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STRATEGIES FOR THE PREVENTION OF UROGENITAL TUBERCULOSIS: EPIDEMIOLOGICAL ASPECTS, EARLY DIAGNOSIS, PERSONALIZATION OF INFECTION CONTROL

Resume: Tuberculosis is an infectious disease caused by pathogenic mycobacteria belonging to the class of Actinobacteria, the order Actinomycetes, the family Mycobacteriaceae, forming the Mycobacterium tuberculosis complex group. Tuberculosis is a systemic infectious disease affecting mainly the respiratory system; osteoarticular and urogenital tuberculosis are in second or third place in terms of frequency of occurrence. Urogenital tuberculosis is an infectious and inflammatory disease of the genitourinary system caused by M. tuberculosis or M. bovis. It develops during the stage of secondary dissemination.

Key words:urogenital tuberculosis; diagnosis; epidemiology; HIV/tuberculosis co-infection; incidence of urogenital tuberculosis.

Tuberculosis of the urinary tract is an infectious and allergic inflammation of the calyx-pelvis complex, upper and lower urinary tract, passing through the stages of edema, infiltration, ulceration and scarring; secondary to nephrotuberculosis. The most common UGT is caused by Mycobacterium tuberculosis (80-95% of cases). Since tuberculosis is an anthropozoonous disease, M. bovis has not lost its relevance, accounting for up to 20% in some countries. M. bovis can cause the development of bladder tuberculosis after BCG therapy for bladder cancer.

Depending on the site of introduction, mycobacteria can initially enter the lung, tonsils, intestines, and so on. Since the causative agent of tuberculosis does not release exotoxin, and the possibilities for its phagocytosis at this stage are very limited, the presence of a small number of mycobacteria in tissues does not appear immediately. MBTs, being extracellular, multiply slowly, while the surrounding tissue remains intact and retains its normal structure.

This condition is defined as latent microbism, in which a macroorganism exhibits tolerance to mycobacteria. Regardless of the entrance gate, mycobacteria with a lymph flow of quite CR505 7 quickly enter the regional lymph nodes, from where they spread throughout the body in a lymphohematogenic way. Primary (obligate) mycobacteriemia occurs. MBTs settle in organs with the most developed microcirculatory bed – in the lungs, lymph nodes, the cortical layer of the kidneys, epiphyses and metaphyses of tubular bones, ampullofimbrionic sections of the fallopian tubes, uveal tract of the eye. However, primary bacteremia does not necessarily entail the development of disseminated tuberculosis, although this explains the occurrence of distant primary affects, for example, in the bone marrow, in the meninges; there may be several primary affects.

The predominant damage to certain organs and systems cannot be explained by a single hit to these organs by a large number of MBTs. Tuberculosis is a clinically heterogeneous disease. There is a primary form of the disease that develops in a previously uninfected organism with mycobacteria, as well as the secondary genesis of tuberculosis, when the disease occurs with repeated infection. Exogenous superinfection as a cause of secondary tuberculosis is many times more frequent than reactivation of old foci.

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In most cases, tuberculosis begins with the formation of a focus of inflammation at the site of the infection gate, followed by damage to the regional lymph nodes, forming the so-called primary complex. Primary infection through the respiratory system is the most common. However, primary infection may not be accompanied by the formation of a focus of inflammation in the area of the infection gate, for example, when infected through mucous membranes.

Then the onset of the disease is a general tuberculous dissemination along the lymphatic and circulatory pathways, resulting in the formation of multiple foci of tuberculous inflammation in the tissues of internal organs, lymph nodes and bone marrow. With the development of tuberculous lymphadenitis, blood flow is disrupted, which causes retrograde spread of MBT and damage to lymph nodes in other areas. The development of isolated local forms is the result of partial healing of some foci of dissemination (most of them) with the progressive development of others. Against the background of waves of hematogenous dissemination, hidden or clinically detectable, various forms of "isolated" or "organ" lesions may develop, which are part of the group of extrapulmonary forms of secondary tuberculosis.

The development of these forms has a pathogenesis similar to that of extrapulmonary lesions developing against the background of CD505 8 hematogenous dissemination in primary tuberculosis. These organ forms develop against the background of general tuberculosis dissemination, some elements of which are cured, others receive progressive development. Kidney tuberculosis, like all other organ forms of tuberculosis, always develops against the background of general hematogenous dissemination, other elements of which may remain active or heal (heal), and the dissemination process may be hidden and not clinically recognized. When determining the fact of a predominant lesion of an organ or organ system, the question arises about the causes of the "focus of least resistance" of tuberculosis infection in the presence of general resistance, ensuring the elimination of phenomena of general dissemination.

The provoking factors in the case of urotuberculosis are mainly disorders of urodynamics and microcirculation. Tuberculosis of the kidney is a local manifestation of tuberculosis disease of a predominantly hematogenous nature, beginning with tubercular lesions of the organ in the cortical layer of the parenchyma and periglomerular zone. The spread of the tuberculosis process from the initial tuberculous foci in the kidney occurs perivascularly along the straight vessels to the medulla, along the interlobular vessels to the calyx-pelvis system, along the stellate vein system to neighboring areas of the cortical layer, along the arcuate vessels to the neighboring segment of the kidney.

Thus, secondary tuberculosis, which includes damage to the organs of the genitourinary system, occurs against the background of altered reactivity of the body, due to the exacerbation of dormant foci of primary infection (endogenous reinfection) or exogenous superinfection, that is, repeated, additional infection. During the second wave of bacteremia, mycobacteria, passing through the blood vessels of the kidney, penetrate into the parenchyma and settle in the periglobular region, causing an influx of macrophages. A granuloma with a Pirogov-Langgans cell in the center, surrounded by lymphocytes and fibroblasts, develops from a cluster of macrophages.

The further course of events depends on the virulence of the MBT, the degree of infection and the resistance of the macroorganism. Under favorable circumstances, the tuberculous tubercle grows with fibrous tissue, otherwise caseous necrosis forms in its center. Spreading to the surrounding tissues, necrosis reaches the papilla of the kidney, or, much less often, it is surrounded by a dense three-layered capsule, forming a subcortical cavity that does not communicate with the cup-pelvic system. Before the advent of anti-tuberculosis drugs, nephrotuberculosis was an extremely common disease: every fifth patient in a urological hospital suffered from tuberculosis of the kidneys, more than 1/3 of all renal suppurations were of tuberculous origin.

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Currently, UGT ranks first in the structure of the incidence of extrapulmonary tuberculosis in countries with a high incidence rate, where the proportion of UGT reaches 33.7–45.5%, and in third place in countries that are well—off in tuberculosis. In developed countries, 2-10% of patients with pulmonary tuberculosis also have UGT. 20% of patients with pulmonary tuberculosis eventually develop an extrapulmonary manifestation, most often in the genitourinary system. In recent years, UGT has lost its leading position in Russia to osteoarticular tuberculosis.

The alertness index should be based on high probability factors, which include:

- close contact with tuberculosis infection,
- tuberculosis of any localization, previously transmitted or active at the time of treatment,
- chronic infections of the urogenital tract, prone to recurrence, resistant to standard therapy,
- persistent dysuria,
- progressive decrease in bladder capacity,
- sterile pyuria,
- pyuria in 3 servings in a patient with epididymitis,
- pyospermia, hemospermia,
- pyuria, hematuria,
- fistulas in the lumbar region, perineum, scrotal fistulas.

However, PVV is not crucial, but of auxiliary importance in the diagnosis of UGT. In addition to the stage of the disease, the diagnosis indicates the bacterial release and the group of dispensary registration (DG), and is also encrypted according to the international classification of diseases ICD-10.

The most severe form of UGT is taken into account (a patient with polycavernous nephrotuberculosis, on the one hand, and papillitis, on the other, is considered as a patient with stage 4 renal tuberculosis).

Thus, the final diagnosis may sound, for example, like this: A18.1 Tuberculosis of the renal parenchyma, MBT+. GDU-1. Or: A18.1.2 Polycavernous tuberculosis of the right kidney with lack of function, tuberculous papillitis on the left. Tuberculosis of the right ureter, stage 3 tuberculosis of the bladder. CPN-1. MBT+. GDU-1.

Tuberculosis of the renal parenchyma is a minimal, initial, non–destructive form of nephrotuberculosis (stage 1), when not only a clinical, but also an anatomical cure is possible. In tuberculosis of the renal parenchyma, the structure of the calyx-pelvis system is normal on urograms, neither destruction nor retention are determined. There may be no pathological changes in urine tests in children, although moderate leukocyturia is usually found in adults.

Mycobacteriuria in healthy kidneys, even during primary or secondary bacteremia, is impossible – the causative agent of tuberculosis is not filtered through healthy glomeruli, therefore, the detection of Mycobacterium tuberculosis in the urine is always a sign of the disease. Bacteriological verification of tuberculosis of the kidney parenchyma is mandatory. It is impossible to identify the sides of the lesion in tuberculosis of the parenchyma, therefore this disease is always considered bilateral. Complications are extremely rare. The prognosis is favorable. Outcome with a favorable course – clinical and anatomical cure; formation of small calcifications in the kidney parenchyma; in case of unfavorable – progression of tuberculous inflammation with the formation of subcortical cavity or tuberculous papillitis.

Tuberculous papillitis (stage 2, limited destructive form) can be one- and two-sided, single and multiple. Mycobacteriuria is not always detected; it is usually complicated by tuberculosis of the urinary tract. It is subject to conservative treatment; with inadequate etiopathogenetic therapy, ureteral stricture may form, which requires prompt correction. The prognosis is favorable, although anatomical

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recovery is impossible. The outcome with a favorable course is the development of cicatricial deformity of the calyx-pelvis complex, the formation of post-tuberculosis pyelonephritis. The outcome of an unfavorable course is the progression of the process with the formation of kidney cavities, the spread of inflammation to the urinary tract.

Cavernous tuberculosis of the kidney – (stage 3, destructive form) – develops pathogenetically in two ways – from tuberculosis of the parenchyma or from papillitis. In the first case, a subcortical cavity is formed that does not communicate with the cup-pelvic system; the clinical picture is similar to that of a kidney carbuncle. Subcortical cavern is diagnosed, as a rule, pathomorphologically after surgery in a general medical network. In the second case, the formation of the cavity occurs due to the spread of papilla destruction.

Cavernous nephrotuberculosis can be one- and two-sided; it is possible that tuberculous papillitis is diagnosed in one kidney and a cavity in the other. In this case, the patient is observed for a more severe form of the disease. Complications develop in more than half of the patients. As a rule, cavernous nephrotuberculosis requires surgical intervention. A complete cure cannot be achieved, although the use of methods of complex etiopathogenetic therapy allows in some cases to transform the kidney cavity into a sanitized cyst. A favorable outcome is the transformation of the cavity into a sanitized cyst; the formation of a post-tuberculosis deformation of the pelvic system.

An unfavorable outcome is the progression of destruction with the development of polycavernous nephrotuberculosis, the development of tuberculosis of the urinary tract. Polycavernous tuberculosis of the kidney (stage 4, widespread destructive form) involves the presence of several cavities, which leads to a sharp decrease in organ function. As an extreme case, pionephrosis with the formation of a fistula is possible. At the same time, self-healing is also possible, the so-called "autoamputation of the kidney" - imbibation of cavities with calcium salts and complete obliteration of the ureter. Complications almost always develop; the presence of a tuberculous focus in the contralateral kidney is likely. As a rule, it is cured by organ-carrying surgery.

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