most common extrathoracic localization after pulmonary tuberculosis and accounts for 30–40% of all cases of extrathoracic tuberculosis in urological practice . Difficulties in timely verification of the diagnosis are due to the vague, non-specific clinical picture in the early stages, a long latent period (sometimes up to 10–15 years after the primary infection) and the absence of pathognomonic symptoms, which often leads to diagnostic errors and progression of the disease up to irreversible destruction of kidney tissue .

The purpose of this review is to systematize modern data on diagnostic and differential diagnostic methods for renal tuberculosis, highlighting key algorithms and emphasizing the importance of an integrated approach to prevent severe complications such as pyonephrosis, nephrosclerosis (“shrunken kidney”), and total calcification. (kidney pimple) or putty kidney ") and chronic renal failure .

Etiopathogenesis and clinical forms: the basis for understanding diagnostic manifestations

The pathogen enters the kidneys primarily through the bloodstream from a primary site, most often located in the lungs. Mycobacteria, settling in the glomeruli of the cortex, form multiple tiny granulomas. With an adequate immune response, the process can stabilize and even reverse. However, immunosuppression (HIV infection, diabetes, immunosuppressant medications, and chronic diseases) can lead to reactivation of the infection .

Pathogenesis is characterized by stages:

1. Tuberculosis of the renal parenchyma (stage I): multiple small foci in the cortex and medullary substance without destruction.
2. Tuberculous papillitis (stage II): damage to the renal papillae with the development of papillary necrosis .
3. Cavernous tuberculosis (stage III): fusion of foci with the formation of isolated cavities (caverns) with caseous masses.
4. Fibrocavernous (polycavernous) tuberculosis (stage IV): multiple cavities, fibrosis, and involvement of the renal pelvis and ureters. This can result in renal malformation —diffuse dystrophic calcification of the entire organ .

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The clinical picture is often asymptomatic . Possible signs include dysuria (frequent urination—up to 10–20 times per day), macrohematuria (especially terminal) or microhematuria, sterile leukocyturia (pyuria), and nonspecific lumbar pain. General symptoms (low-grade fever, weight loss, night sweats) are less pronounced than in the pulmonary form . It is this nonspecificity that dictates the need for a targeted diagnostic investigation.

Modern methods of diagnosing kidney tuberculosis

Diagnostics is based on the integrated use of laboratory, instrumental and functional methods.

1. Laboratory diagnostics

- General urinalysis: A persistent, sharply acidic urine reaction is characteristic , which is an important indirect sign. Leukocyturia , proteinuria, and erythrocyturia are detected , with no growth of common microflora in a standard culture ("sterile pyuria") .
- Microbiological and molecular genetic research:
  - Triple urine culture on specialized nutrient media (the culture method) remains the "gold standard" for detecting mycobacteria, despite its time-consuming nature (several weeks). Its specificity reaches 100%, and its sensitivity is approximately 65%.
  - Polymerase chain reaction (PCR) of urine is a highly sensitive (more than 90%) and specific (more than 95%) method that allows for the detection of Mycobacterium tuberculosis DNA within 6–24 hours, making it indispensable for rapid primary diagnosis .
  - Ziehl-Neelsen staining (or fluorescence microscopy) for the detection of acid-fast bacilli is less sensitive but is widely available .
- Tuberculin diagnostics (Mantoux test, Diaskintest): A positive reaction indicates mycobacterial infection, but does not differentiate active from latent infection . For nephrotuberculosis, the sensitivity of the test is high (up to 90%). Tests in vitro (T-SPOT.TB, QuantiFERON test) , independent of BCG vaccination.
- Tuberculin provocation test: Subcutaneous administration of tuberculin can cause an exacerbation of the process, which is manifested by increased pyuria, proteinuria and the appearance of mycobacteriuria in repeated urine tests, which helps in the diagnosis of questionable cases.

2. Radiation and instrumental diagnostics

Imaging plays a key role in determining the stage, extent of the process and treatment planning.

- Ultrasound examination (US): A first-line method, although it has limited specificity. It can detect hypoechoic foci in the parenchyma at early stages, Progressive kidneys exhibit papillary destruction, cystic lesions (caverns), irregular kidney contours, hydronephrosis, and calcifications . In the terminal stage, a shrunken, calcified kidney is visible .
- Radiographic methods:
  - A retroperitoneal and chest X-ray can detect renal calcifications (occurring in 25-50% of cases), which may have a characteristic triangular shape in papillary necrosis or a total amorphous appearance (" chalked kidney"). A chest X-ray can reveal active or past tuberculosis (although, according to some data, only 10% of patients with nephrotuberculosis have active pulmonary disease) .
  - Excretory (intravenous) urography (IVU) and retrograde ureteropyelography remain highly informative methods for assessing anatomy and function . Early signs on IVU include moth-eaten papillae, phantom calyces (due to destruction), and irregular cavernous contours. Late changes include ureteral and calyceal neck strictures, hydronephrosis, and bladder deformity (thimble bladder) .
- Computed tomography (CT) with intravenous contrast is the most sensitive and specific imaging technique. It allows for a detailed assessment of the parenchyma, renal pelvis, and perirenal tissue. CT findings include: multiple small hypodense lesions in the cortex, papillary necrosis, thin-walled caverns (often multiple), uneven contrast enhancement, ureteral wall thickening and strictures, and calcifications of varying shapes and distribution .

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- Magnetic resonance imaging (MRI): Less commonly used than CT, but useful for assessing inflammatory changes in soft tissues, particularly in patients with contraindications to iodine-based contrast.
- Cystoscopy: Allows visualization of specific changes in the bladder mucosa (tuberculous nodules, ulcers, cicatricial changes, retractions in the area of the ureteral orifices). Targeted biopsy of suspicious areas followed by histological examination (search for epithelioid cell granulomas with Pirogov- Langhans cells) and PCR verifies the diagnosis .

Differential diagnostics: key nosologies and differentiation algorithms

The differential diagnosis of nephrotuberculosis is most complex, as it involves diseases with similar clinical and radiographic presentations. The table below summarizes the main differential diagnoses.

Diagnostic algorithm for suspected nephrotuberculosis :

1. Analysis of anamnesis (contact with patients with tuberculosis, history of pulmonary tuberculosis, immunodeficiency states).
2. Laboratory screening: general urine analysis (acid reaction, sterile leukocyturia), triple urine culture for MBT, urine PCR for MBT, tuberculin diagnostics .
3. Primary imaging: ultrasound of the kidneys and bladder.
4. Advanced visualization for stage assessment: CT scan of the kidneys with contrast (method of choice) or excretory urography.
5. If the results are questionable or confirmation is required: tuberculin provocation test, cystoscopy with biopsy.
6. Diagnostics ex Juvantibus (therapeutic test): In difficult diagnostic cases, especially when differentiating from chronic nonspecific inflammation, a controlled course of specific anti-tuberculosis therapy can be used. Positive dynamics (disappearance of pyuria, improvement of the radiographic appearance) confirms the diagnosis.

5. Conclusion and Prospects

Diagnosis of renal tuberculosis remains a complex task, requiring a high level of suspicion from physicians (urologists, phthisiatricians, radiologist), especially in patients with nonspecific urinary symptoms that are refractory to standard therapy. Key diagnostic triggers include persistently acidic urine with sterile leukocyturia , detection of mycobacteria by PCR or culture, and specific CT findings (multiple calcified cavities , papillary necrosis, strictures).

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